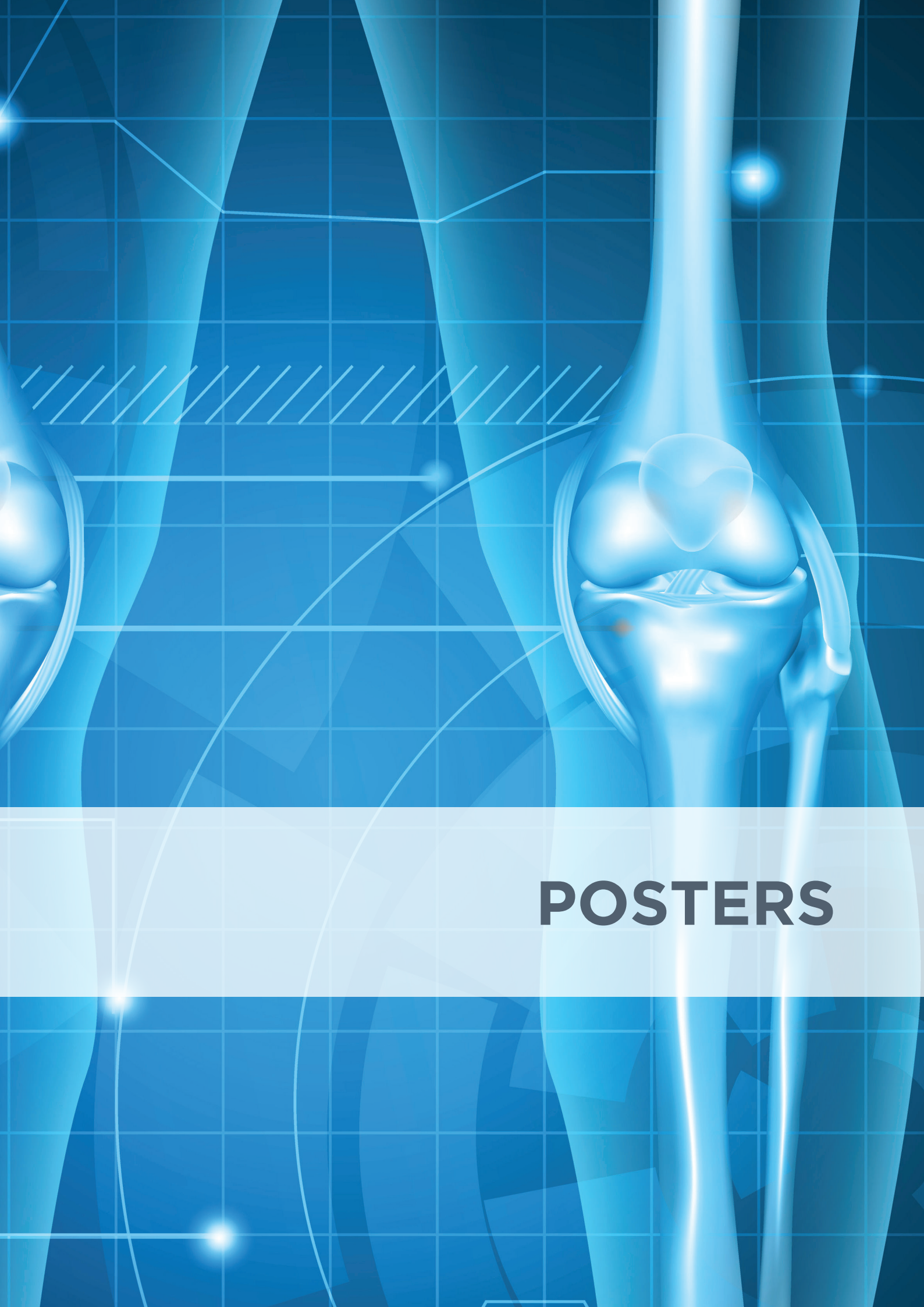


POSTERS

ARP Rheumatology 2023;3:45-191 (SUP)

Trabalho Original

003 - THE ROLE OF IMMUNOSUPPRESSIVE THERAPY IN GASTROINTESTINAL INVOLVEMENT AND ITS IMPACT ON QUALITY OF LIFE IN PATIENTS WITH SYSTEMIC SCLEROSIS - A COHORT STUDY

Inês Genrinho[1,2], Inês Santos[1], Couto M[1], Flavio Costa[3], Tiago Beirão[3], Carolina Mazeda[2,4], Anabela Barcelos[2,4], Adriana Carones[5], Tânia Santiago[6,7], Georgina Terroso[8], Maria Seabra Rato[8], Pedro Lopes Ferreira[9], Paulo Monteiro[1]
[1]Unidade de Reumatologia, Centro Hospitalar Tondela - Viseu, Viseu, Portugal, [2]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/Espinho, Gaia, Portugal, [4]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal, [5]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [6]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal, [7]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [8]Serviço de Reumatologia, Centro Hospitalar de São João, Porto, Portugal, [9]Economics department, Faculty of Economics, University of Coimbra, Coimbra, Portugal

Introduction: Gastrointestinal (GI) disease is a major cause of morbimortality in systemic sclerosis (SSc) and affects up to 90% of patients. GI manifestations are frequently unrecognized in early phases and there are few data suggesting the potential effect of immunosuppression (IS) in GI tract. The aim of this study was to evaluate the role of IS in GI involvement and its impact on health-related quality of life (HRQoL) in SSc patients.
Methods: A cross-sectional multicenter study was conducted, enrolling SSc patients that fulfilled the 2013ACR/EULAR criteria or presented the EUSTAR criteria for very early diagnosis of SSc (VEDOSS). Patients were requested to answer UCLA Scleroderma Clinical Trial Consortium Gastrointestinal Tract, SHAQ and Short Form Health Survey (SF36) measures. Social-demographic and clinical data were collected. GI involvement was determined by the presence of one of the following: GI symptoms for at least 3 of the last 7 days, abnormalities in GI exams and/or use of GI pharmacological therapies. IS was defined as exposure to at least one of the following: mycophenolate mofetil (MMF), cyclophosphamide (CYC), methotrexate (MTX), azathioprine (AZA), leflunomide, glucocorticoids (>10mg/d prednisone-equivalent), rituximab, tocilizumab, and abatacept, for more than 6 months. General descriptive analysis and independent parametric or non-parametric tests were performed using SPSSv26.
Results: One-hundred and one patients were included, 83 female (82.2%), with a mean age of 56.8±12.39 years-old and a mean duration of disease of 67.2±72.5 months. Sixty-four patients (63.4%) had limited SSc, 30 (29.7%) diffuse and 7 (6.9%) presented VEDOSS. Forty patients were under IS therapy, being the MTX the most frequent drug in 21.8%, followed by MMF in 11.9% and AZA in 5.9%. Seventy-two patients (71.3%) had GI involvement, where clinical manifestations were present in 72.2% patients, abnormalities in GI exams in 44.4% and GI pharmacotherapy was used in 41.7% patients. Patients with GI involvement were significantly more frequent under IS therapy (p=0.044), although no differences were found in severity of symptoms in UCLA domains between patients with and without IS. On the other hand, patients without GI involvement and under IS therapy presented significantly higher total UCLA score (mean 0.10±0.11 vs 0.11±0.18 for patients with and without IS therapy respectively, p=0.046). IS group was significantly more frequent under GI pharmacotherapy (p=0.006), being the proton pump inhibitors (PPI) the most significantly prescribed drug (55% vs 16.4% for patients with and without IS respectively, p<0.01). IS group had significantly higher SHAQ scores in total item (p=0.001) and overall disease severity VAS (p=0.009). Regarding HRQoL patients under IS present significantly lower SF36 scores for physical functioning (p=0.001), physical role functioning (p=0.002), physical component summary (p=0.001) and general health perception (p=0.036).
Conclusion: Patients with GI involvement were more frequent under IS, although no differences were found in severity of symptoms in UCLA domains when compared to those without IS. IS therapy was associated with worst HRQoL probably due to more severe multi-organ disease and were treated more often with PPI. Investigation is needed to evaluate the potential effects of IS in GI tract, as patients with absence of GI involvement presented higher UCLA scores when IS therapy was given, which may reflect the potential GI complaints as an adverse effect by therapy, rather than involvement by the SSc.

005 - KL-6 AND IL-18 LEVELS ARE NEGATIVELY CORRELATED WITH RESPIRATORY FUNCTION TESTS AND ILD EXTENT ASSESSED ON HRCT IN PATIENTS WITH SYSTEMIC SCLEROSIS-RELATED INTERSTITIAL LUNG DISEASE (SSC-ILD)

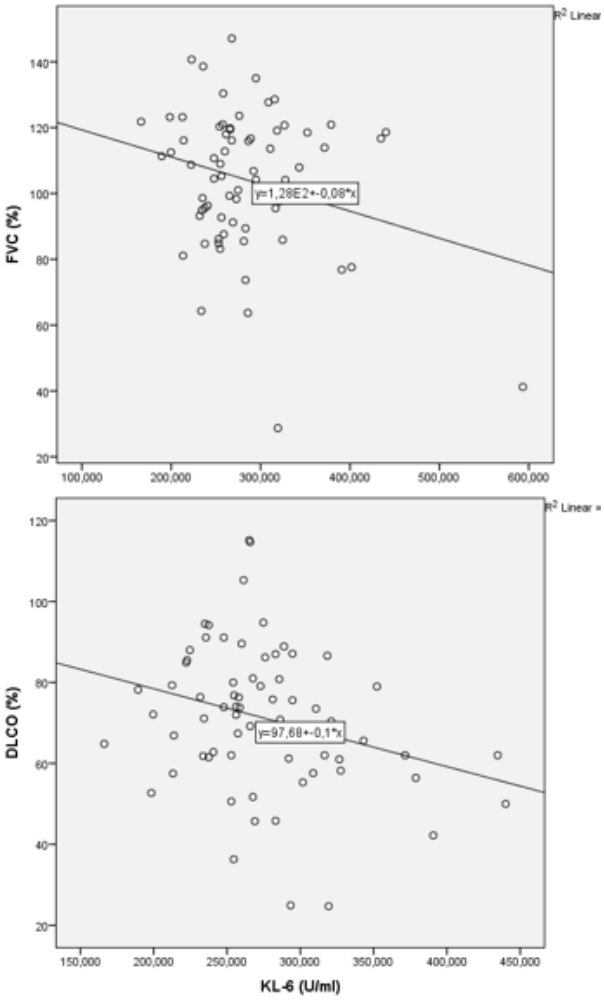
Cristiana Sieiro Santos[1], Sara Calleja[2], Javier de la Calle[3], Carmen López[4], Clara Moriano[1], Elena Bollo[4], Elvira Díez[1]

[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Immunology, Complejo Asistencial Universitario de León, León, Spain, [3]Radiology, Complejo Asistencial Universitario de León, León, Spain, [4]Pneumology, Complejo Asistencial Universitario de León, León, Spain

Background: Interstitial lung disease (ILD) is one of the leading causes of mortality in patients with systemic sclerosis (SSc). Serum biomarkers have been suggested as indicators for pulmonary damage with clinical value in the diagnosis and prognosis of SSc-ILD.
Objectives: To investigate the role of serum biomarkers (Krebs von den Lungen-6 KL-6, IL-18 and IL-18BP) as potential biomarkers reflecting the severity of SSc-ILD as assessed through high-resolution computed tomography (HRCT) and pulmonary function tests (PFT), including forced vital capacity (%FVC) and diffusing capacity of the lung for carbon monoxide (%DLCO).
Methods: A cross-sectional study including patients with SSc fulfilling the 2013 ACR/EULAR criteria was performed. Patients were classified according to disease duration and pulmonary involvement (presence of ILD). All SSc patients underwent chest HRCT scans and pulmonary function test at baseline. Serum concentration of KL-6, IL8 and IL18BP were determined using sandwich ELISA technique (solid phase sandwich Enzyme Linked-Immunosorbent Assay), with kits from MyBiosource for KL-6 and from Invitrogen for IL18 and IL18BP. A semiquantitative grade of ILD extent was evaluated through HRCT scan (grade 1, 0–20%; grade 2, >20%). Extensive lung disease was defined as >20% of lung involvement on HRCT, and FVC <70% predicted and limited lung involvement as ≤20% of ILD involvement on HRCT, and an FVC ≥70% predicted.
Results: 74 patients were included, 27% were male. The mean age at diagnosis was 57.5±15 years. The mean time since diagnosis was 7.67±8 years. 28 patients had ILD (38%). 64 % of patients had <20% of ILD extent classified through HRCT scan. SSc-ILD patients had elevated serum KL-6 and IL-18 levels compared to patients without ILD (p=0.003 and p=0.04), and those findings were preserved after adjusting for age and sex (table 1). Mean erythrocyte sedimentation rate (ESR) was higher in patients with SSc-ILD. A negative correlation between KL-6 levels and %FVC (β=-0.25, p 0.037) and %DLCO (β=-0.28, p 0.02) and between IL-18 levels and %FVC (β=-0.20, p 0.03) and %DLCO (β=-0.14, p 0.04) were found. Linear regression models representing correlation between KL-6 and PFT are represented in the scatter plot in figure 1. Serum KL-6 and IL-18 levels successfully differentiated grades 1 and 2 (p = 0.028 and p=0.021). Semiquantitative grades of ILD on the HRCT scan were significantly proportional to the KL-6 (p 0.01) and IL-18 (p=0.03). A positive correlation between extensive lung disease and KL-6 (β=0.61, p 0.007) but not with IL-18 was found.
Conclusions: Serum KL-6 levels and IL-18 were increased

TO 003 TABLE 1. Demographic and clinical characteristics of patients with SSc-ILD and non-SSc-ILD

	ILD (28)	ILD (-) (46)	P value
Mean age, years	57.3±16	57.7±28	0.97
Sex, male	6 (21%)	13 (28%)	0.51
Mean ESR (mm/h)	23±16	9±8	0.04
Mean CRP (mg/dL)	7.1±4.3	5.03±3.25	0.36
Serum KL-6 (U/mL)	320±68	262.5±39	0.003
Serum IL-18BP (U/mL)	150.46±130	123.23±90	0.21
Serum IL-18 (U/mL)	300.63±167	209.65±180	0.03
Pulmonary function tests			
Mean FVC %	96.2±34	111±22.3	0.01
Mean DLCO%	65.7±18	77.5±19.7	0.001



003 Figure 1.

Conclusions: Serum KL-6 levels and IL-18 were increased

in patients with SSc-ILD and showed a positive correlation with ILD severity as measured using a semiquantitative HRCT grading scale and a negative correlation with PFT parameters. KL-6 is positively correlated with extensive lung disease. Serum KL-6 and IL-18 could be a clinically useful biomarker in screening and evaluating SSc-ILD.

006 - NAILFOLD CAPILLAROSCOPY FINDINGS IN PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOPATHIES AND ITS ASSOCIATION TO AUTOANTIBODIES: A CASE-CONTROL STUDY

Cristiana Sieiro Santos[1], Jose Luis Tandaipan[2], Anais Mariscal[2], Luis Sainz Coma[2], Helena Codes[2], Patricia Moya[2], Berta Magallares[2], Hye Sang Park[2], Hector Corominas[2], Ivan Castellvi[2]

[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Reumatologia, Hospital de La Santa Creu I Sant Pau, Barcelona, Spain

Background: Idiopathic inflammatory myopathies (IIM) are a heterogeneous group of acquired muscle diseases, which have distinct clinical, pathological and histological features. Autoantibodies are clinically useful biomarkers to help the diagnosis of IIM. Raynaud's phenomenon is very frequent and the presence of microvascular changes in IIM have been described however, the role of nailfold videocapillaroscopy NVC) for diagnosis and prognosis in IIM is not clearly established.
Objectives: The aim of this study was to study the relationship between clinical and immunological characteristics and nailfold videocapillaroscopy (NVC) abnormalities in patients with idiopathic inflammatory myopathies (IIMs).
Methods: We performed a retrospective study of IIM patients followed in a University Hospital. Patients underwent a NVC at 200x magnification. Epidemiological, clinical data and antibody status, including myositis and scleroderma antibody panel of all patients was retrieved. NVC findings including loss of capillary density, enlarged and giant capillaries, ramified capillaries, haemorrhages, thrombosis, avascular areas, disorganization of capillary architecture and subpapillary venous plexus presence were recollected, if present. For the comparison of qualitative and/or quantitative variables Fisher's exact Test or T-test was performed when necessary.
Results: 95 patients with NVC performed during the follow-up were included (66% female) with a median age at inclusion of 55.3±24 years. Median IIM duration was 6.8±7 years. 39% had Raynaud's phenomenon at first clinical evaluation and 58% of them showed NVC pathological findings. Table 1 Summarizes the epidemiological, clinical, and autoantibody status of the patients. We found an association between the presence of dysphagia and avascular areas (p=0.02) or abnormal capillary organization (p<0.01) on NVC. ILD was associated with capillary loss (p=0.04) and avascular areas (p=0.004). Anti-MDA5+ was associated with capillary loss (p=0.03), thrombosis (p=0.02) and ramified capillaries (p=0.04). Anti-Mi2+ and anti-Th/To was associated with abnormal capillary organization (p=0.017 and p=0.001). The presence of haemorrhages was associated with anti-Ku+ (p=0.048) and anti-PL12+ (p=0.046). The presence of enlarged capillaries was associated with anti-RNA-pol III (p=0.04) and anti-NXP2 (p=0.044). A significant association between anti-Ro52 (OR 2.69, CI 95% 1.05-6.8, p 0.03) and anti-Jo1 (OR 7.03 CI 95% 1.46-33.7, p 0.01) with ILD was found. Anti-PML (OR 4.32 CI 95% 1.35-10.42, p 0.038) and anti-Th/To (OR 5.82 CI 95% 1.89-13.24, p 0.04) were associated with dysphagia. Anti-MDA5 (OR 5.85 CI 95% 1.92-14.21, p 0.044) was associated with skin involvement.
Conclusions: The presence of certain autoantibodies is related to the degree of microangiopathy in IIM and associates with capillaroscopic changes. Studying the asso-

TO 006 – TABLE 1. Epidemiological, clinical, immunological and NFC features and autoantibody status from patients with IIM

Clinical features	
Muscle weakness	79 (83%)
Skin findings	30 (32%)
ILD	47 (49%)
Dysphagia	18 (19%)
Raynaud's phenomenon	37 (39%)
CK elevation	45 (47%)
Cardiac disease	4 (4%)
NFC features	
Loss of capillary density	27 (28%)
Enlarged and giant capillaries	37 (39%)
Haemorrhages	36 (38%)
Thrombosis	19 (20%)
Avascular areas	26 (27%)
Disorganization of capillary architecture	24 (25%)
Subpapillary venous plexus	36 (38%)
Antibody status	
Disease-specific antibodies	
Anti-MDA5	8 (8%)
Anti-TIF1G	11 (12%)
Anti-MI2	11 (12%)
Anti-NXP2	6 (6%)
Anti-synthetase antibodies	
Anti-Jo1	13 (14%)
Anti-PL7	9 (9%)
Anti-PL12	5 (5%)
Disease-associated antibodies	
Anti-Ro52	27 (28%)
Anti-KU	6 (6%)
Antinuclear antibodies	61 (64%)
Others (EJ, SRP, PM-SCL75, PM-SCL100, CN1A)	22 (23%)

ciation between capillaroscopic changes with diagnostic and pathogenic autoantibodies in IIM can provide useful information regarding the current knowledge about pathogenesis, classification, and prognosis of the disease.

007 - EXTRAGLANDULAR INVOLVEMENT AND AUTOANTIBODY STATUS AS RISK FACTORS FOR CARDIOVASCULAR DISEASE IN PRIMARY SJOGREN'S SYNDROME (PSS): A 20 YEAR-FOLLOW UP STUDY

Cristiana Sieiro Santos[1], Ruben Rego Salgueiro[2], Clara Moriano[1], Carolina Álvarez[1], Elvira Díez[1]
[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Medicina Interna, ULS Guarda, Guarda, Portugal

Background: Primary Sjögren's syndrome (pSS) is an autoimmune disorder characterized by chronic multisystem inflammation with shared pathophysiology with SLE and RA. Cardiovascular events have emerged as major causes of morbidity and mortality in patients with autoimmune diseases however, the clinical significance of cardiovascular disease in patients with pSS remains unclear.
Objectives: To study the association between cardiovascular disease and primary Sjögren's syndrome (pSS) and analyze the risk of cardiovascular disease accordingly to glandular/extraglandular involvement and anti-Ro/SSA and/or Anti-La/SSB autoantibody status.
Methods: pSS patients fulfilling the 2016 ACR/EULAR classification criteria for pSS were consecutively evaluated and followed in our department between 2000 and 2022. We evaluated the prevalence and clinical significance of cardiovascular risk factors with primary Sjögren's syndrome (SS), focusing on the possible association with clinical and immunological features, the therapies administered, and the impact on cardiovascular disease. A two-tailed value of $p<0.05$ was taken to indicate statistical significance. Potential risk factors associated with cardiovascular involvement were determined by multivariate regression analyses.
Results: A total of 102 pSS patients were included. 90% were female, with a mean age of 65±24 years and a disease duration of 9.9 ±7 years. The baseline prevalence of comorbidities was 59% for hypertension, 29% for cardiovascular diseases, 34% for dyslipidemia, 15% of diabetes, 29% for obesity, 12% had history of stroke and 17% had arterial/venous thromboembolism. 39% of patients had a history of smoking. Patients with extraglandular involvement had a higher prevalence of cardiovascular risk factors, including arterial hypertension (OR 2.28 95% CI (1.01-5.09), p 0.04), dyslipidemia (OR 4.4 95% CI (1.67-11.6), p 0.003), higher LDL mean values (116±48 vs 99±44, p 0.038), uric acid (6.58±1.7 vs 4.3±1.03, p 0.04) and higher risk for myocardial ischemia (OR 4.09 95% CI (1.46-11.4), p 0.01) after adjustment for age, sex, disease duration, and the significant variables in the univariate analysis. Patients positive for both Ro/SSA and La/SSB autoantibodies had a substantially higher risk of arrythmia (OR 3.4 95% CI (1.01-10.6), p 0.04), arterial and venous thromboembolism (OR 5.5 95% (1.18-25.7), p 0.03) and stroke (OR 3 95% (1.02-8.8), p 0.04). In the multivariate logistic regression analysis, extraglandular organ involvement (p=0.008), beta2microglobulin levels (p=0.001), hypocomplementemia of C3 (p=0.01), the use of glucorticoids (p=0.02), hypergammaglobulinemia (p=0.02), ESR levels (p=0.007), treatment with HCQ (p=0.03) and an ESSDAI (Sjögren's syndrome disease activity index)>13 (p=0.02) were found to be factors associated with increased or decreased odds ratio for cardiovascular events in pSS patients. Anti-Ro/SSA and anti-La/SSB were significant predictors in univariate but not in multivariate analysis.
Conclusions: pSS patients are more vulnerable to cardiovascular diseases (CVDs). In addition to traditional CVD risk factors, we identified risk factors independently associated with cardiovascular involvement in pSS patients, which suggests the need for early detection and prevention measures to improve the prognosis in those patients.

TO 007 – TABLE. Significant logistic regressions for predictors for CV risk

	OR 95% IC	P value
Extraglandular involvement	16.5 (4.51-28.5)	0.008
Beta2microglobulin (mg/dL)	7.83 (3.16-12.5)	0.001
C3 (mg/dl)	0.92 (0.24-0.98)	0.01
Corticoids	7.2 (2.34-15.7)	0.02
Hypergammaglobulinemia	10.2 (4.5-21.2)	0.02
ESSDAI >13	1.8 (1.13-4.52)	0.02
ESR (mm/h)	1.4 (1.10-3.45)	0.007
HCQ	0.82 (0.26-0.92)	0.03

008 - ASSOCIATION BETWEEN NAILFOLD VIDEOCAPILLAROSCOPY FINDINGS AND SKL-6 LEVELS IN PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOPATHIES-RELATED INTERSTITIAL LUNG DISEASE

Cristiana Sieiro Santos[1], Jose Luis Tandaipan[2], Helena

Codes[2], Luís Saínz[2], Berta Magallares[2], Patricia Moya[2], Berta Magallares[2], Hye Sang Park[2], Hector Corominas[2], Ivan Castellvi[2]
[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Reumatologia, Hospital de La Santa Creu I Sant Pau, Barcelona, Spain

Background: Idiopathic inflammatory myopathies (IIM) are a heterogeneous group of acquired muscle diseases with distinct clinical, pathological and histological features. interstitial lung disease (ILD) is a frequent pulmonary manifestation in IIM (IIM-ILD) and considerably influences morbidity and mortality. Krebs von den Lungen 6 (sKL-6) has been proposed as a potential biomarker reflecting the severity of ILD in connective tissue diseases. Raynaud's phenomenon is very frequent and the presence of microvascular changes in IIM have been described however, the role of nailfold videocapillaroscopy (NVC) in diagnosis and prognosis in IIM is not clearly established.
Objectives: To determine if there is any association between NVC findings, sKL-6 levels and pulmonary involvement in patients with inflammatory myopathies.
Methods: We performed a retrospective study of IIM patients followed in a reference center and compared them according to the presence of ILD. Epidemiological, clinical and immunological data, pulmonary function tests (forced vital capacity and diffusing capacity for carbon monoxide), sKL-6 levels and NVC finding were retrieved. NVC findings including loss of capillary density, enlarged and giant capillaries, ramified capillaries, haemorrhages, thrombosis, avascular areas, disorganization of capillary architecture and subpapillary venous plexus presence were recollected, if present. Statistical analysis was performed by T-test and Fisher's exact test to compare qualitative and/or quantitative variables and multiple logistic regression modelling to identify correlation between pulmonary function tests, NVC findings and sKL-6 levels. Values of $p<0.05$ were considered statistically significant.
Results: 95 patients were included, 47 patients (49%) with ILD. 34% were male with a median age at inclusion of 55.3±24 years and a median disease duration of 6.8±7 years. Avascular areas and capillary loss showed a significant association with the presence of ILD (OR 2.43, 95% CI 1.3-5.7, p 0.004) and (OR 1.7, 95% CI 1.48-3.1, p 0.04). A negative correlation between capillary loss and enlarged capillaries was also found with FVC% (β=-0.46, p 0.001 and β=-0.57, p <0.0001) and DLCO% (β=-0.32, p 0.04 and β=-0.23, p 0.03), respectively. When we studied the correlation between sKL-6 levels, positive correlations with the presence of ILD (β=0.77, p 0.0004), the presence of hemorrhages (β =0.21, p 0.04) and avascular areas in NVC (β =0.64, p 0.03) and negative correlations with FVC% (β=-0.47, p 0.001) and DLCO% (β=-0.59, p, 0.005) were found. Multiple logistic regression identified as predictors for developing IIM-ILD are summarized in table 1 and represented in the scatter plot in figure 1. Male sex, respiratory symptoms, %FVC and %DLCO, sKL-6 levels, anti-Jo1 positivity and the presence of avascular areas and enlarged capillaries in NVC were identified as IIM-ILD predictors (R^2=0.974, p =0.006).

TO 008 – TABLE 1. Significant logistic regressions for predictors for IIM-ILD

Predictor	P value	P value
Male sex	0.186	0.036
Respiratory symptoms	0.40	0.002
%FVC	-0.322	0.01
%DLCO	-0.59	0.001
sKL-6 levels	0.53	0.002
Anti-Jo 1	0.28	0.03
Avascular areas	0.72	0.006
Enlarged capillaries	0.49	0.04

Conclusions: Capillary loss and avascular areas showed a significant association with the presence of ILD, worse FVC and DLCO values and sKL-6 levels. We identified 9 predictors for developing ILD in IIM. NVC assessment and sKL-6 levels can have a predictive role for studying pulmonary function and assessing the prognosis of IIM-ILD.

009 - PREGNANCY IN CONNECTIVE TISSUE DISEASES: A 30 YEAR FOLLOW-UP STUDY OF 465 PREGNANCIES FROM A SPANISH MONOCENTRIC REGISTRY

Cristiana Sieiro Santos[1], Clara Moriano[1], Carolina Álvarez[1], Elvira Díez[1]
[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain

Background: Pregnancy in patients with connective tissue diseases are known to be at high risk for the occurrence of adverse pregnancy outcomes.
Objectives: To evaluate the pregnancy outcomes in patients with systemic autoimmune diseases, including systemic lupus erythematosus (SLE), systemic sclerosis (SSc), primary Sjögren's syndrome (pSS) and undifferentiated connective tissue disease (UCTD).
Methods: A retrospective and descriptive study was conducted from 1990 to 2020. All data were collected

TO 009 – TABLE 1.

	SLE	SSc	pSS	UCTD	P value
Total of pregnancies	192	88	120	65	
Age at pregnancy	32.4±4.5	29.5±7.2	30.4±3.5	33.5±2.7	0.45
Smokers	32 (26%)	17 (34%)	25 (31%)	12 (30%)	0.27
Birth	103 (67%)	68 (77%)	90 (75%)	47 (72%)	0.28
Abortion	57 (37%)	12 (14%)	18 (15%)	10 (15%)	0.03
Mean abortion number	2.7±0.7	1.1±0.6	2.4±0.3	0.9±0.5	0.03
Preeclampsia	15 (9.8%)	3 (3%)	2 (2%)	2 (3%)	0.04
Ectopic pregnancy	10 (6.5%)	0	1 (1%)	1 (2%)	0.03
Placental abnormalities	8 (5%)	5 (5.7%)	2 (2%)	3 (5%)	0.21
Premature rupture of membranes (PROM)	5 (3.3%)	0	2 (2%)	1 (2%)	0.24
Preterm delivery	20 (13%)	0	8 (6%)	5 (8%)	0.02
Postpartum haemorrhage	21 (14%)	0	4 (4%)	1 (2%)	0.01

from the medical records of childbearing age women with SLE, SSc, SS and UTCD enrolled in our clinic at the time of their pregnancy and childbirth. The obstetric, maternal and fetal outcomes were collected and compared regarding diagnosis and adverse outcomes.
Results: The study group included 295 patients, 125 patients (42%) with SLE, 50 patients (17%) with SSc, 80 patients (27%) with Sjogren's, 40 patients (14%) with UCTD. A total of 465 pregnancies were registered. The maternal and fetal outcomes are detailed in table 1 and figure 1. The mean age at delivery was 31.5±8.5 years and the mean duration of disease was 7.2±5.6 years. Pregnancy loss occurred in 21% of patients, live births in 66% of pregnancies, preterm delivery in 8%, postpartum haemorrhage in 6%, preeclampsia in 5%, placental abnormalities in 4%, ectopic pregnancy in 3%, premature rupture of membranes in 2%. Treatment with HCQ was received in 115 pregnancies in SLE (59%), 21 pregnancies in SSc (24%) 62 pregnancies in pSS (52%) and 32 pregnancies in UCTD (49%). Exposure to corticosteroids and biologics during pregnancy was 23 (18.4%), 6 (12%), 15 (19%) and 3 (7.5%), respectively. Patients with SLE had a higher risk of fetal morbidity, including abortion (p=0.03), mean abortion rate (p=0.03), preeclampsia (p=0.04), ectopic pregnancy (p=0.03), preterm delivery (p=0.02) and postpartum haemorrhage (p=0.01) than patients without SLE. The multivariate model adjusted for age, nulliparity, active disease activity during pregnancy, smoking and exposure to biologics, HCQ and corticosteroids found an association between unfavourable pregnancy outcomes and disease activity (OR 2.4 95% CI (1.3-7.2), p 0.003), whilst HCQ during pregnancy (OR 0.23 95% CI (0.03-0.82) had a protective effect.
Conclusions: 66% of pregnancies in patients with autoimmune diseases resulted in live births. Patients with SLE had higher rates of fetal and maternal morbidity than SSc, pSS and UCTD. Disease activity was associated with unfavourable pregnancy outcomes. Exposure to HCQ had a protective effect during pregnancy. Pregnancy planning and counselling prior to conception of patients with connective tissue diseases leads to a reduction in maternal and perinatal complications.

011 - NON-INFECTIOUS AORTITIS: CLINICAL AND HISTOLOGICAL CORRELATION

Cristiana Sieiro Santos[1], Patricia Moya[2], Clara Moriano[1], Ivan Castellvi[2], Berta Magallares[2], Hye Sang Park[2], Helena Codes[2], Elvira Díez[1], Hector Corominas[2]
[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Reumatologia, Hospital de La Santa Creu I Sant Pau, Barcelona, Spain

Background: Isolated aortitis is considered a single organ vasculitis in the latest 2012 International Chapel Hill Consensus Conference revision. Despite being an increasingly diagnosed condition thanks to imaging tests, given the difficulty in completing the histological study, it is frequently unknown whether it is an isolated entity or a clinical manifestation of other vasculitis or rheumatologic entities.
Objectives: To describe the histological findings in samples of inflammatory aortic aneurysms and correlate them with their clinical presentation.
Methods: A descriptive cross-sectional study was performed. Aortic biopsies from elective aortic aneurysm surgery performed at a university hospital from 2019 to 2021 were reviewed. The samples with a non-infectious inflammatory pattern were selected. Cases of aortitis

secondary to infection and/or atherosclerosis were excluded. The samples were reviewed again by a pathologist specialized in vascular wall disease and classified according to the 2015 consensus on surgical pathology of the aorta of the Society for Cardiovascular Pathology and the European Association of Cardiovascular Diseases in granulomatous pattern, lymphoplasmacytic pattern and mixed inflammatory pattern. Likewise, the pathologist, taking into account the previous classification and the findings, assigned a diagnostic orientation, only taking into account the histological findings. Demographic, clinical, laboratory data and the diagnostic orientation of the rheumatologist were collected.

Results: Of the 116 aortic tissue samples reviewed, inflammatory findings were observed in 10 (9%) of the biopsies. Characteristics of the patients whose biopsies were included are shown in Table 1. 80% of the samples came from the proximal aorta, 10% from the aortic arch, and the remaining 10% from the abdominal aorta. The mean diameter of the aneurysm was 61.7 ± 21 millimeters. The rheumatologist's clinical diagnosis for the patients included was: 1 patient with Giant Cell Arteritis, 1 patient with Takayasu's disease, 1 patient with Behçet's disease, 1 patient with HLA-B27-associated spondyloarthropathy, and 6 patients with idiopathic aortitis. Histologically, 8 patients presented a lymphoplasmacytic pattern, 1 patient a granulomatous pattern, and 1 patient a mixed inflammatory pattern. The diagnostic orientation of the pathologist coincided with the clinician in 9/10 of the cases. The most frequent final diagnosis was nonspecific isolated aortitis (7/10), followed by Giant Cell Arteritis (1/10), Takayasu's arteritis (1/10) and Behçet's disease (1/10). There is a concordance between the histological pattern and the diagnostic orientation of the pathologist with the diagnostic orientation of the clinician in 9 of the 10 cases.

Conclusions: There is a high concordance between histopathologic and clinical diagnosis in the patients included in this study. Despite this, there is a high prevalence of non-specific aortitis, yet to be identified. Non-granulomatous lymphoplasmacytic pattern is the most reported in our series of cases.

TO 011 – TABLE 1.

Variables	N = 10
Male, n (%)	7 (70)
Age at diagnosis (years), median ± SD	69.4 ± 18
CV risk factors	
Smoking history	7 (70)
Diabetes	1 (10)
Dyslipidaemia	4 (40)
Arterial hypertension	4 (40)
Coronary Disease, n (%)	1 (10)
BMI (kg/m²), median ± SD	29.4±10.2
Treatment, n (%)	
Aspirin	1 (10)
Antihypertensive	5 (50)
Statins	4 (40)
Beta-blockers	3 (30)
Anticoagulant	1 (10)
Symptoms, n (%)	
Cranial symptoms	0 (0)
PMR	2 (20)
Constitutional symptoms	3 (30)
Thoracic pain	4 (40)
Analytical variables at baseline, median ± SD	
CRP (mg/L)	37.7±18.5
ESR (mm/h)	87.3±33
TG (mg/dL)	108.3±64
Cholesterol (mg/dL)	147.1±21
LDL (mg/dL)	95.8±24
HDL (mg/dL)	45.2±12

012 - NAILFOLD CAPILLAROSCOPY FOR PREDICTION OF NOVEL SEVERE ORGAN INVOLVEMENT IN SYSTEMIC SCLEROSIS

Cristiana Sieiro Santos[1], Ruben Rego Salgueiro[2], Clara Moriano[1], Carolina Álvarez[1], Elvira Díez[1]
[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Medicina Interna, ULS Guarda, Guarda, Portugal

Background: Nailfold capillaroscopy (NFC) has been suggested as a potential biomarker of disease severity in systemic sclerosis (SSc). Several studies report the association between capillary loss and disease severity however, the association of NFC abnormalities with novel severe organ involvement/progression in SSc has not been evaluated.

Objectives: We aim to evaluate the association of nailfold capillaroscopy (NFC) with novel major organ involvement/progression in SSc.

Methods: Follow-up data from patients with SSc registered between 2000 and 2022 were analysed. Patients underwent NFC at baseline. Novel severe organ involvement/progression was defined as new or progressive involvement of peripheral vasculature, lungs, heart, skin, gastrointestinal, kidney, musculoskeletal at 12 and 24 months of follow-up. The following NFC parameters were evaluated: capillary density, haemorrhages, enlarged and giant capillaries, avascular areas, organization of capillary architecture and scleroderma pattern (early/active/late). Logistic regression modelling was run to assess associations between NFC parameters and the occurrence of novel severe organ involvement and/or progression and risk factors.

TO 012 – TABLE 1. Associations between NFC and novel severe organ involvement/progression during follow-up

Novel severe involvement or progression	Loss of capillary density	P value	Haemorrhages	P value	Enlarged capillaries	P value	Avascular areas	P value	Scleroderma pattern	P value
Overall future organ involvement	3.21 (1.02-5.45)	0.002	1.82 (0.82-4.52)	0.82	1.09 (0.52-3.23)	0.72	2.1 (1.34-6.23)	0.03	1.82 (1.12-4.25)	0.03
Peripheral vascular involvement	1.7 (1.10-3.19)	0.03	1.72 (0.82-2.72)	0.62	1.92 (0.64-4.2)	0.52	2.62 (0.72-3.2)	0.72	1.82 (1.2-3.9)	0.04
New ILD	2.45 (1.32-4.23)	0.04	1.2 (0.52-3.21)	0.75	1.32 (0.72-4.21)	0.82	1.82 (1.12-3.42)	0.03	1.98 (1.32-3.42)	0.004
Progression of ILD	0.98 (0.23-2.14)	0.32	0.45 (0.14-1.98)	0.45	0.78 (0.21-2.34)	0.72	1.32 (1.10-3.52)	0.02	1.45 (1.12-3.82)	0.03
New PAH	0.72 (0.14-1.98)	0.42	0.32 (0.15-1.67)	0.62	0.34 (0.21-1.52)	0.65	0.85 (0.62-2.14)	0.73	0.92 (0.52-1.45)	0.62
Skin progression	1.42 (1.12-3.29)	0.01	1.32 (0.62-2.81)	0.34	0.72 (0.32-1.30)	0.39	1.19 (0.63-3.62)	0.06	2.3 (1.45-3.14)	0.04
Novel heart involvement	0.78 (0.42-1.42)	0.14	0.62 (0.32-1.32)	0.29	0.42 (0.29-1.82)	0.73	0.35 (0.13-1.51)	0.66	1.82 (0.42-2.37)	0.78
Novel GI involvement	1.62 (0.24-1.34)	0.24	1.54 (0.32-1.54)	0.43	0.48 (0.26-1.31)	0.32	0.42 (0.24-1.23)	0.39	1.83 (0.52-3.37)	0.40
Novel SRC	0.72 (0.34-1.32)	0.52	0.86 (0.42-1.52)	0.64	0.94 (0.52-2.02)	0.76	0.66 (0.23-1.87)	0.43	0.65 (0.44-1.32)	0.32
Novel musculoskeletal involvement	0.23 (0.10-0.42)	0.24	0.18 (0.09-0.39)	0.19	0.28 (0.14-0.54)	0.34	0.25 (0.11-0.43)	0.29	0.87 (0.50-1.42)	0.29

Results: 113 patients with SSc were included, 70 patients (61%) developed novel overall severe organ involvement/progression: 39 patients (56%) during the first 12 months and 31 patients (44%) from 12 to 24 months of follow-up. 11% of patients developed novel peripheral vascular involvement, 21% developed novel interstitial lung disease (ILD), 11% had progression of known ILD, 6% had novel pulmonary hypertension, 11% had skin progression, 10% had novel heart involvement, 10% had novel gastrointestinal involvement, 6% had scleroderma renal crisis and 13% had novel musculoskeletal involvement. Table 1 summarizes the associations between NFC and novel severe organ involvement/progression during follow-up. Loss of capillary density was associated with overall severe organ involvement (p 0.002), peripheral vascular involvement (p 0.03), new ILD (p 0.04) and skin progression (p 0.01); avascular areas were associated with overall severe organ involvement (p 0.03), new ILD (p 0.03) and progression of ILD (p 0.02) and scleroderma pattern was associated with overall severe organ involvement (p 0.03), peripheral vascular involvement (OR p 0.04), new ILD (p 0.004), progression of ILD (p 0.03) and skin progression (p 0.04).

Conclusions: NFC may be a potential biomarker in SSc for predicting novel severe organ involvement and/or progression. Abnormal capillary density, avascular areas and scleroderma pattern are predictors of overall severe organ involvement, peripheral vascular involvement, novel and progression of ILD and skin progression.

020 - PREVALENCE AND CLINICAL CHARACTERISTICS OF LATE ONSET AXIAL SPONDYLOARTHRITIS: RESULTS FROM A MULTICENTRE NATIONWIDE STUDY

Margarida Lucas Rocha[1], Rita Pinheiro Torres [2, 3], Sofia Ramiro[4], Alice Castro[5], Alice Neves[6], Ana Martins[7, 8], Ana Teodósio Chícharo[1], Beatriz Mendes[9], Carolina Ochôa Matos[10], Catarina Soares[11], Cláudia Miguel[6], C. Pinto Oliveira[12], Hugo Parente[11], José Melo Gomes[6, 13], Mariana Luis[9], Mariana Santos[2], Couto M[14], Miguel Bernardes[7, 8], Paula Valente[15], Roberto Costa[10], Sandra Sousa[5], Jaime C. Branco[2, 3], Fernando Pimentel-Santos[2, 3], Alexandre Sepriano[2, 3]

[1]Serviço de Reumatologia, Centro Hospitalar e Universitário do Algarve, Faro, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Lisboa Ocidental | Hospital Egas Moniz, Lisboa, Portugal, [3]Rheumatology Lab, NOVA Medical Research, Nova Medical School, Universidade Nova de Lisboa, Lisboa, Portugal, Lisboa, Portugal, [4]Department of Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [5]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [6]Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal, [7]Rheumatology Department, Centro Hospitalar Universitário de São João, Porto, Portugal, [8]Medicine Department / Rheumatology, Faculdade de Medicina da Universidade do Porto (FMUP); Rheumatology Service, Centro Hospitalar Universitário S. João (CHUSJ) - Porto, Porto, Portugal, [9]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [10]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa,

TO 020 – TABLE 1. Comparison of patient and disease characteristics between patients with late and early onset axSpA.

	All patients (N=2165)	Late onset axSpA age at symptom onset ≥ 45 years (N=273)	Early onset axSpA age at symptom onset <45 years (N=1892)	p-value*
Current age – years (mean, SD)	49.0 (12.8)	62.3 (8.8)	47.1 (12.2)	**<0.001**
Age at symptom onset - years (mean, SD)	31.7 (10.3)	51.4 (6.0)	28.9 (7.3)	**<0.001**
Age at diagnosis† - years (mean, SD)	38.0 (11.7)	55.1 (7.1)	35.5 (10.0)	**<0.001**
Diagnostic delay† - years (mean, SD)	6.3 (7.8)	3.7 (4.9)	6.6 (8.1)	**<0.001**
Symptom duration – years (mean, SD)	17.3 (11.6)	10.9 (6.9)	18.19 (11.9)	**<0.001**
Male gender (n, %)	1209 (56)	150 (55)	1059 (56)	0.749
Current Smokers$^{\Phi}$ (n, %)	393 (26)	22 (12)	371 (26)	**<0.001**
Current employment status$^{\Phi}$ (n, %)	1047 (67)	91 (48)	956 (70)	**<0.001**
Damage on X-SIJ (mNY)$^{\cup}$ (n, %)	1489 (85)	172 (82)	1317 (85)	0.284
HLA-B27† (n, %)	1346 (63)	136 (51)	1210 (65)	**<0.001**
Elevated CRP (ever)† (n, %)	1343 (63)	166 (62)	1177 (63)	0.073
Family history of SpA† (n, %)	289 (14)	21 (8)	268 (14)	**0.003**
Inflammatory back pain (ever)† (n, %)	1850 (87)	216 (81)	1634 (88)	**0.001**
Acute anterior uveitis (ever)† (n, %)	413 (19)	34 (13)	379 (20)	**0.003**
Good response NSAIDs (ever)† (n, %)	1169 (55)	141 (53)	1028 (55)	0.435
Peripheral arthritis (ever)† (n, %)	621 (29)	96 (36)	525 (28)	**0.010**
Heel enthesitis (ever)† (n, %)	444 (21)	56 (21)	388 (21)	0.976
Dactylitis (ever)† (n, %)	87 (4)	14 (5)	73 (4)	0.312
Psoriasis (ever)† (n, %)	54 (3)	7 (3)	47 (3)	0.930
IBD (ever)† (n, %)	45 (2)	7 (3)	38 (2)	0.542
BASDAI (0-10)$^{\theta}$ (mean, SD)	2.1 (2.5)	2.5 (2.8)	2.1 (2.5)	0.051
ASDAS$^{\Phi}$ (mean, SD)	2.1 (1.0)	2.2 (1.1)	2.1 (1.0)	0.208
BASFI (0-10)$^{\Phi}$ (mean, SD)	3.3 (2.5)	3.5 (2.9)	3.3 (2.8)	0.351
Treatment with csDMARDs (n, %)	524 (24)	64 (23)	460 (24)	0.754
Treatment with NSAIDs (n, %)	1059 (49)	135 (50)	924 (49)	0.850
Treatment with bDMARDs (n, %)	1662 (77)	213 (78)	1449 (77)	0.599

† Missing data <5%. θ Missing data <10%. Ʊ Missing data <25%. Φ Missing data <35%.
* Independent samples t-test for continuous variables and Chi2 for categorical variables.

Portugal, [11]Serviço de Reumatologia do Centro Unidade Local de Saúde do Alto Minho, Viana do Castelo, Portugal., Viana do Castelo, Portugal, [12]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [13]Clínica Dr. Melo Gomes, Lisboa, Portugal, [14]Unidade de Reumatologia, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [15]Rheumatology, Centro Hospitalar Entre o Douro e Vouga, Santa Maria da Feira, Portugal

Background/Purpose: Axial spondyloarthritis (axSpA) typically starts before the fourth decade of life. Consistent with that, the Assessment of SpondyloArthritis international Society (ASAS) classification criteria for axSpA should be applied only in patients with chronic back pain starting before 45 years of age. It has, however, been suggested that axSpA can sometimes start later in life with a distinctive phenotype, the so-called 'late onset axSpA' (lo-axSpA). There is, nevertheless, only limited data in support of the existence of such phenotype. We aimed to evaluate the occurrence of lo-axSpA and if these patients differ from those with early onset axSpA (eo-axSpA).
Methods: We performed a cross-sectional, multicentre, nationwide study using data from Reuma.pt, the Portuguese registry of patients with rheumatic diseases. Adult patients with the clinical diagnosis of axSpA, according to their treating rheumatologist, and with available information on the age of symptom onset were included. Lo-axSpA was defined as axSpA with a symptom onset ≥ 45 years of age. Demographic characteristics (e.g., age, gender, smoking status, and employment), SpA features [12 features (see Table) recorded as ever present, i.e., any time in the past or at the current study visit], measures of disease activity (ASDAS and BASDAI), disability (BASFI) and treatment with NSAIDs, csDMARDs and bDMARDs were compared between patients with lo-axSpA and eo-axSpA at the last available visit at the time of data extraction (13/12/2022).
Results: In total, 2165 patients with axSpA were included. The mean (standard deviation; SD) age at symptom onset was 32 (10) years and the mean (SD) symptom duration was 17 (12) years. The majority of the patients were male (56%), most had definite damage on pelvic radiographs according to the modified New York criteria (85%) and were treated with bDMARDs (77%). Out of the total 2165 patients, 273 (13%) had symptom onset ≥ 45 years and were therefore labelled as lo-axSpA. There were no differences in disease activity, disability or treatment between patients with lo-axSpA and eo-axSpA (Table). There were, however, some notable differences between the two groups. Patients with lo-axSpA were less often positive for HLA-B27 (51% vs 65%), less likely to have family history of SpA (8% vs 14%), acute anterior uveitis (13% vs 20%) and inflammatory back pain (81% vs 88%) than patients with eo-axSpA. On the contrary, patients with lo-axSpA had more peripheral arthritis (36% vs 28%) than patients with eo-axSpA.
Conclusion: This study shows that axSpA indeed starts before 45 years of age in the vast majority of the patients. Even though recall bias cannot be entirely ruled out, clinicians should however be aware that late-onset disease, though infrequent, may in some cases exist. This minority phenotype has a weaker association with HLA-B27, a lower probability of family history, inflammatory back pain or uveitis but more peripheral involvement.

023 - UVEITIS IN JUVENILE IDIOPATHIC ARTHRITIS AND THE IMPORTANCE OF THE TRANSITIONAL CARE

Rafaela Nicolau[1], Tiago Beirão[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Santos[4], Mariana Rodrigues[4], Iva Brito[4]
[1]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/Espinho, Gaia, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Unidade de Reumatologia Pediátrica e do Jovem Adulto, Centro Hospitalar e Universitário de São João, Porto, Portugal

Introduction: Uveitis is a serious extra-articular manifestation of juvenile idiopathic arthritis (JIA) associated with a risk of blindness. Evidence suggests that uveitis may persist up to adulthood in some cases, potentially causing severe visual impairment. We aimed to highlight the importance of pediatric rheumatologists and transitional care in preventing blindness due to JIA-associated uveitis (JIA-U).
Objectives: To evaluate the characteristics of ocular disease in patients with JIA-U who still exhibit uveitis in adulthood. Data on clinical features, treatment, complications and visual outcomes were collected.
Methods: We conducted a retrospective study on a series of patients aged 18 years or older with JIA-related active uveitis who had transitioned from pediatric to adult rheumatology in our Pediatric and Young Adult Rheumatology Unit. Data on clinical features, treatment, complications and visual outcomes were collected.
Results: 19 JIA-U patients were included (10 males, 9 females; median age 29 years, range 23–31 years). The onset of uveitis preceded the onset of arthritis in 7 (36.8%) patients and the median age of uveitis onset was 10 years (range 6.0–15.0 years). Oligoarticular JIA was present in 11 (52.6%) patients, enthesitis related JIA in 8 (42.1%) and poliarticular JIA in 1 (5.3%). 11 (57.9%) patients had anti-nuclear antibody (ANA) positivity, all with a homogeneous or speckled-pattern subtype, and

7 (36.8%) had HLA-B27 positivity. 16 (84.2%) had at least one uveitis flare during the transitional care, mainly anterior (89.5%) and bilateral (68.4%) uveitis. All patients were treated with topical glucocorticoid eye drops. Ten (52.6%) patients received methotrexate and 2 (10.5%) sulfasalazine. Monoclonal anti-TNF agents were administered in 11 (57.9%) cases while tocilizumab was used in 1 patient due to non-responsiveness to anti-TNF drugs. Two patients started Anti-TNF after transition. The most common ocular complications in our sample were cataract (68.4%), posterior synechiae (21.1%), band keratopathy (15.8%) and glaucoma (15.8%). Eleven (57.9%) patients underwent ophthalmic surgery for cataracts or glaucoma at an early age (≤ 18 years). One patient developed blindness after the transition period due to therapeutic noncompliance. 11 (57.9%) of patients were in remission for at least 2 years. An earlier onset of JIA-U was associated with the presence of ocular sequelae complications (p = 0.01).

Conclusion: The rate of complications of JIA-U has progressively declined over time due to extensive ophthalmological screening, highly effective treatments, and also due to an integrated multidisciplinary team which includes transition to adult care. This study highlights the importance of continuity of care for successful transitions, improving their health outcomes.

024 - GENDER DIFFERENCES IN CLINICAL MANIFESTATIONS OF PEDIATRIC BEHÇET'S SYNDROME: CROSS-SECTIONAL ANALYSIS IN A PORTUGUESE COHORT

Rafaela Nicolau[1], Tiago Beirão[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Santos[4], Mariana Rodrigues[4], Iva Brito[4]

[1]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/Espinho, Gaia, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Unidade de Reumatologia Pediátrica e do Jovem Adulto, Centro Hospitalar e Universitário de São João, Porto, Portugal

Introduction: Behçet's syndrome (BS) is a relapsing systemic disease with highest prevalence seen along the Silk Road. Similar to adult onset BS, pediatric onset is also seen equally in both genders. Gender differences in clinical manifestations have been reported, but scarce data are available.

Objectives: To characterize demographic and clinical features of a single-center cohort of portuguese BS patients with pediatric onset.

Methods: Longitudinal, retrospective, and descriptive study that included pediatric BS patients followed at our Pediatric and Young Adult Rheumatology Unit. Demographic and clinical data from medical records were collected retrospectively and the gender differences were evaluated.

Results: Twenty-four patients were included, mainly female patients (60.9%). Mean±SD age at symptom-onset and at diagnosis was 10.48±4.5 and 13.74±4, respectively. Those ages did not differ between two genders. All of the patients fulfilled International Criteria for BS, 18 (78.3%) Pediatric BS group criteria and 16 (69.6%) fulfilled International Behçet's Study Group criteria. No cases of familial history of BD were recorded. During disease course, oral aphtous ulcers (100%), genital ulcers (91%) and pseudofolliculitis lesions (47.8%) prevailed among the mucocutaneous signs,

TO 024 TABLE 1. GENDER DIFFERENCES OF CLINICAL MANIFESTATIONS

Table 1. Gender differences of clinical manifestations

Clinical findings	Females n (%)	Males n (%)	*p*
Oral aphthous ulcer	14 (100)	9 (100)	-
Genital ulcer	14 (100)	7 (77.8)	0.065
Ocular involvement	0(0)	4 (44.4)	**0.006**
Erythema nodosum	0(0)	2 (22.2)	0.065
Pseudofolliculitis lesions	6(42.9)	5(55.5)	0.552
Vascular involvement	0(0)	3(33.3)	**0.021**
Neurologic involvement	4(28.6)	0(0)	0.078
Gastrointestinal involvement	4(28.6)	3(33.3)	0.809
Joint involvement	1 (7.1)	4(44.4)	**0.034**
Cardiac involvement	0(0)	1(11.1)	0.202
HLAB-51 antigen Positivity	1 (7.1)	2 (22.2)	0.295

while the most common major organ-related manifestations were vascular and ocular involvement, with 7 (29.2%) cases uveitis including 3 (12.5%) cases of retinal vasculitis. Panuveitis (75%) was the predominant type. Vascular, ocular and joint involvement were significantly higher in males (p<0.001, p=0.021 and p=0.034, respectively). Although erythema nodosum (8.7%) and cardiac involvement (4.3%) were more common in males, the difference was not significant. Genital ulcers, pseudofolliculitis and gastrointestinal involvement were similar in two groups. Concerning the therapeutic approaches, conventional disease modifying anti- rheumatic drugs (cDMARDs) were the most frequently prescribed medication (91.3%), followed by colchicine (60.9%) and azathioprine (52.3%).
Conclusion: Our results are in line with what has been described in the literature. Ocular, vascular and joint manifestations had a higher frequency in pediatric males. This highlights importance of multidisciplinary monitoring to achieve better outcomes and avoid complications.

026 - THE IMPORTANCE OF THE TRANSITIONAL CARE IN PEDIATRIC BEHÇET'S SYNDROME: A RETROSPECTIVE SERIES OF 17 CASES

Rafaela Nicolau[1], Tiago Beirão[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Santos[4], Mariana Rodrigues[4], Iva Brito[4]
[1]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/Espinho, Gaia, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Unidade de Reumatologia Pediátrica e do Jovem Adulto, Centro Hospitalar e Universitário de São João, Porto, Portugal

Introduction: Behçets syndrome (BS) is characterized by relapses and remissions, and its management remains challenging due the wide clinical spectrum. Ocular involvement is one of the most common causes of morbidity, as well as neurological, vascular, and gastrointestinal involvement which may be life-threatening. Youth with rheumatic diseases often experience a gap in care or are lost to follow-up after transfer to adult rheumatology care. We aimed to highlight the importance of pediatric rheumatologists and transitional care in preventing comorbidities related to BS.
Objectives: To describe the transitional care experience in pediatric BS patients.
Methods: We conducted a retrospective study on a series of BS patients aged 18 years or older who had transitioned from pediatric to adult rheumatology and followed-up over a maximum 14-year period in our Pediatric and young adult Rheumatology Unit. Data on clinical features, treatment and complications were collected.
Results: 17 caucasian BS patients were included [11 females and 6 males; median age 26 years (IQR 24-34 years)]. Median age at symptom onset was 12 (IQR 8.5-14.5), while the median age at the diagnosis was 13 (IQR 12.5-17). HLA-B51 positivity was reported in 9 cases (52.9%). All patients presented oral apthous ulcers, 15 (88.2%) genital ulcers, 8 (47.1%) pseudofolliculitis and 2 (11.8%) erythema nodosum. Concerning major organ-related manifestations, articular (5 patients, 29.4%) and ocular involvement (3 patients, 17.6%) were the most common, while cardiac, gastrointestinal and neurological signs were present in only in 11.8%. 2 patients (11.8%) presented retinal vasculitis. Concerning the therapeutic approaches, colchicine was the most frequently prescribed medication (94.1%), followed by conventional disease modifying anti-rheumatic drugs(cDMARDs) (58.9%) - 40% methotrexate, 30% cyclosporine and 30% azathioprine. 2 patients were treated with Monoclonal Anti-TNF (Infliximab and Adalimumab). 3 patients received hypocoagulation (1 deep vein thrombosis, 1 venous CNS thrombosis, 1 intracardiac thrombosis). During the transitional care, 5 (29.4%) had at least one oral aphtous episode, 3 (17.6%) had recurrent arthralgias and 2 (11.8%) had pseudofollicutis episode. No ocular complications were reported. No patients were lost to follow-up.
Conclusion: Pediatric rheumatologists and transitional care play a major role in management of BS patients by improving the independence and skills of youth to meaningfully engage in their own care and ensure a good prognosis.

031 - PULMONARY INVOLVEMENT IN VERY EARLY SYSTEMIC SCLEROSIS (VEDOSS): REPORT FROM A SINGLE CENTER

Marcelo Neto[1], Ana Isabel Maduro[1], André Saraiva[1], JAP da Silva[1, 2], Tânia Santiago[1, 2], Maria João Salvador[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal

Background: Previous work has described interstitial lung disease (ILD) in very early systemic sclerosis (VEDOSS), but its progression over time is unknown. This can contribute decisively to the delineation of screening and monitoring strategies.
Objectives: To evaluate the prevalence of ILD and its course in a cohort of patients who persist in a VEDOSS state, followed in a tertiary systemic sclerosis unit, over 10 years.
Methods: Retrospective observational study of patients

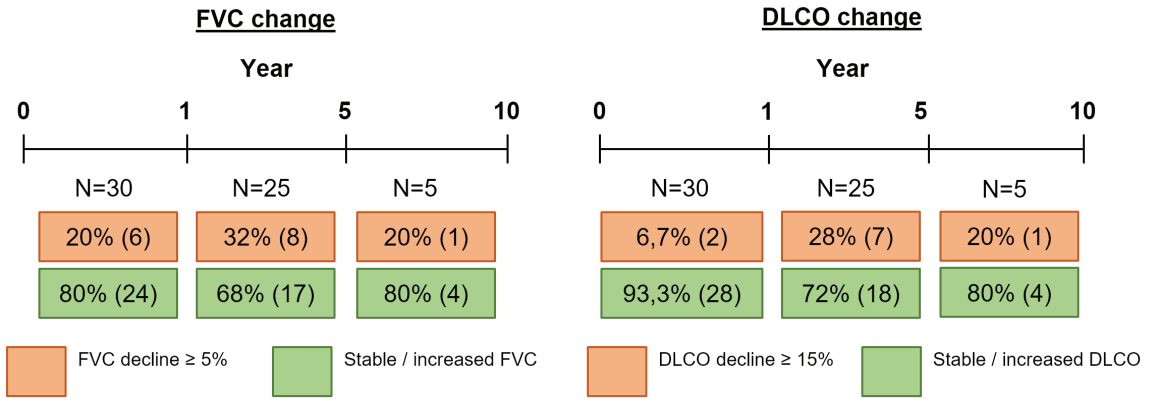
persistently fulfilling VEDOSS criteria, with sequential measurement of diffusing lung capacity for carbon monoxide (DLCO) and forced vital capacity (FVC), at 0, 1, 5 and 10 years. Chest CT at baseline was considered, when available. A DLCO or FVC <80% of predicted values was considered indicative of lung involvement. A decline of FVC or DLCO of ≥5% and ≥15% in any of the time intervals was considered clinically relevant. Demographic, clinical, and immunological variables at baseline were taken as potential independent variables, including age, gender, disease duration, arthritis, capillaroscopy pattern, and autoantibody profile. Pearson χ^2 test, Fisher's exact test or independent sample t-test were used for independent measures as appropriate. Repeated-measures ANOVA was used to compare variables over time.

Results: 30 patients (86,7% women, mean baseline age 46,9±12,87 yrs) were included; median time from first symptoms to diagnosis was 4,4 (IQR 7,6) yrs. At baseline, 4 patients had mildly decreased DLCO values (60-79% of predicted), results being normal in all remaining persons (Table 1). Every available baseline CT-scan (13/30 patients) was negative for ILD. During the first year of follow-up, a ≥5% decrease in FVC was observed in 6/30 patients (20%) and a DLCO decrease of ≥15% in 2/30 (6,7%). There was a non-significant trend for a higher probability of FVC and DLCO decrease in males vs females (M:50,0% vs F:15,3%F and M:25,0% vs F3,8%, respectively). None of the other variables was associated with FVC or DLCO decrease. Pulmonary tests were available for the 25 and 5 patients who reached 5 and 10 years of follow-up. No association was found between any of the clinical variables and significant change of FVC and DLCO at these time points. Mean values of FVC and DLCO consistently declined over time, without reaching statistical significance (Table 1). The incidence of significant change in FVC and DLCO between contiguous evaluations is represented in Figure 1. There was a non-significant trend for a higher probability of decline as follow-up time increased.

Conclusion: Our study provides data on the long-term assessment of FVC and DLCO in VEDOSS patients. There was a relevant decrease in FVC during the first year in 20% of the patients, and 20% had impaired DLCO (<80%) at 5 years, which suggests that a subset of patients who persist in VEDOSS state overtime still endure significant subclinical pulmonary involvement.

TO 031 – TABLE 1. FVC AND DLCO MEASUREMENTS AT BASELINE AND AT 1, 5 AND 10 YEARS

	Baseline (n=30)	1 year (n=30)	5 years (n=25)	10 years (n=5)	p-value
FVC (% predicted), mean ± SD	112,7±15,9	111,5±15,5	109,6±18,4	110,8±26,1	NS
FVC <80% predicted, % (n)	0 (0)	0 (0)	4,0 (1)	20,0 (1)	NS
DLCO (% predicted), mean ± SD	96,6±14,1	97,8±16,1	90,7±13,9	85,7±10,6	NS
DLCO <80% predicted, % (n)	13,3 (4)	16,7 (5)	20,0 (5)	20,0 (1)	NS



TO 031 – Figure 1. FVC and DLCO change at 1, 5 and 10 years

REFERENCES

Valentini G, Vettori S, Cuomo G, et al. Early systemic sclerosis: short-term disease evolution and factors predicting the development of new manifestations of organ involvement. Arthritis Res Ther 2012

Avouac J, Fransen J, Walker UA, et al. Preliminary criteria for the very early diagnosis of systemic sclerosis: results of a Delphi Consensus Study from EULAR Scleroderma Trials and Research Group. Ann Rheum Dis 2011

Hoffmann-Vold AM, Allanore Y, Alves M, et al. Progressive interstitial lung disease in patients with systemic sclerosis-associated interstitial lung disease in the EUSTAR database. Ann Rheum Dis 2021

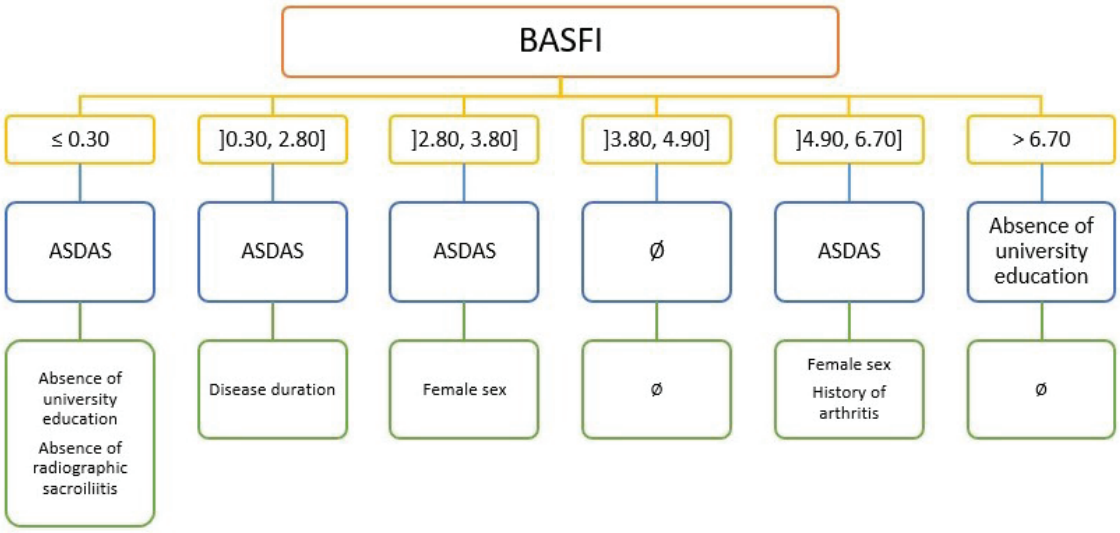
034 - HIERARCHY OF DETERMINANTS OF WORK IMPAIRMENT IN SPONDYLOARTHRITIS: DATA FROM THE ASSESSMENT OF SPONDYLOARTHRITIS INTERNATIONAL SOCIETY HEALTH INDEX (ASAS-HI) INTERNATIONAL VALIDATION STUDY

Frederico Rajão Martins[1], Pedro David Carvalho[2, 3], Désirée van der Heijde[4], Manoulk de Hooge[5, 6], Boonen A[7], J Braun[8], Uta Kiltz[8], Pedro M Machado[9, 10]

[1]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, Faro, Portugal, [2]Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Algarve Biomedical Center, Faro, Portugal, [4]Department of Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [5]Department of Rheumatology, Ghent University Hospital, Ghent, Belgium, [6]Molecular Immunology and Inflammation Unit, VIB-UGent Center for Inflammation Research, Zwijnaarde, Belgium, [7]Division of Rheumatology, Maastricht University Medical Center, Maastricht, Netherlands, [8]Rheumazentrum Ruhrgebiet, Ruhr-University Bochum, Herne, Germany, [9]Centre for Rheumatology & Department of Neuromuscular Diseases, University College London, London, United Kingdom, [10]Rheumatology, Northwick Park Hospital, London North West University Healthcare NHS Trust, London, United Kingdom

Background: Spondyloarthritis (SpA) is a disease of the working-age individual, with diverse economic and societal implications, including decreased employment rates. Our aim was to investigate the hierarchy of determinants contributing to work impairment in SpA.

Methods: Data was retrieved from the Assessment of SpondyloArthritis international Society (ASAS)-Health Index (HI) international validation study, a cross-sectional international observational study with a longitudinal component for reliability and responsiveness. Patients >60y reporting to be retired were excluded from the analyses (according to usual retirement ages across the 23 participating countries: www.oecd-ilibrary.org). The Work Productivity and Activity Impairment (WPAI) questionnaire was used to assess work productivity loss outcomes: absenteeism, presenteeism, overall work impairment, overall activity impairment. Initial univariable analyses using generalised estimated equations (GEE) explored the association between work-related outcomes (dependent variables) and sociodemographic and clinical variables (independent variables). A manual forward stepwise variable selection procedure was used for the selection of best-fit multivariable models. To avoid collinearity, separate models were built using ASDAS and BASDAI+CRP as independent variables. Lastly, a decision tree was built using Chi-square Auto-



TO 034 – Figure. Decision tree (CHAID) analysis for overall work impairment. ASDAS, axial spondyloarthritis disease activity score-C-reactive protein; BASFIm Bath ankylosing spondylitis function activity score; b-DMARD, biological disease-modifying antirheumatic drug; GEE, generalised estimated equations. Ø - no covariate included in this level of analysis.

TO 034 – TABLE. Best-fit multivariable model (adjusted B and 95%CI, p-values) for work productivity loss outcomes (GEE analysis), using ASDAS as independent variable

Covariates	Work productivity loss outcomes							
	Absenteeism	p-value	Presenteeism	p-value	Overall work impairment	p-value	Overall activity impairment	p-value
ASDAS-CRP	0.39 (0.13, 0.65)	0.003	0.44 (0.36, 0.52)	<0.001	0.39 (0.30, 0.49)	<0.001	0.32 (0.28, 0.36)	<0.001
BASFI	0.20 (0.10, 0.30)	<0.001	0.12 (0.09, 0.16)	<0.001	0.12 (0.08, 0.16)	<0.001	0.11 (0.10, 0.13)	<0.001
bDMARD use	-0.29 (-0.81, 0.23)	0.27	0.25 (0.10, 0.40)	0.001	0.19 (0.01, 0.37)	0.04	-0.01 (-0.09, 0.08)	0.86
History of uveitis	——	——	-0.34 (-0.50, -0.17)	<0.001	——	——	——	——
Disease Duration	——	——	-0.01 (-0.02, 0.002)	0.10	-0.01 (-0.02, -0.001)	0.03	-0.01 (-0.01, -0.004)	<0.001
Radiographic sacroiliitis	-0.63 (-1.12, -0.15)	0.01	-0.16 (-0.30, -0.02)	0.03	-0.29 (-0.46, -0.12)	0.001	——	——
Number of comorbidities	——	——	——	——	0.11 (0.06, 0.17)	<0.001	0.04 (0.01, 0.06)	0.003
BASDAI	0.20 (0.06, 0.33)	0.004	0.27 (0.23, 0.31)	<0.001	0.23 (0.18, 0.28)	<0.001	0.22 (0.20, 0.24)	<0.001
CRP	0.01 (0.01, 0.03)	0.05	0.001 (-0.004, 0.01)	0.71	0.01 (-0.001, 0.01)	0.09	0.001 (-0.001, 0.003)	0.31
BASFI	0.21 (0.10, 0.31)	<0.001	0.07 (0.03, 0.10)	<0.001	0.09 (0.05, 0.12)	<0.001	0.08 (0.06, 0.10)	<0.001
History of arthritis	0.33 (-0.14, 0.90)	0.17	——	——	——	——	——	——
Disease duration	——	——	-0.01 (-0.02, -0.01)	0.01	-0.01 (-0.02, -0.002)	0.02	——	——
bDMARD use	——	——	——	——	——	——	-0.10 (-0.17, -0.02)	0.02
NSAID use	——	——	——	——	0.27 (0.10, 0.45)	0.002	——	——
Radiographic sacroiliitis	-0.46 (-0.95, 0.02)	0.06	——	——	——	——	——	——
University education	——	——	-0.22 (-0.34, -0.09)	<0.001	——	——	-0.10 (-0.17, -0.02)	0.01

p-values in bold reflect significant associations. Variables tested in the univariable analyses and considered for multivariable models included: sex, age, symptom duration, disease duration, university education, history of arthritis, history of dactylitis, history of enthesitis, history of uveitis, history of psoriasis, history of inflammatory bowel disease, HLA-B27 status, CRP, NSAID use, cDMARD use, bDMARD use, number of comorbidities, radiographic sacroiliitis (modified New York criteria), anterior syndesmophytes (radiography), ASDAS-CRP, BASDAI and BASFI. Abbreviations: ASDAS, axial spondyloarthritis disease activity score-C-reactive protein; BASDAI, Bath ankylosing spondylitis disease activity index; BASFI, Bath ankylosing spondylitis function activity score; bDMARD, biological disease-modifying antirheumatic drug; GEE, generalised estimated equations; NSAID: non-steroidal anti-inflammatory drugs.

matic Interaction Detector (CHAID), a method of unbiased hierarchical multivariable analysis.

Results: From the original 1548 patients, a total of 1450 patients were included in the analysis, 345 of which had a follow-up reliability/responsiveness visit. Most patients were male (65%), mean age was 40 (±12) years and mean disease duration was 13 (±10) years. Most patients had axial SpA (84%) and a minority had peripheral SpA (16%). Medication use was: NSAIDs 64%, csDMARDs 26% and bDMARDs 38%. Most patients worked full-time (57.1%). Levels of absenteeism were 16% (±32%), presenteeism 29% (±26%), overall work impairment 39% (±34%), and overall activity impairment 41% (±29%). Worse physical function (measured by BASFI) and higher disease activity (measured by ASDAS or by BASDAI+CRP) were independently and consistently associated with all work productivity loss outcomes (Table). Other variables less consistently associated with work productivity loss outcomes were bDMARD and NSAID use, history of uveitis and peripheral arthritis, disease duration, presence of radiographic sacroiliitis, and level of education (Table). In the CHAID analysis (Figure), BASFI was the variable with higher discriminative power in predicting over-

all work impairment; ASDAS was the second-level discriminative variable. University education, disease duration, sex, radiographic sacroiliitis and history of arthritis were the third-level parameters. Similar results were observed for other work productivity loss outcomes (data not shown).

Conclusion: Loss of physical function and higher disease activity are major contributors to work productivity loss and are hierarchically superior to the contribution provided by other demographic and clinical variables.

036 - ADVERSE EVENTS OF METHOTREXATE USED IN RHEUMATOID ARTHRITIS IN CLINICAL PRACTICE: EXPERIENCE OF SINGLE CENTRE

Maria Pontes Ferreira[1], Hugo Parente[1], Catarina Soares[1], Sara Paiva Dinis [2], Anita Cunha[1], Francisca Guimarães[1], Soraia Azevedo[1], Daniela Peixoto[1], Filipa Teixeira[1], Carmo Afonso[1], Daniela Santos-Faria[1], José Tavares-Costa[1]

[1]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [2]Serviço de Reumatologia, Unidade Local de Saúde - Hospital Sousa Martins, Guarda, Portugal

Background: Methotrexate (MTX) is one of the most used conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs) in rheumatoid arthritis (RA). The commonest adverse event (AE) reported in the literature is raised liver enzymes, however long-term MTX use is rather favourable.[1]

Objective: To investigate the prevalence MTX's AE treatment for RA and to compare patients with and without AE.

Methodology: A retrospective single-centre observational study was performed including all RA patients, fulfilling 2010 ACR/EULAR classification criteria, who had Rheumatology appointment in our department during April 2022. Sociodemographic data, disease characteristics, disease activity based on Disease Activity Score 28 using C-reactive protein (DAS28-CRP), comorbidities, MTX dose, associated DMARDs, and prednisolone (PDN) dose were collected from clinical records. Adverse effects (AE) and its clinical/therapeutic consequences were also recorded. Severe AE (SAE) was defined according to the Common Terminology Criteria for Adverse Events as causing marked limitation in activity, or requiring medical intervention/therapy or hospitalization.

TO 036 – TABLE 1. Patient and disease characteristics at baseline and comparison between minor and severe adverse effects groups, and description of adverse effects and consequences.

	All patients N=93	No AE N=23	AE, N=68		p-value
			Minor AE N=33	Severe AE N=35	
Sociodemographic characteristics					
Age, years – mean±SD	61.1±14.6	62.1±14.2 62,7±16,8	60.8±16.0 58,8±15,1	NS	NS
Gender, Female – n/N (%)	56/93 (60.2)	15/25 (60.0) 21/33 (63.6)	41/68 (60.3) 20/35 (57.1)	NS	NS
Disease characteristics					
Age at diagnosis, years – mean±SD	54.3±15.8	58.5±14.2 56,94±16,68	55.3±16.0 53,64±15,41	NS	NS
Positive RF – n/N (%)	50/93 (53.8)	12/25 (48) 16/33 (48.5)	38/68 (55.9) 22/35 (62.9)	NS	NS
RF titre – mean±SD	248±242	266±272 303±310	242±235 184±111	NS	NS
Positive ACPA – n/N (%)	50/92 (54.3)	10/25 (40.0) 18/33 (54.5)	40/67 (59.7) 22/34 (64.7)	NS	NS
ACPA titre – mean±SD	213.5±22.2	114±82.72 333.7±296.4	230±235.0 126.4±73.2	NS	NS
Erosive disease – n/N (%)	21/93 (22.6)	4/25 (16.0) 6/33 (18.2)	17/68 (25) 11/35 (31.4)	NS	NS
Subcutaneous nodules – n/N (%)	6/93 (6.5)	1/25 (4.0) 2/33 (6.1)	5/68 (7.4) 3/35 (8.6)	NS	NS
Extra-articular manifestations – n/N (%)	3/93 (3.2)	0 2/33 (6.1)	4/68 (4.4) 1/35 (2.9)	NS	NS

continues on the next page

TO 036 – TABLE 1. Continuation

	All patients N=93	No AE N=23	AE, N=68		p-value
			Minor AE N=33	Severe AE N=35	
Comorbidities	45/93 (48.4)	14/25 (56) 14/33 (42.4)	31/68 (45.6) 17/35 (48.6)	NS	NS
Number of comorbidities – mean±SD	1.1±1.4	1.20±1.29 0,9±1,2	1.0±1.5 0,3±0,8	NS	NS
Hypertension – n/N (%)	26/93 (28.0)	8/25 (32) 8/33 (24.2)	18/68 (26.5) 10 (28.6)	NS	NS
Diabetes mellitus – n/N (%)	10/93 (10.8)	3/25 (12) 3/33 (9.1)	7/68 (10.3) 4/35 (11.4)	NS	NS
Dyslipidaemia – n/N (%)	23/93 (24.7)	6/25 (24) 7/33 (21.2)	17/68 (25) 10/35 (28.6)	NS	NS
Overweight/obesity – n/N (%)	7/8 (87.5)	2/2 (100) 1/2 (50)	5/6 (83.3) 4/4 (100)	NS	NS
Hepatic disorder – n/N (%)	6/93 (6.5)	1/25 (4) 2/33 (6.1)	4/68 (5.9) 2/35 (5.7)	NS	NS
Previous neoplasia – n/N (%)	5/93 (5.4)	1/25 (4) 1/33 (3)	4/68 (5.9) 3/35 (8.6)	NS	NS
Smoker/ Ex-smoker – n/N (%)	15/26 (57.7)	6/9 (66.7) 5/9 (55.6)	9/17 (52.9) 4/8 (50)	NS	NS
Moderate/heavy drinker – n/N (%)	8/19 (42.1)	3/7 (42.9) 3/6 (50)	5/12 (41.7) 2/6 (33.3)	NS	NS
Number of daily medications – mean±SD	3.0±**2.5**	3.7±2.9 2,8±**2,**1	2.66±2.32 1,3±**1,5**	NS	NS
At baseline of MTX					
MTX dose – mean±SD	15.9±2.6	16.1±2.03 15,8±**2,5**	15.83±2.8 16,7±**3,4**	NS	NS
MTX+other cDMARDs – n/N (%)	22/93 (23.7)	2/25 (8) 5/34 (14.7)	20/68 (29.4) 15/34 (44.1)	***0.015***	***0.050***
MTX+bDMARDs – n/N (%)	8/93 (8.6)	1/25 (4) 3/34 (8.8)	7/68 (10.3) 4/34 (11.8)	NS	NS
ESR (mm/h) – mean±SD	32±24	28±19 29±21	33.9±24.9 35±28	NS	NS
CRP (mg/dL) – mean±SD	2.9±6.9	1.6±1.6 4,4±11,0	3.4±8.0 2.0±1,8	NS	NS
DAS28-CRP – mean±SD	3.7±1.7	3.69±1.0 3.1±3.7	3.7±1.9 4.2±2.2	NS	NS
Adverse Effects					
Number of AE – mean±SD	2.0±1.9	- 2.3±1,4	2.8±1.6 1.3±0.5	-	-
MTX dose at AE (mg/week) – mean±SD	18.9±4.0	- 19,2±**4,**1	18.92±4.03 20,0±**3,**2	NS	-
Time until MTX suspension, years – mean±SD	3.1±2.9	- 3.5±4.2	3.5±3.5 3.4±2.7	NS	NS
Hospitalization – n/N (%)	2/93 (2.2)	- 0	2/64 (3.1) 2/35 (5.7)	NS	-
Consequence – n/N (%) – MTX dose eduction	23/93 (24.7)	-	23/68 (33.8) 8/33 (24.2)	15/35 (42.9)	- NS
Consequence – n/N (%) – MTX suspension	19/93 (20.4)	-	18/67 (26.9) 2/33 (6.1)	17/35 (48.6)	NS ***0.000***
General symptoms – n/N (%)				8 (8.6)	
Hepatic – n/N (%)				20 (21.5)	

continues on the next page

TO 036 – TABLE 1. Continuation

	All patients N=93	No AE N=23	AE, N=68		p-value
			Minor AE N=33	Severe AE N=35	
Haematological – n/N (%)				23 (24.7)	
Gastrointestinal – n/N (%)				23 (24.7)	
Cutaneous – n/N (%)				5 (5.4)	
Infections – n/N (%)				42 (45.2)	
Cardiovascular – n/N (%)				3 (3.2)	
Other – n/N (%)				3 (3.2)	

Legend: ACPA – Anti-citrullinated protein antibodies, AE – adverse effect, b/tsDMARDs – biological/targeted synthetic disease-modifying antirheumatic drugs (bDMARDs), cDMARDs – conventional disease-modifying antirheumatic drugs, CRP – C-reactive protein, DAS28-CRP – Disease Activity Score 28 using CRP, ESR – erythrocyte sedimentation rate, MTX – methotrexate, RF – rheumatoid factor, NS – not significant, n – number of patients, N – number of patients with available data. Categorical variables are presented as number/total population (percentage); continuous variables are presented as mean ± standard deviation.

SPSS V.25 was used for statistical analysis. Patients with and without adverse events were compared using Chi-squared test and student t-test/Mann-Whitney tests, as appropriate. Significant level was 2-sided<0.05.

Results: Ninety-three RA patients were enrolled in this study; 60.2% female, with a mean age of 61 years-old. Most had positive RF and ACPA (247.8±241.8 and 213.5±22.2, respectively), 22.6% had erosive disease, with a mean DAS28-CRP of 3.7 (±1.7) and a mean disease duration of 15.3 (±5.0) years. The mean dose of MTX was 15.9 (±2.6) mg/week, and at the last available appointment, the patients were on MTX for a mean of 3.1 (±2.9) years. Forty-eight percent of the patients had at least one comorbidity, namely overweight/obesity, smoking and drinking habits and hypertension. There were no differences concerning sociodemographic and clinical characteristics, namely disease activity, between the minor and severe AE groups, however, addition of a second csDMARD was associated with increased risk of AE and SAE (29.4%, p=0.049 and 44.1%, p=0.015, respectively).

A total of 188 AE were recognised in 68 out of 93 patients, with a prevalence rate of 73.1%. Of these, 76.6% were considered minor and 44 severe, identified in 33 and 35 patients, respectively. The most common AE identified were infections (45.2%), followed by haematological AE (24.7%, mostly anaemia) and gastrointestinal (GI) (24.7%, nausea being the most frequent GI AE reported). AE led to MTX dose reduction in 24.7% of patients, with higher proportion on the severe AE group (24.2% vs 42.9%). The suspension rate was 20.4%, significantly higher on the severe AE group (6.1 vs 48.6%, p<0.001).

Conclusions: Adverse events were common in our cohort, yet the majority was considered minor (76.6%), leading to definitive MTX suspension in 20.4% of the patients The most frequently reported AE was respiratory infections (39.8% of the patients). As expected, suspension rate was higher in patients with severe AE. Adding a second csDMARDs to MTX was associated to a higher risk of a more serious AE. Although most of the AE identified were minor, and causality may be difficult to ascertain, the prevalence of AE was rather high in this cohort, alerting for the need of adequate monitoring of patients treated with MTX.

REFERENCES:

1. Salliot C et al. Long-term safety of methotrexate monotherapy in patients with rheumatoid arthritis: a systematic literature research. Ann Rheum Dis. 2009

039 - SYNOVITIS, ACNE, PUSTULOSIS, HYPEROSTOSIS AND OSTEITIS (SAPHO) SYNDROME: A PORTUGUESE CASE SERIES

Mariana Diz-Lopes [1, 2], Teresa Martins-Rocha[1, 2], Carlos Marques Gomes[1, 2], Lúcia Costa[1]

[1]Rheumatology Department, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Department of Medicine, Faculty of Medicine, University of Porto, Porto, Portugal

Introduction: Synovitis, Acne, Pustulosis, Hyperostosis and Osteitis (SAPHO) syndrome is a chronic inflammatory disease with cutaneous and osteoarticular manifestations, first described in 1987. It is controversial whether SAPHO represents a distinct entity or should be considered part of the spondyloarthritis spectrum. Diagnostic criteria have been proposed but need further validation. The "bull head" sign in bone scintigraphy is highly specific. Given its rarity and wide range of manifestations, several case series have been published, increasing knowledge about these patients.

Objectives: To describe the characteristics of patients with SAPHO syndrome in a portuguese center.

Methods: We conducted a cross-sectional descriptive single-center study including patients with the diagnosis of SAPHO syndrome. Sociodemographic, clinical, scinti-

TO 039 – TABLE 1. Sociodemographic, clinical, scintigraphic and treatment data of the SAPHO syndrome patients (n=11).

Variable	
Sociodemographic data	
Sex	
Male, n (%)	3 (27,3)
Female, n (%)	8 (72,7)
Age at diagnosis (years), mean (SD)	47 +/- 12,2
Smoking habit, n (%)	5 (45,5)
Clinical Features	
First symptom	
Skin, n (%)	7 (63,6)
Osteoarticular, n (%)	1 (9,1)
Skin + Osteoarticular, n (%)	3 (27,3)
Cutaneous involvement, n (%)	10 (90,9)
Palmoplantar Pustulosis, n (%)	9 (90)
Acne, n (%)	1 (10)
Hidradenitis Suppurativa, n (%)	2 (20)
Osteoarticular Involvement, n (%)	11 (100)
Anterior chest wall, n (%)	11 (100)
Spine, n (%)	7 (63,6)
Mandible, n (%)	1 (9,1)
Sacroiliac joints, n (%)	6 (54,5)
Peripheral joints, n (%)	7 (63,6)
Focal infection, n (%)	2 (18,2)
Type of infection	Acute tonsillitis, n=1 Dental infection, n=1
Comorbidities	
Other inflammatory systemic diseases, n (%)	2 (18,2)
Inflammatory Bowel Disease, n (%)	0
Bone Scintigraphy performed, n (%)	9 (81,8)
Bull Head sign, n (%)	5 (55,6)
Increased tracer uptake in sternocostoclavicular joints, n (%)	9 (100)
Increased tracer uptake in sacroiliac joints, n (%)	4 (44,4)
Increased tracer uptake in peripheral joints, n (%)	5 (55,6)
Treatment	
NSAIDs, n (%)	10 (90,9)
Glucocorticoids, n (%)	4 (36,4)
Biphosphonates (Pamidronate), n (%)	5 (45,5)
csDMARDs, n (%)	6 (54,5)
Methotrexate, n (%)	4 (66,7)
Sulfasalazine, n (%)	2 (33,3)
bDMARDs, n (%)	3 (27,3)
TNF-alpha inhibitors, n (%)	2 (66,7) Adalimumab, n=1 Etanercept, n=1
IL-12/IL-23 inhibitors, n (%)	1 (33,3) Ustekinumab, n=1
Effect of the treatment	
Overall improvement, n (%)	9 (81,8)
Osteoarticular improvement	9 (81,8)
With NSAIDs alone, n (%)	3 (33,3)
Cutaneous improvement	9 (90)

graphic and treatment data were collected from medical records. Descriptive analysis was performed with mean and standard deviation for continuous data, and frequency counts and percentages for categorical variables.

Results: A total of 11 SAPHO patients were included (Table 1). Most patients (72,7%) were women, with a mean age at diagnosis of 47 years (+/-12,2). Almost half were smokers (45,5%). A focal infection before symptoms started was identified in 2 patients (acute tonsillitis, dental infection).

Regarding clinical features, osteoarticular involvement was present in all patients and 10 had skin manifestations. Of note, 7 patients presented initially with cutaneous manifestations. Of the patients with skin involvement, 90% had palmoplantar pustulosis (PPP). Acne and hidradenitis suppurativa were present in 18,2% (n=2) of patients, respectively. Anterior chest wall (ACW) involvement was the most common osteoarticular manifestation, being present in all patients. Involvement of spine, sacroiliac and peripheral joints was reported in 7 patients (63,6%).

Investigation with bone scintigraphy was performed in 81,8% of patients, and the "bull head" sign (uptake in manubrium, bilateral sternoclavicular joints and clavicular) was present in 5 (55,6%). Increased tracer uptake was more commonly observed in the sternocostoclavicular joints (n=9), followed by peripheral (n=5) and sacroiliac joints (n=4).

Of the 11 patients, 10 (90,9%) were treated with non-steroidal anti-inflammatory drugs (NSAIDs), 4 (34,6% received glucocorticoids, 5 (45,5%) biphosphonates, 6 (54,5%) conventional DMARDs (csDMARDs) and 3 (27,3%) were started on biologic DMARDs (bDMARDs). Overall, 9 (81,8%) patients had a clinical improvement in osteoarticular and cutaneous manifestations. NSAIDs alone were effective in only 3 patients (33,3%), with most requiring additional therapy.

Conclusion: SAPHO syndrome often presents with skin involvement, simultaneously or followed by osteoarticular manifestations. Along with existing evidence, middle-aged women were the most affected. Focal infection before the onset of symptoms can be identified and is described to be associated with the development of PPP. ACW involvement is highly characteristic and was present in all patients. Scintigraphic "bull head" sign is very specific, but its sensitivity is reported to be far from ideal. In our series, only half of the patients had this scintigraphic pattern. The lack of NSAIDs response observed could be related with the high prevalence of women as well as peripheral joints and ACW involvement, as described in other studies. A trial of biphosphonates and csDMARDs are reasonable treatment options and bDMARDs are reserved for patients with a refractory disease.

040 - MULTICENTER ASSESSMENT OF ACRO-OSTEOLYSIS IN PORTUGUESE PATIENTS WITH SYSTEMIC SCLEROSIS

Beatriz Samões[1], Diogo Guimarães da Fonseca[1], Tiago Beirão[1], Flávio Campos Costa[1], Romana Vieira[1], Georgina Terroso[2], Raquel Miriam Ferreira[2], Rafaela Nicolau[3], André Saraiva[4], Maria João Salvador[4], Ana Catarina Duarte[5], Ana Cordeiro[5], Joao Paulo Vilas-Boas[6], Inês Genrinho[3], Ana Bento da Silva[7, 8], Laura Gago[7, 8], Catarina Resende[9], Patrícia Martins[9, 10], Nathalie Madeira[11], Sara Paiva Dinis [11], Couto M[3], Inês Santos[3], Filipe Araújo[12], Ana Filipa Mourão[7, 8], Miguel Guerra[13], Margarida Oliveira[13], Alexandra Daniel[14], Marília Rodrigues[14], Catarina Soares[15], Hugo Parente[15], Carolina Furtado[16], Tomás Fontes[16], Joana Aleixo[1]
[1]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [2]Rheumatology Department, Centro Hospitalar Universitário de São João, Porto, Portugal, [3]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [4]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal, [5]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [6]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [7]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [8]Comprehensive Health Research Center – CHRC, Centro de Estudos de Doenças Crónicas (CEDOC), NOVA Medical School, Faculdade de Ciências Médicas, Lisboa, Portugal, Lisboa, Portugal, [9]Rheumatology Department, Centro Hospitalar Universitário de Lisboa Norte, Lisboa, Portugal, [10]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal, [11]Rheumatology Department, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [12]Hospital Ortopédico de Sant 'Ana, Cascais, Portugal, [13]Rheumatology Department, Centro Hospitalar e Universitário Cova da Beira, Covilhã, Portugal, [14]Serviço de Reumatologia, Centro Hospitalar de Leiria, Leiria, Portugal, [15]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [16]Rheumatology Department, Hospital do Divino Espírito Santo, Ponta Delgada, Portugal

Background: Acro-osteolysis is a disabling manifestation of Systemic Sclerosis (SSc). Few is known about its prevalence and clinical associations.
Objectives: To define the prevalence of acro-osteolysis among Portuguese SSc patients, to determine the most sensitive radiological location for its diagnosis and to clarify the phenotype of SSc patients with acro-osteolysis.
Methods: A cross-sectional multicenter study was conducted evaluating SSc patients from 14 Portuguese centers, registered in the Rheumatic Diseases Portuguese Registry (Reuma.pt), that fulfilled Leroy/Medsger 2001 or ACR/EULAR classification criteria for SSc. The presence of acro-osteolysis was assessed through systematic plain radiographs of hands and feet. Statistical analysis with independent parametric or non-parametric tests, multivariate logistic regression of the significant variables in univariate analysis and sensitivity calculation of each radiographed site for the diagnosis of acro-osteolysis were performed.
Results: We included 195 patients, of whom 167 (85.6%) were female, with a median [min, max] age of 64 [20, 90] years-old and a median [min, max] disease duration of 11.2 [2, 57] years. Regarding disease classification, 146 (75.3%) patients had limited SSc, 32 (16.5%) had diffuse SSc, 9 (4.6%) had sine scleroderma SSc and 7 (3.6%) had early SSc. Acro-osteolysis was present in 62 (31.8 %) patients, with the hands (n=49, 25.1%) being more frequently affected than the feet (n=22, 11.3%). The most sensitive location to detect acro-osteolysis was the hand (79.0%). The presence of overall acro-osteolysis was significantly associated with digital ulcers (p=0.001), flexion contractures (p=0.031), oesophageal (p=0.006), gastric (p=0.014) and lung (p=0.004) involvements, higher mRSS score (p<0.001), anti-topoisomerase I positivity (p=0.034) and radiological calcinosis (p=0.018). Patients with hand acro-osteolysis had longer disease duration (p=0.02) and were more often affected by digital ulcers (p=0.004), flexion contractures (p=0.041), oesophageal (p=0.001), gastric (p=0.015) and lung (p=0.001) involvements, higher mRSS score (p<0.001), anti-topoisomerase I positivity (p=0.004) and radiological calcinosis (p=0.008). No significant differences were found between patients with and without feet acro-osteolysis. In multivariate analysis, anti-topoisomerase I positivity (OR 4.6, 95%CI 1.3-16.4, p=0.017) was predictor of hand acro-osteolysis.
Conclusions: The prevalence of acro-osteolysis found in Portuguese SSc patients is within the range found in the literature (20-40%).1 The hands were the most sensitive location to detect acro-osteolysis – a novel aspect since, to our knowledge, this is the first study evaluating acro-osteolysis in the feet besides the hands. Our data replicated some previously reported associations between acro-osteolysis and other hand involvements (digital ulcers, calcinosis and flexion contractures) and systemic complications (esophagogastric and pulmonary systems).2-4 Anti-topoisomerase I positivity appears to increase the risk for hand acro-osteolysis.

REFERENCES:

1. PMID: 31812353; [2] PMID: 33427611; [3] PMID: 31214867; [4] PMID: 22923763

041 - TNF INHIBITORS AND PREGNANCY: A RETROSPECTIVE MULTICENTRIC STUDY IN SPONDYLARTHRITIS PATIENTS

Tiago Beirão[1], Rafaela Nicolau[2], Inês Santos[2], Francisca Guimarães[3], Francisca Aguiar[4], Nádia Martins[2], Sara Ganhão[4], Mariana Rodrigues[4], Anabela Rocha[5], Sofia Bessa[5], Iva Brito[4]

[1]Rheumatology Department, Centro Hospitalar Vila Nova de Gaia/Espinho, Gaia, Portugal, Vila Nova de Gaia, Portugal, [2]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Unidade de Reumatologia Pediátrica e do Jovem Adulto, Centro Hospitalar e Universitário de São João, Porto, Portugal, [5]Ginecologia e Obstetrícia, Centro Hospitalar de São João, Porto, Portugal

Introduction: Tumor necrosis factor (TNF) inhibitors are effective treatments in spondylarthritis patients. In the last years, their use during pregnancy is increasing due to higher experience in the area. However, there is still limited data on the effects of these medications on neonatal outcomes.

Objectives: The aim of this study is to determine neonatal outcomes in patients with spondylarthritis treated with TNF inhibitors.

Methods: This multicentric Portuguese retrospective study between 2020 and 2022, analysed 50 pregnancies of spondylarthritis patients and their exposure to TNF inhibitors. Statistical analysis was performed to compare the outcomes between the two groups.

Results: In total, 16 out of 50 patients were treated with TNF inhibitor throughout pregnancy (1 with adalimumab and 15 with certolizumab). Regarding treatment, the TNF inhibitor group had a lower percentage of prednisolone prescription, although not statistically significant (9.10% vs 29.40%, p=0.065). Whilst no preterm births were recorded in this group (0.0% vs 13.3%), the only case of foetal malformation and intrauterine growth restriction was in a patient treated with TNF inhibitor(certolizumab). 1 case was admitted to the neonatal intensive care unit. No statistically significant difference was found on the gestational age at birth and birth weight.

Conclusion: There was no statistically significant association between the use of TNF inhibitors during pregnancy andadverse neonatal outcomes in spondyloarthritis patients. However, this study was limited by its retrospective design and small sample size. Further research is needed to confirm these results and investigate potential long-term effects of TNF inhibitor treatment during pregnancy.

	TNF inhibitor	Not TNF inhibitor	Total	p-score
Patient Characteristics				
Age (years)	32.71±4.77 (22-31)	33.87±4.32 (25-42)	33.90±4.43 (22-42)	0.778
Months since disease onset	66.42±40.80 (12-168)	50.10±36.08 (5-132)	58.10±41.36 (2-182)	0.114
Previous pregnancies	1.00±0.81 (37-40)	1.54±1.12 (0-4)	1.20±1.16 (0-4)	0.020
Previous miscarriage	2/16 (22.20%)	7/32 (21.90%)	9/48(18.80%)	0.433
Preconception				
Preconception consultation	11/14 (36.70%)	19/31 (63.30%)	30/45(66.70%)	0.255
bDMARD started in preconception	6/16 (37.50%)	3/34 (8.80%)	9/50(18.00%)	0.014
Treatments				
Prednisolone	1/16 (9.10%)	10/34 (29.40%)	11/50 (22.00%)	0.065
Sulfasalazine	2/16 (22.20%)	7/34 (20.60%)	9/50 (18.00%)	0.487
Pregnancy outcomes				
Flare after pregnancy	2/13 (15.40%)	3/31 (9.70%)	5/44 (11.40%)	0.586
Flare during pregnancy	1/14 (7.10%)	9/32 (28.10%)	10/46 (21.70%)	0.112
Successful pregnancy	12/16 (75.00%)	30/34(88.20%)	42/50(84.00%)	0.234
Birth outcomes				
Gestational age (weeks)	38.64±0.81 (37-40)	38.10±1.37 (35-40)	32.60±13.54 (2-41)	0.746
Weight (grams)	3076.27±391.93 (2230-3500)	3302.96±433.33 (2660-4340)	2656.20±1310.60 (2-4340)	0.115
Foetal malformation	1/12 (8.30%)	0/29 (0.00%)	1/41 (2.40%)	0.116
Preterm birth	0/12(0.00%)	4/30(13.30%)	4/42(9.50%)	0.184
Intrauterine growth restriction	1/30(3.30%)	0/12(0.00%)	1/42(2.40%)	0.522

TO 041 – Figure 1. Comparison between TNFi and not TNFi

042 - HLA-B51 AND CLINICAL MANIFESTATIONS IN JUVENILE BEHÇET´S SYNDROME

Tiago Beirão[1], Rafaela Nicolau[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Ganhão[4], Mariana Rodrigues[4], Iva Brito[4]

[1]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [2]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Unidade de Reumatologia Pediátrica, Centro Hospitalar de São João, Porto, Portugal

Introduction: Behçet's syndrome (BS) is a rare, chronic, multisystem inflammatory disorder of unknown etiology. The age of onset is typically between 20 and 30 years, but 15-20% of patients have onset before the age of 16 (juvenile BS). The human leukocyte antigen (HLA) B51 has been linked to BS in several populations[1]. However, the association between HLA-B51 and clinical manifestations in JBD is not well established.

Objectives: The objective of this study is to investigate the association between HLA-B51 and clinical manifestations in JBS.

Methods: We conducted a retrospective cohort study

TO 042 – TABLE.

	Positive HLA-B51	Negative HLA-B51	Total	p-score
Patient Characteristics				
Sex (male)	5/10 (50.00%)	1/5 (20.00%)	6/15 (40.00%)	0.264
Race (Caucasian)	10/10 (100.00%)	5/5 (100.00%)	15/15 (100.00%)	-
Age first symptoms (years)	11.70±3.71 (3-15)	7.00±3.54 (3-12)	10.13±4.21 (3-15)	0.036
Age diagnosis (years)	13.90±4.41 (5-21)	11.40±2.30 (8-13)	13.07±3.94 (5-21)	0.137
Time obtaining diagnosis (years)	2.20±1.75 (0-6)	4.40±4.27 (1-10)	2.93±2.89 (0-10)	0.489
Clinical manifestations				
Oral ulcers	10/10 (100.00%)	5/5 (100.00%)	15/15 (100.00%)	-
Genital ulcers	9/10 (90.00%)	5/5 (100.00%)	14/15 (93.33%)	0.464
Pseudofolliculitis	6/10 (60.00%)	2/5 (40.00%)	8/15 (53.33%)	0.464
Erythema nodosum	1/10 (10.00%)	0/5 (0.00%)	1/15 (6.67%)	0.464
Uveitis	3/10 (30.00%)	0/5 (0.00%)	3/15 (20.00%)	0.171
Retinal vasculitis	2/10 (20.00%)	0/5 (0.00%)	2/15 (13.33%)	0.283
Cardiac manifestations	0/10 (0.00%)	0/5 (0.00%)	0/15 (0.00%)	-
Neurologic manifestations	2/10 (20.00%)	0/5 (0.00%)	2/15 (13.33%)	0.283
Vascular manifestations	2/10 (20.00%)	0/5 (0.00%)	2/15 (13.33%)	0.283
Articular manifestations	2/10 (20.00%)	2/5 (40.00%)	4/15 (26.67%)	0.409
Gastrointestinal manifestations	0/10 (0.00%)	2/5 (40.00%)	2/15 (13.33%)	0.032
Treatments				
Corticosteroids	7/10 (0.00%)	3/5 (60.00%)	10/15 (66.67%)	0.699
Colchicine	10/10(100.0%)	5/5 (100.00%)	15/15 (100.00%)	-
cDMARDs	9/10(90.00%)	1/5 (20.00%)	10/15 (66.67%)	0.007
bDMARDs	1/10(10.00%)	0/5 (0.00%)	1/15 (6.67%)	0.464
Hypocoagulation	2/10(20.00%)	0/5 (0.00%)	2/15 (13.33%)	0.283

of 15 JBD patients followed in a tertiary hospital. HLA-B51 genotyping was performed using a PCR-based method. We compared the clinical manifestations and treatments between HLA-B51 positive and negative patients.

Results: Of the 15 patients, 10 (66.67%) were HLA-B51 positive. The HLA-B51 positive group had a later disease onset compared to the HLA-B51 negative group (mean age: 11.70 vs. 7.00 years, p=0.036). The HLA-B51 positive group also had a lower frequency of GI manifestations compared to the HLA-B51 negative group (0.00% vs. 40.00%, p=0.032) and a higher frequency of use of cDMARDs (90.00% vs. 20.00%, p=0.007).

Conclusion: Our study suggests that HLA-B51 is associated with a later disease onset, fewer GI manifestations, and increased use of cDMARDs in JBD patients. These findings may have important clinical implications for the diagnosis and management of JBD, especially in populations with a high prevalence of HLA-B51. The study with highest number of patients linked HLA-B51 with genital ulcers, ocular involvement and skin manifestations[2]. Further studies with larger sample sizes and more diverse populations are needed to confirm these results and investigate the underlying mechanisms of the observed associations.

043 - PRECONCEPTION COUNSELING IMPACT IN PREGNANCY OUTCOMES IN PATIENTS WITH SPONDYLARTHRITIS

Tiago Beirão[1], Rafaela Nicolau[2], Inês Santos[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Ganhão[4], Mariana Rodrigues[4], Nádia Martins[2], Anabela Rocha[5], Sofia Bessa[5], Iva Brito[4]

[1]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [2]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Paediatric and Young Adult Rheumatology Unit, Centro Hospitalar Universitário de São João, Porto, Portugal, [5]Ginecologia e Obstetrícia, Centro Hospitalar de São João, Porto, Portugal

Purpose: Spondyloarthritis (SpA) is a group of chronic inflammatory diseases, often affecting women in reproductive age. These diseases can have a significant impact on the reproductive health of women. Preconception counseling and medication adjustments have shown to reduce flares and improve pregnancy outcomes in women with rheumatoid arthritis. However, in women with SpA data of the impact of preconception counselling on pregnancy outcomes is scarce. The aim of this study is to evaluate that.

Methods: In this retrospective multicentric study, data was collected from medical records of women who gave birth from 2020 to 2022. The study included 45 pregnancies, which were divided into two categories whether they received preconception consultation or not. Data was collected on patient characteristics, disease duration, medications used, and preconception counselling. Outcomes were divided into two groups: maternal and fetal outcomes.

Results: 30 out of 45 pregnancies (66.67%) had received preconception counselling, having a significantly lower percentage of flares occurring after pregnancy compared to the non-counselling group (36.6% vs 6.4%, p=0.031) and lower percentage of contraindicated medication during pregnancy (20.0 vs 0.0%, p=0.011).

Conclusion: Preconception counselling in women with SpA can increase the likelihood of medication adjustments before pregnancy and decrease the occurrence of flares after pregnancy. These findings suggest that preconception counselling should be implemented in the management of pregnant women with SpA to improve pregnancy outcomes. Further studies are needed to confirm the effectiveness of preconception counselling and to determine the optimal approach.

TO 043 – TABLE.

	Preconception counselling	No preconception counseling	Total	p-score
Patient Characteristics				
Age (years)	33.43±4.34 (22-41)	33.67±4.81 (27-42)	33.90±4.43 (22-42)	0.990
Months since onset disease	61.93±39.19 (12-168)	42.47±32.95 (5-132)	58.10±41.36 (2-182)	0.177
Previous pregnancies	1.20±1.10 (0-4)	1.73±0.96 (1-4)	1.20±1.16 (0-4)	0.072
Previous abortion	6/23 (26.09%)	3/14(21.43%)	9/43(20.93%)	0.955
Preconception				
Pregnancy with contraindicated medication	0/30 (0.00%)	3/15 (20.00%)	3/45 (6.67%)	0.011
bDMARD started in preconception	5/30 (16.67%)	2/15 (13.33%)	7/45 (15.56%)	0.771
Medication				
Prednisolone	6/30 (20.0%)	4/15 (26.67%)	10/45 (22.22%)	0.612
Sulfasalazine	2/30 (6.67%)	7/15 (46.67%)	9/45 (20.0%)	0.002
Certolizumab	10/30 (33.3%)	1/15 (6.67%)	11/45 (24.44%)	0.050
Pregnancy outcomes				
Flare during pregnancy	4/28 (14.29%)	5/15 (33.33%)	9/43 (20.93%)	0.143
Flare after pregnancy	1/26 (3.85%)	4/15 (26.67%)	5/41 (12.19%)	0.031
Successful pregnancy	24/30(80.0%)	15/15(100%)	39/45(86.67%)	0.063
Birth outcomes				
Gestational age	38.13±1.33 (35-40)	38.46±1.13 (36-40)	32.60±13.54 (2-41)	0.087
Newborn Weight (grams)	3243.65±439.45 (2330-4340)	3227.67±428.37 (2520-4200)	2656.20±1310.60 (2-4340)	0.288
Fetal malformation	1/23 (4.35%)	0/15 (0.00%)	1/38 (2.63%)	0.413
Preterm birth	3/24 (12.50%)	1/15 (6.67%)	4/39 (10.26%)	0.559
Intrauterine growth restriction	0/24 (0.00%)	1/15 (6.67%)	1/39 (2.56%)	0.200

045 - SPONDYLOARTHROPATHY AND PREGNANCY: A RETROSPECTIVE MULTICENTRIC STUDY

Tiago Beirão[1], Inês Santos[2], Rafaela Nicolau[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Ganhão[4], Mariana Rodrigues[4], Nádia Martins[2], Anabela Rocha[5], Sofia Bessa[5], Iva Brito[4]

[1]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [2]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [3]Pediatrics, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Unidade de Reumatologia Pediátrica, Centro Hospitalar de São João, Porto, Portugal, [5]Ginecologia e Obstetrícia, Centro Hospitalar de São João, Porto, Portugal

Purpose: Spondyloarthropathy is a group of rheumatologic diseases that affect the axial skeleton and occasionally peripheral joints. The impact of different subtypes of spondyloarthropathy on pregnancy outcomes is not well understood. The purpose of this retrospective multicentric study was to evaluate that.

Methods: A retrospective study in several rheumatology centres in different regions of Portugal, with inclusion of 50 pregnant women with previous spondyloarthropathy diagnosis, which were divided into five categories according the spondyloarthropathy subtypes: ankylosing spondylitis, non-radiographic spondyloarthritis, psoriatic arthritis, enteropathic arthritis, and undifferentiated spondyloarthropathy. Data on patient characteristics, medication use, pregnancy outcomes, and fetal outcomes were collected and compared between the five spondyloarthropathy subgroups.

Results: The most frequent disease ankylosing spondylitis (38.0%). Among the five spondyloarthropathy subgroups, ankylosing spondylitis and non-radiographic spondyloartritis were the ones with longer disease duration. Sixty percent of the women had preconception consultation, with the highest prevelance observed in the psoriatic arthritis group. Analysing the therapies, we observed that bDMARD was started in 18.0% of patients in preconception appointment, specifically certolizumab-pegol. 10.0% of patients had a flare during pregnancy and 20.0% afterwards. About foetal outcomes, one case of foetal malformation and one case of intrauterine growth restriction occurred and 8.0% of births were preterm.

Conclusion: This study shown that pregnancy can have an impact on disease activity. Still, only a low percentage of patients had a disease flare. This can be related to high preconception counseling and optimization of therapies, including DMARDs. This study suggests that with proper management and monitoring, most women with spondyloarthritis can have successful pregnancies and healthy babies, but further research is

TO 045 – TABLE.

	Ankylosing spondylitis (n=19)	Non-radiographic spondyloarthritis (n=15)	Psoriatic arthritis (n=10)	Enteropatic Spondylarthritis (n=4)	Undifferentiated spondylarthritis (n=2)	Total (n=50)
Patient characteristics						
Age (years)	35.33±3.90 (28-40)	33.74±4.83 (22-41)	33.00±3.74 (25-39)	34.25±6.13 (28-42)	28;30	33.90±4.43 (22-42)
Disease duration	66.73±46.63 (24-182)	61.67±39.69 (12-168)	62.50±38.99 (12-137)	28.50±24.69 (9-64)	5;48	58.10±41.36 (2-182)
Previous pregnancies	1.58±1.16(0-4)	1.29±1.05(0-3)	1.10±0.74 (0-3)	2.25±1.50(1-4)	1;0	1.20±1.16 (0-4)
Previous abortion	4/19 (21,1%)	4/15 (26,7%)	8/10 (80.0%)	1/4 (25.0%)	0/2(0.0%)	9/43(20.9%)
Preconception						
Preconception consultation	12/17(70.6%)	8/12 (66.7%)	8/10 (80.0%)	1/4 (25.0%)	1/2(50.0%)	30/50(60.0%)
bDMARD started in preconception	2/19 (33.3%)	4/15 (66.7%)	1/10 (10.0%)	2/4(50.0%)	0/2(0.0%)	9/50(18.0%)
Medication						
Prednisolone	5/19 (26.3%)	2/15 (13,3%)	3/10 (30.0%)	1/4(25.0%)	0/2(0.0%)	11/50(22.0%)
Sulfasalazine	4/19 (21.1%)	2/15 (13,3%)	3/10 (30.0%)	0/4(0.0%)	0/2(0.0%)	9/50(18.0%)
Certolizumab	6/19 (31.6%)	5/15 (33,3%)	1/10 (10.0%)	1/4(25.0%)	0/2(0.0%)	13/50(26.0%)
Pregnancy outcomes						
Flare after pregnancy	3/15 (16.7%)	1/11 (8.3%)	0/8 (0.0%)	1/4(25.0%)	0/2(0.0%)	5/50(10.0%)
Flare during pregnancy	6/13 (31.6%)	1/12 (7.7%)	2/8(25.0%)	1/4(25.0%)	0/2(0.0%)	10/50(20.0%)
Successful pregnancy	17/19(89.5)	12/15(80.0%)	7/10(70%)	4/4(100%)	2/2(100.0%)	42/50(84.0%)
Fetal outcomes						
Gestational age	37.67±1.78 (35-40)	39.05±1.03 (37-41)	38.43±0.79 (37-39)	37.75±0.96 (37-39)	39;38	32.60±13.54 (2-41)
Newborn Weight (grams)	3116.67±288.10 (2720-3480)	3296.71±403.26 (2330-4340)	3278.33±530.56 (2320-4030)	3417.50±632.37 (2750-4200)	3250;2660	2656.20±1310.60 (2-4340)
Fetal malformation	1/16 (5.9%)	0/12 (0.0%)	0/6 (0.0%)	0/4 (0.0%)	0/2(0.0%)	1/50(2.0%)
Preterm birth	0/17(0.0%)	4/12(33.3%)	0/7 (0.0%)	0/4 (0.0%)	0/2(0.0%)	4/50(8.0%)
Intrauterine growth restriction	0/17(0.0%)	1/11(8.3%)	0/7 (0.0%)	0/4 (0.0%)	0/2(0.0%)	1/50/2.0%)

needed related to high preconception counseling and optimization of therapies, including DMARDs.

050 - IMPACT OF COMORBIDITY BURDEN AS SCORED USING THE RHEUMATIC DISEASE COMORBIDITY INDEX (RDCI) ON RESPONSE TO TREATMENT WITH THE FIRST BDMARD AMONG PSORIATIC ARTHRITIS PATIENTS

Rafaela Nicolau[1], Ana Martins[2, 3], Daniela Oliveira[3, 4], Miguel Bernardes[2, 3], Lúcia Costa[3]
[1]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [2]Department of Medicine, Faculty of Medicine, University of Porto, Porto, Portugal, [3]Rheumatology Department, Centro Hospitalar de São João, Porto, Portugal, [4]Faculdade de Medicina da Universidade do Porto, Departamento de Medicina e CINTESIS, Porto, Portugal

Background: In addition to the impact of Psoriatic Arthritis (PsA) on risk of death and increased morbidity, other factors, such as comorbidity burden (CB), have also an important role. Identifying comorbidities and estimating their impact on the disease course are critical to safely and effectively treating a patient with PsA, as these comorbidities have often implications on therapy selection.
Objectives: To measure the impact of CB on PsA patients treated with a first-line Biologic Disease Modifying AntiRheumatic Drug (bDMARD) using the Rheumatic Disease Comorbidity Index (RDCI).
Methods: Retrospective observational cohort-study of patients with diagnosis of PsA, fulfilling the CASPAR criteria and treated with a first bDMARD, using data from the Reuma.pt database. Patients were divided in 2 groups according the RDCI (RDCI=0 and RDCI≥1) for evaluating its role in clinical response and disease activity at baseline and follow up (6 and 12 months). An univariate analysis was performed followed by a multivariate analysis.
Results: A total of 115 patients were included. 53% (n=61) were females, mean age was 47(±10.7) years old and the median disease duration 10.5 years [1.4-44.1]. At baseline, most of patients exhibited a very high or high disease activity (median DAS284V 4.44, ASDAS-CRP 3.98, BASDAI 6.69) and 68% were concomitantly treated with other DMARD and/or corticosteroids. 43% of patients had at least one comorbidity and the median RDCI score was 1.1 [0.0-6.0]. The most frequently reported comorbidities were cardiovascular disorders (37.7%), depression (13.2%) and pulmonary disease (6.1%). When comparing baseline demographic and clinical characteristics of the 2 subgroups, stratified according RDCI score, we found statistically significant differences in age, age at diagnosis, disease duration, BMI and smoking status. RDCI was poorly correlated with CRP (r=0.162, p<0.01), DAS284V

TO 050 – TABLE.

	RDCI=0 (n=70)	RDCI≥1 (n=45)	*p*
Current age,years	48.7±9.8	58.8±7.7	**<0.001**
Sex (M/F)	34/70	20/45	0.665
Age of diagnosis,years*	37.8±7.9	58.7±6.5	**<0.001**
Disease duration,years*	6.6±4.1	10.1±4.4	**0.02**
Smoking status,n(%)	26(10.7)	41(13.6)	**0.013**
Psoriasis,n(%)	55(78.5)	33(73.3)	0.706
Nail involvement,n(%)	29(41.4)	12(26.6)	0.107
Axial involvement,n(%)	37(53)	27(0.6)	0.452
Dactylitis,n(%)	27(38.5)	15(33)	**<0.001**
Enthesitis,n(%)	27(38.5)	7(15.5)	0.873
BMI(*)	25.4±4.7	31.2±4.9	**<0.001**
DAS28 4VCRP baseline*	3.6±1.4	3.6±1.1	0.736
ASDAS CRP baseline*	3.9±1.1	4.0±0.9	0.295
BASDAI baseline*	6.8±2.1	7.1±1.5	0.978
BASFI baseline*	5.8±2.7	6.2±1.9	0.378
ESR baseline*	29.1±23.5	39.1±22.8	**0.02**
CRP baseline*	2.1±3.5	2.3±2.6	0.275
HAQ baseline*	0.6±0.7	0.7±0.6	**0.006**
DAS28 4VCRP 12M*	2.0±0.7	2.9±1.1	**0.022**
ASDAS CRP 12M*	2.0±0.9	2.8±1.9	**0.04**
BASDAI 12M*	3.2 ±2.6	4.4±2.1	**0.106**
BASFI 12M*	2.6 ±2.7	5.3±2.7	**<0.001**
HAQ 12M*	0.6±0.7	1.1±0.6	**0.006**
EULAR response 12M,n(%)	57/63(90.5)	30/43(69.7)	**0.02**
ASDAS response 12M,n(%)	47/70(67.1)	24/45(53.3)	0.137
PSARC response 12M,n(%)	45/60(75)	23/37(62)	0.180
Therapeutic,n(%)			
cDMARDs	48(68.5)	29(64.4)	0.862
Glucocorticoids	40(57.1)	23(51.1)	0.526
Antidepressants	4(5.8)	9(20)	**0.01**

(mean±SD)

(r=0.263, p=0.008), ASDAS CRP (r=0.284, p<0.001) at 12 months. We also found moderate correlations in RDCI with BASFI (r=0.370, p<0.001) and HAQ-DI (r=0.351, p<0.01). Patients with higher RDCI had lower chance to obtain low disease activity (OR 0.69 (95%CI, 0.61–0.79); p < 0.001). EULAR response rate at 12 months was lower in PsA patients with comorbidities (69.7%vs90.5%, p=0.02).

Conclusion: To conclude, our study demonstrates that baseline CB is an independent predictor for insufficient 12 months' response to the first bDMARD. This confirmed the hypothesis that comorbidity should be treated to improve the long-term prognosis in PsA patients.

054 - MATERNAL AND PERINATAL OUTCOMES IN PREGNANT WOMEN WITH RHEUMATIC DISEASES TREATED WITH BIOSIMILAR TNF INHIBITORS

Ana Teodósio Chícharo[1, 2], Ana Rita Lopes[1, 3], Sofia C Barreira[1, 3], Luísa Pinto[4], Ana Rita Cruz-Machado[1, 3], Susana Capela[1, 3]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, Faro, Portugal, [3]Rheumatology Research Unit, Instituto de Medicina Molecular João Lobo Antunes, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal, [4]Departamento de Obstetrícia, Ginecologia e Medicina da Reprodução, Hospital de Santa Maria, CHLN, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Purpose: The use of biosimilars (Bs) in Rheumatology is increasing, widening access to biologic therapy worldwide. Despite the consequent increase in the exposure to biosimilars (Bs) at conception, there is scarce data about their use in pregnancy, and for this reason they are not addressed in the latest EULAR and ACR guidelines on reproductive health. Bs are expected to have comparable effects to the original molecules (1). Our goal is to describe maternal and perinatal outcomes in women who used Bs TNF inhibitors (TNFi) during conception and pregnancy.

Methods: Single-center observational retrospective study of pregnant women with rheumatic diseases exposed to Bs at a rheumatology-obstetric clinic.

Results: Five pregnancies in three caucasian women were included. Rheumatic diagnoses were rheumatoid arthritis (RA), axial spondyloarthritis (axSpA) and psoriatic arthritis (PsA). Table 1 summarizes maternal and perinatal outcomes and relevant clinical data. At the time of conception, all patients were in remission under Bs therapy: one on Bs infliximab (IFXb) and two on Bs etanercept (ETAb).

TO 054 – TABLE 1. Maternal and perinatal outcomes in women with rheumatic diseases treated with biosimilar TNF inhibitors during conception and pregnancy.

	Patient	Age at conception (1)	Obstetric history	Diagnosis	Disease duration (2)	DMARD at conception and during pregnancy (with dosing scheme)	Biosimilar last dose (3)	Flare in pregnancy	Flare in post-partum	Maternal infections	Gestational age at delivery (3)	Birth weight (g)	Breast-feeding	Timing of biosimilar reinitiation (4)	Newborn infections	Adverse pregnancy outcomes
A	Pregnancy 1	35	G1	Axial spondylo-arthritis	12	IFXb 5mg/kg every 9 weeks	18	No	No$	No	39+3	3160	Yes$	28	No	No
	Pregnancy 2	38	G2P1		15	IFXb 5mg/Kg every 9 weeks	24	Yes - Sacroiliitis at 18th WG		No	40+1	2990		16*	No	No
B	Pregnancy 3	29	G1	Rheumatoid arthritis	8	SSZ 1000mg/day and ETAb 50mg (weekly before conception, every 2 weeks during pregnancy)	7	No		No	N/A	N/A		N/A	N/A	Miscarriage at 8 WG
	Pregnancy 4	30	G2P0A1		9		30	No		No	40+2	2915		4	No	No
C	Pregnancy 5	32	G3P0A2	Psoriatic arthritis	7	ETAb 50mg weekly	26	No		Yes - urinary tract infection at 26th WG	37+5	2982		4*	Yes – urinary tract infection at 9 days-old	No

Legend: DMARDs - Disease-modifying antirheumatic drugs; IFXb - infliximab biosimilar; ETAb - etanercept biosimilar; SSZ - sulfasalazine; WG - weeks of gestation, N/A – not applicable; 1. In years; 2.In years before conception; 3. In weeks of gestation; 4. In weeks postpartum; 5. $ N/A for patient B pregnancy 3 *expected.

Patient A: Nulliparous woman aged 35, diagnosed with axSpA with axial and peripheral involvement and history of uveitis. After conception, IFXb 5 mg/kg every 9 weeks was maintained up to the 18th week of gestation (WG). No adverse pregnancy outcomes (APO) or disease activity flares were noticed. Three years later, during a subsequent pregnancy, the same patient remained on IFXb monotherapy at the same dosing scheme. At 18th WG, she experienced a sacroiliitis flare, which resolved after ultrasound-guided joint injection with methylprednisolone. IFXb was last administered at the 24th WG, and the remaining pregnancy was uneventful.
Patient B: A 30-year-old woman diagnosed with RA, treated with ETAb 50mg weekly, sulfasalazine 1g/day and prednisolone (≤5mg/day). After conception, ETAb dosage was adjusted to 50mg every other week until 30th WG due to sustained remission. No flares or APO were recorded. This woman had experienced a spontaneous miscarriage at 8th WG the year before, while on remission and under the above-mentioned treatment.
Patient C: A 32-year-old woman with PsA with dominant peripheral involvement, under ETAb 50mg weekly in monotherapy. ETAb was continued up to the 26th WG, when it was stopped due to a Streptococcus agalactiae mild urinary tract infection (UTI), successfully treated with a course of antibiotics. Although the arthritis remained under control, psoriasis relapsed, requiring the use of topical emollients.
All women underwent vaginal deliveries and gave birth to healthy term babies. No congenital abnormalities were recorded and there were no neonatal complications during the first week of life. All women decided to breastfeed. The male neonate of the woman with PsA, who had no known congenital urinary tract abnormalities, developed a UTI on the 9th day, requiring hospital admission. There were no maternal disease flares at postpartum. The Bs were resumed in the postpartum period in two cases. The patient with axSpA decided not to resume IFXb while breastfeeding, and the woman with PsA programmed to restart ETAb 4 weeks after delivery.
Conclusion: No new safety or efficacy issues arose from the use of IFXb or ETAb during conception or pregnancy. These data are reassuring and are in line with a previous observational study (2). In patients with stable disease under Bs TNFi, it is important to continue the usual treatment to reduce maternal and perinatal complications.

055 - PREGNANCY OUTCOMES IN SYSTEMIC SCLEROSIS: EXPERIENCE OF A RHEUMATOLOGY-OBSTETRIC JOINT CLINIC

Ana Teodósio Chícharo[1, 2], Ana Rita Lopes[1, 3], Sofia C Barreira[1, 3], Patrícia Martins[1], Luísa Pinto[4], Ana Rita Cruz-Machado[1, 4], Susana Capela[1, 3]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, [3]Rheumatology Research Unit, Instituto de Medicina Molecular João Lobo Antunes, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal, [4]Departamento de Obstetrícia, Ginecologia e Medicina da Reprodução, Hospital de Santa Maria, CHLN, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background: Systemic sclerosis (SSc) is a rare multisystemic connective tissue disease characterized by progressive fibrosis of the skin and visceral organs, accompanied by non-inflammatory vasculopathy. Women diagnosed with SSc seem to be at increased risk of developing adverse pregnancy outcomes (APO), but maternal and perinatal outcomes remain poorly understood in these patients. (1,2)
Objectives: To describe maternal and perinatal outcomes in women with SSc.
Methods: Retrospective observational study including pregnant women with SSc followed at a rheumatology-obstetric clinic from 01/2009 to 12/2022.
Results: A total of 12 pregnancies in 9 patients with SSc were identified. Disease phenotypes included: 1 diffuse cutaneous SSc (dcSSc), 3 limited cutaneous SSc (lcSSc), 2 very early diagnosis of systemic sclerosis (VEDOSS), 2 SSc sine scleroderma (ssSSc) and 1 overlap syndrome of SSc and dermatomyositis with predominance of SSc manifestations. Six women were positive for anti-centromere antibodies, while three others exhibited anti-Scl-70. Only one patient had major organ involvement – a case of usual interstitial pneumonia (UIP) in a woman with ssSSc. Table 1 summarizes maternal clinical data and pregnancy outcomes. The mean age at conception was 35.9 ± 4.9 years with a median disease duration of 19 (IQR 8.5) years. All women had their disease stable at the time of conception under no teratogenic drugs. Out of the 12 pregnancies, there were 10 live births and 2 miscarriages [at 4 and 6 weeks of gestation (WG)]. The mean gestational age at delivery was 38.2 ± 1.8 WG. There was one preterm birth at 35 WG in the woman with ssSSc with UIP after spontaneous preterm premature rupture of membranes (PPROM). Fetal growth restriction was diagnosed at 35 WG in a woman with lcSSc, and three other babies were born small for gestational age (SGA), from mothers with VEDOSS, lcSSC and ssSSc. There were no cases of gestational hypertension or pre-eclampsia. Two patients underwent scheduled cesarean sections for reasons not related to SSc disease. No congenital

TO 055 – TABLE 1.

Patient	Pregnancy	Disease phenotype	Clinical manifestations	Major organ involvement	Antibodies profile	Disease duration at conception (in years)	Obstetric history	csDMARD at conception	csDMARD during pregnancy	Other drugs during pregnancy	Gestation age at delivery (in WG)	Birth weight (grams)	Adverse pregnancy outcomes	Disease flares during pregnancy	Disease flares during post-partum
A	1	Diffuse cutaneous Ssc	RP, puffy fingers, digital ulcers, arthritis	No	Anti-Scl 70	9	G2P1A0	0	0	Nifedipine 20mg daily	-	-	Miscarriage at 4th WG	-	-
	2					10	G3P1A1	0	Hydroxycloroquine 400mg daily	Nifedipine 20mg daily, AAS 150mg daily	40	3320	0	0	Yes (worsening of RP, inflammatory arthralgia and aggravated skin thickening)
B	3	Limited cutaneous Ssc	RP, puffy fingers, inflammatory arthralgia, GER	No	Anti-centromere	7	G1	Hydroxy-cloroquine 400mg daily	Hydroxycloroquine 400mg daily	AAS 150mg daily, PDN 2.5mg daily	37	2720	0	0	0
C	4	Limited cutaneous Ssc	RP, sclerodactyly, telangiectasia	No	Anti-centromere	3	G2A0P1	0	0	0	37	2355	Fetal growth restriction	0	0
D	5	Limited cutaneous Ssc	RP, digital ulcers, calcinosis cutis, telangiectasia	No	Anti-centromere	18	G1	0	0	0	39	3300	0	0	0
	6					25	G2P1A0	0	0	Nifedipine 20mg daily	41	3300	0	Yes (worsening of RP, digital ulcers and calcinosis)	
E	7	VEDOSS	RP, puffy fingers, arthritis	No	Anti-centromere	4	G1P1A0	Hydroxycloroquine 400mg daily	Hydroxycloroquine 400mg daily	AAS 150mg daily	39	2920	SGA	0	0
F	8	VEDOSS	RP, puffy fingers, calcinosis cutis	No	Anti-centromere	8	G2P1A0	0	0	AAS 150mg daily	-	-	Miscarriage at 6th WG	-	-
	9					10	G3P1A1	0	0	AAS 150mg daily	39	3980	0	0	0
G	10	Sine scleroderma SSc	RP, puffy fingers, GER, dysphagia	No	Anti-centromere	14	G2P1A0	0	0	AAS 150mg daily	37	2330	SGA	0	0
H	11	Sine scleroderma SSc	RP, puffy fingers, GER interstitial lung disease	Yes - UIP	Anti-Scl 70	6	G2P1A0	Azathioprine 125mg daily	Azathioprine 125mg daily	PDN 5-10mg daily	35	2820	Preterm delivery with PPROM	Yes (alveolitis)	0
I	12	Overlap of Ssc and dermato-myositis	RP, digital ulcers, sclerodactyly, calcinosis, gottron papules, heliotrope rash, polymyositis	No	Anti-Scl 70	10	G2P1A0	0	0	AAS 150mg daily, PDN 2.5 mg daily	38	2635	SGA	0	0

Legends: AAS - acetylsalicylic acid, GER - gastroesophageal reflux; PDN - prednisolone; PPROM - preterm premature rupture of membranes; SSC – systemic sclerosis; SGA - small for gestational age; UIP – usual interstitial pneumonia; VEDOSS – Very early diagnosis of systemic sclerosis; WG - weeks of gestation

abnormalities or neonatal infections were recorded. All women decided to breastfeed. During gestation, all women experienced improvement in their Raynaud phenomenon (RP), except for a patient with lcSSc that developed digital ulcers that improved with nifedipine 30mg/day, as well as calcinosis in elbows and fingers that persisted in the postpartum period. There were also frequent complaints of gastroesophageal reflux in 3 (33%) patients. The patient with ssSSc with UIP reported a dry cough at 27th WG. After exclusion of respiratory tract infection, she was diagnosed with alveolitis related to her underlying lung disease. Prednisolone was increased up to 10mg/day and her symptoms improved. It was then gradually tapered to 5mg before delivery at 35 WG due to PPROM. One patient with dcSSc relapsed in the post-partum period: she reported worsening of RP, inflammatory arthralgia and aggravated skin thickening of hands and feet. Regarding immunomodulatory treatment, 4 patients (44%) were under conventional DMARDs (azathioprine and hydroxychloroquine) and 3 (33%) women received prednisolone (Table 1). No associations were found between disease phenotype, auto-antibodies profile or disease flares and the risk of APO.

Conclusion: Most pregnant women with SSc managed at our multidisciplinary unit experienced successful gestations. However, they may still be at risk for developing APO and disease flares. In our series, SGA was the most frequent APO recorded followed by pregnancy losses.

058 - CHECKAP: PREVALENCE OF PSORIATIC ARTHRITIS (PSA) AND PERFORMANCE EVALUATION OF THE EARP QUESTIONNAIRE IN THE POPULATION OF PORTUGUESE PATIENTS WITH PSORIASIS FOLLOWED IN A DERMATOLOGY SETTING

Ana Rita Henriques[1], Fernando Pimentel-Santos[2, 3], [2], Rita Pinheiro Torres [2], [2], Bernardo Pimentel[2], Maria Helena Lourenço[2], Sandra Barão Neves[2], Ana Palmeiro[2], Laura Gago[2], Mariana Emília[2], Ana Teodósio Chícharo[2], Anabela Barcelos[1, 4], Manuela Loureiro[4], Susana P. Silva[4], Elsa Vieira-Sousa[5], Antunes J[6], Carolina Ochôa Matos[5], Miguel Alpalhao[6], S Magina[7], Alexandra Bernardo[7], Maria Seabra Rato[7], Pedro Ponte[8], Tiago Torres[9], Tiago Meirinhos[9], Martinha Henrique[10], Marília Rodrigues[10], Diogo Jesus[10], Alexandra Daniel[10], Luisa Brites[10], Pedro Mendes Bastos[11], Patrícia Nero[11], Valada MP[1], David G.Lopes[1], Rute de Sousa[1], Helena Canhão[1], Rodrigues AM[1, 8]

[1]Comprehensive Health Research Centre (CHRC), NOVA Medical School | Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa, Lisboa, Portugal, [2]Hospital Egas Moniz, Centro Hospitalar Lisboa Ocidental, CHLO-E.P.E, Lisboa, Portugal, [3]iNOVA4Health, Rheumatology Lab, NOVA Medical Research, Lisboa, Portugal, Lisboa, Portugal, [4]Serviço de Dermatologia e Serviço de Reumatologia do Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [5]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [6]Departamento de Dermatologia, Centro Hospitalar e Universitário Lisboa Norte, Hospital de Santa Maria,, Lisboa, Portugal, [7]Centro Hospitalar Universitário de São João, Porto, Portugal, [8]Hospital dos Lusíadas, Lisboa, Portugal, [9]Instituto Médico de Estudos Imunológicos, Porto, Portugal, [10]Hospital de Santo André, Centro Hospitalar de Leiria, E.P.E., Leiria, Portugal, [11]Hospital CUF Descobertas, Lisboa, Portugal

Objective: To estimate the percentage of Psoriatic Arthritis (PsA) and the performance of the EARP Portuguese version (EARP-PT) questionnaire, in Portuguese psoriasis (Pso) patients in a dermatology setting.

Methods: A multicentre national, cross-sectional, observational study with two independent assessments (dermatologist and rheumatologist), was performed. Patients were recruited at Portuguese study sites (private and public) according to the criteria: adults (≥ 18 years old), diagnosis of Pso made by a dermatologist, understand the Portuguese language. PsA diagnosis was defined by a combination of expert opinion and CASPAR criteria. PsA estimated prevalence was computed. The EARP-PT questionnaire screening accuracy to identify PsA patients was evaluated.

Results: Pso patients (n=172) were included with a mean age (SD) of 53.8 (14.5) years, 53.5% male. The prevalence of PsA in patients with Pso in our sample was 8.70% (95% CI: 4.8-14.2), more frequent in women (9.09% vs 8.33% in men) and in the group of 31-59 years old. The EARP-PT questionnaire showed a good internal consistency (Cronbach's α=0.81). Considering the cut-off point of 3, as in the initial validation study, the sensitivity and specificity were 71.4% and 40.1%, respectively and the AUC was 0.558 (95% CI: 0.429-0.687). Based on the Youden index, the optimal cut-off, for our population seems to be 5, with a sensitivity and specificity of 57.1% and 64.6%, respectively and the AUC was 0.636 (95% CI: 0.481-0.791).

Conclusion: We found that 1 in 11 patients with psoriasis have PsA. The EARP-PT questionnaire offer a moderate utility in screening of PsA for dermatologists.

Funding: This study is funded by an unrestricted research grant from Novartis Portugal, SA.

059 - PROGRESSIVE INTERSTITIAL LUNG DISEASE IN SYSTEMIC SCLEROSIS

Catarina Abreu[1], Ana Cordeiro[1], Maria José Santos[2, 3]

[1]Reumatology Department, Hospital Garcia de Orta, EPE, Almada, Portugal, [2]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [3]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background: Interstitial lung disease (ILD) is the leading cause of death and a major cause of morbidity in systemic sclerosis (SSc), with a highly variable clinical presentation and evolution. Progressive ILD has been defined according to pulmonary function tests (PFTs) as a decline in forced vital capacity (FVC) of ≥10% or a decline in FVC of 5–9% in association with a decline in diffusing capacity of the lung for carbon monoxide (DLco) of ≥15% at 1-year follow-up. Early identification of patients with progressive ILD is crucial to prevent further progression and reduce associated morbidity and mortality.

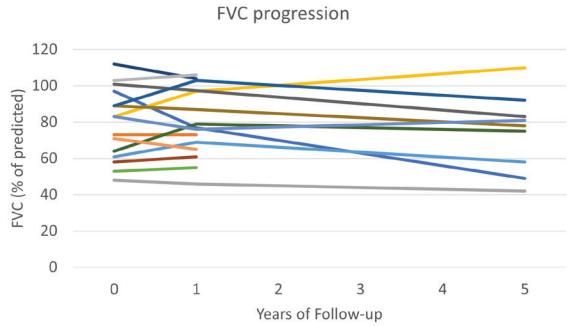
Objective: Our aim is to determine the prevalence of progressive ILD in SSc and to describe demographic and clinical characteristics of ILD progressors based on PFTs.

Methods: Adult patients with the diagnosis of SSc and ILD with available measurements of FVC at baseline and at 1-year or 5-year follow-up were included. SSc-ILD

TO 059 – TABLE 1.

	Total (N=15)		1-year follow-up (N=14)				5-year follow-up (N=9)			
			Progressors (N=4)		Non-progressors (N=10)		Progressors (N=4)		Non-progressors (N=5)	
Female – no (%)	11	(73.3)	4	(100)	6	(60)	3	(75)	4	(80)
lcSSc – no (%)	11	(73.3)	4	(100)	7	(70)	3	(75)	4	(80)
Baseline FVC - % of predicted	76.5 ± 19.9		90.7 ± 17.7		72.1 ± 18.2		83.8 ± 24.4		76.0 ± 12.6	
Baseline DLCO - % of predicted	53.1 ± 18.0		71.5 ± 5.3		45.0 ± 15.3		45.0 ± 19.2		56.3 ± 19.3	
Clinical Manifestations										
Digital Ulcers – no (%)	6	(40)	1	(25)	4	(40)	4	(100)	0	
Pulmonary Hypertension – no (%)	3	(20)	0		3	(30)	1	(25)	0	
Calcinosis – no (%)	1	(6.7)	0		1	(10)	0		0	
Arthralgias – no (%)	8	(53.3)	2	(50)	6	(60)	2	(50)	2	(40)
Myositis – no (%)	3	(20)	1		2	(20)	2	(50)	1	(20)
Esophageal involvement – no (%)	10	(66.7)	2	(50)	8	(80)	3	(75)	4	(80)
Cardiac involvement – no (%)	8	(53.3)	1	(25)	6	(60)	3	(75)	2	(40)
Immunology										
anti-Scl70 – no (%)	8	(55.5)	1	(25)	6	(60)	2	(50)	2	(40)
ACA – no (%)	2	(13.3)	1	(25)	1	(10)	0		0	
ARA – no (%)	1	(6.7)	1	(25)	0		0		1	(20)
anti-Ro60 – no (%)	1	(6.7)	1	(25)	0		1	(25)	0	
Capillaroscopy pattern										
No scleroderma pattern – no (%)	3	(23)	1	(33.3)	0		0		3	(60)
Early – no (%)	3	(23)	1	(33.3)	1	(11.1)	2	(50)	0	
Active – no (%)	3	(23)	1	(33.3)	2	(22)	2	(50)	0	
Late – no (%)	4	(30.8)	0		4	(44.4)	0		2	(40)
Chest CT pattern										
UIP – no (%)	4	(57.1)	1	(50)	3	(60)	3	(100)	0	
NSIP – no (%)	3	(42.9)	1	(50)	2	(40)	0		2	(100)

*FVC – Forced Vital Capacity; DLCO - diffusing capacity for carbon monoxide; anti-Scl70; ACA anti-centromere antibodies; ARA – anti RNA-polymerase III antibodies; CT – Computed tomography; UIP – Usual interstitial pneumonia; NSIP – nonspecific interstitial pneumonia



TO 059 – Figure.

progressors, were defined as having a decline in FVC ≥5% at 1-year follow-up (significant ≥10% or moderate 5-10%) and at 5-year follow-up (major ≥20%; significant 10-20% or moderate 5-10%).

Results: Out of 189 patients with SSc, 42 were identified with SSc-ILD of whom 15 fulfilled the previous criteria. Eleven patients (73.3%) were female and 13 (86.7%) were Caucasian. The mean age at diagnosis of SSc was 52.6±15.9 years. The mean baseline FVC was 76.5±19.9 (% of predicted) and mean DLco was 53.1±18.0 (% of predicted). Cutaneous involvement was limited (lcSSC) in 11 patients (73.3%) and diffuse (dcSSC) in 4 (26.7%). The predominant SSc-associated autoantibody was anti-Scl70 (N=8; 55.5%) followed by anti-centromere (ACA) in 2 (13.3%). The evolution of the individual FVC over 5 years is represented in Figure 1.

At 1-year follow-up we identified 4 progressors, all female with lcSSc and a mean age at diagnosis of SSc of 64±7.5 years: one had a significant (≥10%) decline in FVC and 3 had a moderate (5-10%) decline in FVC (one with a decline in DLCO ≥15%; one with a decline <15% and one with no available DLCO). Considering their immunological panel, one had ACA, one anti-Scl70, one anti-RNA polymerase III and one anti-Ro60. The mean FVC at diagnosis was 90.7±17.7 and DLCO was 71.5±5.3. The mean DLCO at baseline was inferior in non-progressors than progressors.

At 5-year follow-up 4 patients were identified as progressors, 3 were female with a mean age at diagnosis of 60.5±10.7 years: 1 had a major decline in FVC (≥20%), 2 had a significant decline (10-20%) and 1 had a moderate decline (5-10%). The patient with a significant decline in the first year of follow-up was the same presenting with a major decline at 5-year follow-up. Three patients (75%) had lcSSc and 2 (50%) were positive for anti-Scl70 antibodies. On chest computed tomography 3 patients (75%) had usual interstitial pneumonia pattern. Regarding capillaroscopy findings, 50% had active scleroderma pattern and the remaining had early scleroderma pattern (Table 1).

Conclusion: The prevalence of SSc-ILD progression at 1-year follow-up was 28.4% and 44.4% at 5-year follow-up. The DLco at baseline was lower in non-progressors than progressors, which may be due to a higher prevalence of pulmonary hypertension in non-progressors or to a later diagnosis of SSc, since the diagnosis of ILD and SSc was simultaneous in most patients.

We want to highlight the importance of early SSc diagnosis, screening and monitoring for ILD and evaluation of progression of PFTs values, despite FVC absolute values and immunological profile.

060 - HOW DIFFICULT-TO-TREAT CAN PSORIATIC ARTHRITIS BE?

Catarina Abreu[1], Vanessa Fraga[1], Alice Castro[1], Maria José Santos[1, 2]

[1]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background: Despite the growing number of biological and targeted synthetic disease modifying antirheumatic drugs (b/tsDMARDs) for the treatment of psoriatic arthritis (PsA), it is not uncommon to find patients in clinical practice who have high disease activity and fail to respond to multiple therapeutic agents. In 2020, the European Alliance of Associations for Rheumatology (EULAR) proposed a definition for difficult-to-treat (D2T) rheumatoid arthritis (RA). However, a similar definition has not been coined for PsA. Recently, Anand Kumthekar et al1, published the first proposed definition for D2T PsA which differs from D2T RA definition on the first criterion suggesting failure of 3 or more b/tsDMARDs.

Objective: Our aim is to determine the prevalence of D2T PsA, describe clinical characteristics of refractory patients and compare them with the non-refractory.

Methods: Adult patients with the diagnosis of PsA according to Classification for Psoriatic Arthritis (CASPAR) criteria and registered in the Rheumatic Diseases Portuguese Registry (Reuma.pt) single centre database were included. The EULAR criteria for D2T RA were applied to identify patients with D2T PsA.

Results: Among 141 PsA patients, 6 (4.3%) fulfilled D2T criteria, of whom 66.7% (N=4) were female. The mean age at diagnosis was 39.8±8.3 years and 37.8±9.7 at symptom onset. Symmetric polyarthritis (N=5; 83.3%) was the predominant phenotype, followed by asymmetric oligoarthritis (N=1). The mean time from symptom onset to the initiation of first b/tsDMARD was 3.8±3.5 years. At the beginning of first b/tsDMARD the mean score of Disease Activity Index for Psoriatic Ar-

TO 060 – TABLE 1. Clinical and demographic characteristics of PsA patients.

	D2T (N=6)	Non-refractory (N=135)
Female Sex – no (%)	4 (66.7)	64 (47.4)
Caucasian – no (%)	6 (100)	132 (97.7)
Age at diagnosis – years	39.8 ± 8.3	48.4 ± 14.5
Age at symptom onset – years	37.8 ± 9.7	45.5 ± 14.4
Symmetric polyarthrtitis – no (%)	5 (83.3)	83 (61.5)
Asymmetric olygoarthritis – no (%)	1 (16.7)	34 (25.2)
Predominant distal interphalangeal – no (%)	0	14 (10.4)
Arthritis mutilans – no (%)	0	1 (0.7)
Predominant axial involvement – no (%)	0	14 (10.4)
Extra-articular manifestations – no (%)	6 (100)	129 (95.6)
Psoriasis – no (%)	6 (100)	126 (93.3)
Dactylitis – no (%)	3 (50)	25 (18.5)
Enthesitis – no (%)	3 (50)	24 (17.8)
Uveitis – no (%)	0	2 (1.5)
Nail dystrophy – no (%)	2 (33.3)	35 (26)
Cardiovascular disease – no (%)	3 (50)	70 (51.9)
Tender joints – no	8.4 ± 7.0	13.4 ± 6.6
Swollen joints – no	11 (15)	3 (5)
CRP – mg/dL	1.1 (2.3)	0.95 (1.6)
ESR – mm/hr	21.5 (62)	36 (41)
VAS - pain	80 (30)	60 (30)
DAPSA	38.1 ± 19.8	27.7 ± 11.4
Current b/tsDMARD		
Infliximab – no (%)	3 (50)	-
Ixekizumab – no (%)	2 (33.3)	-
Adalimumab – no (%)	1 (16.7)	-

CRP – C-reactive protein; ESR – Erythrocyte sedimentation rate; VAS – Visual analogic scale; DAPSA – Disease Activity Score for Psoriatic Arthritis; b/tsDMARD - Biological and targeted synthetic disease modifying antirheumatic drugs

thritis (DAPSA) in D2T patients was 38.2±19.8 with a mean C-reactive protein of 1.05±2.35 mg/dL and erythrocyte sedimentation rate of 21 (62) mm/hr.
The mean number of b/tsDMARDs used was 4.2±1.2, with a maximum of 6. Tumor necrosis factor inhibitors (TNFi) were the first bDMARD used in 83.3% (N=5) - adalimumab or etanercept - and the second in 66.67% (N=4); sekukinumab was the second line in the remaining patients. All patients failed at least two mechanisms of action (MOA), all of them failed at least one TNFi and 4 (66.7%) failed anti-interleukin 17 inhibitor.
Apart from psoriasis, which was present in all, 5 (83.3%) D2T patients presented at least one extra-articular manifestation (dactylitis, uveitis or enthesitis). There were no cases of obesity, depression, or fibromyalgia, and 50% had cardiovascular comorbidities.
The bivariate analysis performed between b/tsDMARDs refractory and non-refractory patients showed no statistically significant differences.
Discussion/Conclusion: When applying the established criteria for D2T RA, we found a prevalence of 4.3% of D2T PsA, however the shorter availability of different MOA in PsA compared with RA may have led to an underdiagnosis of D2T patients. No differences in clinical presentation or presence of comorbidities were observed. The management of D2T PsA represents a significant challenge in clinical practice and adherence to treatment and diagnostic certainty should always be questioned.
The small sample size is a limitation, and we intend to analyse a larger sample to explore potential factors contributing to treatment refractoriness.
Although a standardized definition for D2T PsA is lacking, identification of these patients can enhance treatment decision-making and optimize outcomes.

REFERENCES

1. Kumthekar A, Ashrafi M, Deodhar A. Difficult to treat psoriatic arthritis - how should we manage? Clin Rheumatol. 2023 Apr 25. doi:10.1007/s10067-023-06605-9; PMID:37097525.

061 - AXIAL PSORIATIC ARTHRITIS, DATA FROM RHEUMATIC DISEASES PORTUGUESE REGISTRY: PRELIMINARY RESULTS

Catarina Abreu[1], Vanessa Fraga[1], Mariana Diz-Lopes [2, 3], Sofia Ferreira Azevedo[4], Carolina Vilafanha[4], João Rovisco[5], Inês Almeida[6], Maria Pontes Ferreira[7], Hugo Parente[7], Anita Cunha[7], Catarina Soares[7], Dina Medeiros[8], Duarte Augusto[9], Mariana Rocha Sebastião[10], Lígia Silva[11], Catarina Cortesão[8], Tiago Meirinhos[12], Joana Silva-Dinis[13], Filipe Araújo[14, 15], José Tavares-Costa[7], António Vilar[16], Alice Castro[1], Maria José Santos[1, 17]
[1]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [2]Rheumatology Department, Centro Hospitalar de São João, Porto, Portugal, [3]Department of Medicine, Faculty of Medicine, University of Porto, Porto, Portugal, [4]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [5]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [6]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [7]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [8]Portuguese Institute of Rheumatology, Lisboa, Portugal, [9]Rheumatology Department, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [10]Serviço de Reumatologia, Hospital do Divino Espírito Santo, Ponta Delgada, Portugal, [11]Rheumatology Department, Centro Hospitalar Trás-os-Montes e

Alto Douro, Vila Real, Portugal, [12]Rheumatology Unit, Centro Hospitalar do Tâmega e Sousa, Penafiel, Portugal, [13]Rheumatology Department, centro Hospitalar de Lisboa Central, Lisboa, Portugal, [14]Hospital Ortopédico de Sant 'Ana, Cascais, Portugal, [15]Serviço de Reumatologia, Hospital CUF Cascais, Cascais, Portugal, [16]RORVILAR - Clínica Roma, Lisboa, Portugal, [17]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

TO 61 – TABLE 1. Clinical and demographic characteristics of patients with axPsA and comparative analysis of exclusive vs. concomitant with peripheral involvement axPsA

	axPsA (N=854)	Exclusive axPsA (N=186)	axPsA concomitant with peripheral (N=668)	p-value	
Female sex - no %	342 (40)	74 (39.8)	268 (40.1)	0.934	
Diagnosis					
PsA - no %	754 (88.3)	134 (72)	620 (92.8)	<0.001	OR=5.01; 95% CI [3.25 – 7.74]
SpA + psoriais - no %	100 (11.7)	52 (28)	48 (7.2)		
PsA phenotype					
Predominant axial inv. - no %	340 (39.8)	134 (72)	206 (30.8)		
Symetric polyarthritis - no %	242 (28.3)	-	242 (28.3)		
Predominant DIP - no %	18 (2.1)	-	18 (2.1)	*	
Asymmetric olygoarthritis - no %	148 (17.3)	-	148 (17.3)		
Arthritis Mutilans - no %	6 (0.7)	-	6 (0.9)		
Race					
European Caucasian - no %	635 (97.1)	123 (94.6)	512 (97.7)		
Non-european Caucasian - no %	10 (1.5)	5 (3.8)	5 (1)	*	
Other - no %	8 (1.3)	2 (1.6)	6 (1.2)		
HLA-B27 - no %	183 (21.4)	73 (50.7)	110 (26.3)	<0.001	OR=0.35; 95% CI [0.23-0.52]
Age at diagnosis - years	41.9 ± 12.9	41.7 ± 13.3	43.2 ± 12.8	0.177	
Age at symptom onset – years	38.5 ± 13	35.7 ± 13.3	39.2 ± 12.8	0.002	
Extra-articular Manifestations					
Psoriasis - no %	829 (98)	186 (100)	643 (97.4)	*	
Nail dystrophy - no %	284 (35.9)	38 (22.5)	246 (39.6)	<0.001	OR=2.26; 95% CI [1.52-3.35]
Uveitis - no %	74 (9.6)	27 (16.2)	47 (7.8)	0.001	OR=0.44; 95% CI [0.26-0.72]
Enthesitis - no %	283 (35.9)	32 (32)	251 (39.7)	<0.001	OR=2.83; 95% CI [1.87-4.31]
Dactylitis - no %	257 (32.7)	10 (10)	247 (40.4)	<0.001	OR=10.20; 95% CI [5.29-19.60]
IBD - no %	18 (2.3)	6 (3.7)	12 (2)	0.241	
Imagiologic Findings - no %	257 (83.7)	76 (88.4)	181 (81.9)	0.168	
Radiographic SI - no %	195 (69.4)	61 (81.3)	150 (64.7)	0.07	
SI grade (mNYC)					
Grade 2 - no %	22 (14.9)	4 (9.8)	18 (16.8)		
Grade 3 - no %	75 (50.7)	18 (43.9)	57 (53.5)	0.147	
Grade 4 - no %	51 (34.5)	19 (46.3)	32 (29.9)		
SI symmetry - no %	94 (60.6)	29 (69)	65 (57.5)	0.192	
SI on MRI - no %	54 (76.1)	16 (69.6)	38 (79.2)	0.375	
Syndesmophytes - no %	93 (42.1)	26 (47.3)	67 (40.4)	0.368	

*not possible to compute

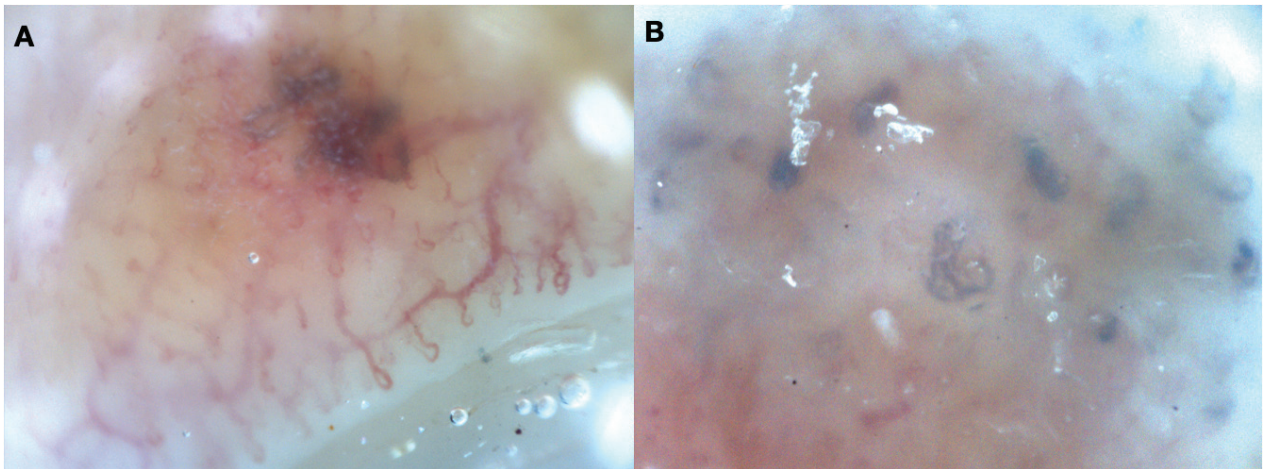
Number of missings per variable: race 200; HLA-B27 292; psoriasis 9; nail dystrophy 64; uveitis 81; dactylitis 67; enthesitis 66; IBD 86; radiographic SI 573; syndesmophytes 633; SI grade 706; SI symmetry 699; SI on MRI 783; PsA – Psoriatic Arthritis; SpA – Spondyloarthritis; inv. – involvement; DIP – distal interphalangeal; IBD – Inflammatory bowel disease; SI – sacroiliitis; mNYC – modified New York Criteria; MRI – magnetic resonance.

Background: Psoriatic Arthritis (PsA) is a heterogeneous inflammatory arthropathy that encompasses 5 different phenotypes according to Moll and Wright. Axial Psoriatic Arthritis (axPsA) can be exclusive or it can occur concomitantly with peripheral involvement. It is estimated that exclusive axPsA occurs in 5% of PsA patients, but when concomitant with peripheral manifestations its prevalence rises to 25-70%. However, the absence of a consensus definition for axPsA makes the true prevalence difficult to estimate.
Objectives: 1) determine the prevalence of axPsA among PsA patients and how it is diagnosed in clinical practice and 2) describe the demographic and clinical characteristics of axPsA patients (exclusive and concomitant with peripheral involvement).
Methods: Adult patients diagnosed with PsA or axial spondyloarthritis (SpA) concomitant with psoriasis registered in the Rheumatic Diseases Portuguese Registry (Reuma.pt) were included. Patients with peripheral PsA not fulfilling CASPAR criteria and patients with exclusive axial involvement without psoriasis were excluded. Axial involvement was defined as physician reported spondylitis and/or the presence of imagiologic findings suggestive of axial involvement (radiographic sacroiliitis (SI) according to modified New York Criteria (mNYC), SI in magnetic resonance imaging (MRI) or presence of syndesmophytes in axial radiography).
Results: A cohort of 2304 PsA patients was analysed. The prevalence of axPsA was 37.1% (N=854), of whom 21.8% (N=186) had exclusive axPsA and 78.2% (N=668) had concomitant peripheral involvement. Among the patients with axPsA, 88.3% (N=754) had a clinical diagnosis of PsA, in which cases the prevailing phenotype was predominant spinal involvement (N=340; 39.8%), followed by symmetric polyarthritis (N=242; 28.3%) and asymmetric olygoarthritis (N=148; 17.3%). The remaining patients had a clinical diagnosis of SpA with psoriasis (N=100; 11.7%). When considering exclusive axPsA, the prevalence of the diagnosis of SpA with psoriasis increased to 28%. The biomarker HLA-B27 was present in 21.4% of patients and was significantly associated with exclusive axPsA (OR=2.88; 95%CI [1.94-4.26]; p<0.001). At symptom onset, patients with exclusive axPsA were younger (35.7±13.3 vs 39.2±12.8; p=0.002).
Uveitis was more prevalent in patients with exclusive axPsA whereas nail dystrophy, dactylitis and enthesitis were associated with concomitant peripheral involvement when compared with exclusive axPsA.
AxPsA was identified by suggestive imaging findings in 30.1% of patients, of which radiographic SI was the most common (N=195; 75.9%) followed by presence of syndesmophytes (N=93; 36.2%) and SI on MRI (N=54; 21%). In the remaining 69.9% of cases diagnosis was based on physician reported spondylitis, since no imaging exams were available.
Conclusion: The prevalence of exclusive axPsA was 8.1% and increased to 37.1% when concomitant with peripheral involvement. The most common method of diagnosis was physician reported spondylitis. However, when imaging findings were present, the most prevalent was radiographic SI. The scarce number of available imaging exams may have led to an underdiagnosis of axPsA since subclinical disease might not have been reported.
Exclusive axPsA patients had distinct clinical and laboratorial features. Further analysis will be performed to better understand clinical and demographic characteristics of patients with axPsA versus exclusive peripheral involvement.

062 - RHEUMATIC MANIFESTATIONS AND CAPILLAROSCOPIC CHANGES OF ALKAPTONURIA

Roberto Costa[1, 2], Nikita Khmelinskii[1, 2], Eduardo Dourado[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background: Alkaptonuria is a rare autosomal recessive disorder characterized by a deficient activity of homogentisate 1,2-dioxygenase, leading to the accumulation of homogentisic acid (HA). This accumulation causes ochronosis, visible as deposits of a dark pigment in the sclerae, skin, and subcutaneous cartilage. Alkaptonuria also leads to accelerated osteoarthritis in large joints, tendon and ligament ruptures, and bone fragility. Although dark urine is present from birth, symptoms typically develop in mid-to-late adulthood when ochronosis becomes evident. Diagnosis is usually confirmed by measuring HA levels in a 24-hour urine chromatography or through genetic testing. Capillaroscopy, a non-invasive bedside examination, can assess the nailfold microcirculation. Capillaroscopic findings in patients with alkaptonuria haven't been previously reported.
Objectives: This study aimed to evaluate and describe the rheumatic manifestations and capillaroscopic changes in patients with alkaptonuria.
Methods: Adult patients with alkaptonuria followed in our Rheumatology department were systematically screened for clinical manifestations and previous medical history, and clinical photographs and capillaroscopic images were taken. All patients provided written informed consent.
Results: Out of the seven patients, five were female



TO 062 – Figure 1. Capillaroscopic findings in patients with alkaptonuria. Standard view (A) of blue-blackish deposits not visible to th

(71.4%), and the median age at diagnosis was 54 (IQR 21) years. Three patients (42.9%) were diagnosed based on the presence of black tendons, ligaments, and bones observed during surgical procedures (one tendon rupture correction and two spinal surgeries). Four patients were siblings, and three of them were diagnosed through genetic screening after their sibling was diagnosed. The seventh patient was diagnosed as an infant due to darkened urine. All patients, except one, reported darkened urine since infancy (85.7%), and all patients exhibited ochronosis. The first symptoms, aside from urinary discoloration, were reported at a median age of 36 (IQR 6) years and included spinal pain in all patients (100%) and pain in large joints in half of the patients (50%). Six patients (85.7%) experienced osteoarthritis and tendinopathy in the large joints and spine. One patient had a normal capillaroscopy, while five patients (71.4%) showed microscopic blue-blackish deposits (Figure 1), four patients (57.1%) had tortuous capillaries, and four patients (57.1%) had abnormal capillary shapes without a reduction in capillary density. Two patients (28.6%) exhibited microhemorrhages, and two patients (28.6%) had capillary dilations.

Conclusion: Alkaptonuria is a rare disease with a high prevalence of rheumatic manifestations, including early and severe osteoarthritis and tendon ruptures. Capillaroscopy revealed changes in most patients, with the commonest finding being microscopic blue-blackish deposits that were not visible to the naked eye. These capillaroscopic changes have not been previously described. Capillaroscopy may serve as an easy, quick, and non-invasive screening tool for individuals at risk of the disease, such as relatives of affected individuals or patients with early and atypical osteoarthritis or frequent tendon ruptures. Longitudinal studies could further investigate if capillaroscopic lesions change over time.

064 - THROMBOCYTOPENIA IN SYSTEMIC LUPUS ERYTHEMATOSUS - THE THERAPEUTIC ARSENAL

Roberto Costa[1, 2], Duarte Pereira Vinha[1, 2], Nikita Khmelinskii[1, 2], Carla Macieira[2]

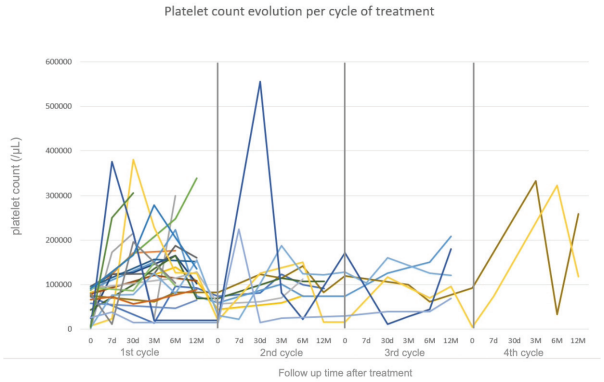
[1]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Systemic lupus erythematosus (SLE) is an immune-mediated multiorganic disease. Hematologic involvement is common, affecting around 50% of patients. Although no targeted therapy is needed in most cases of thrombocytopenia, therapies include glucocorticoids (GC), intravenous immunoglobulin (IVIg), rituximab (RTX) and splenectomy.

Objectives: To describe the effectiveness and safety of the therapies of thrombocytopenia in patients with SLE.

Methods: Single-centre observational retrospective study including patients registered at the Rheumatic Diseases Portuguese Registry (Reuma.pt), who fulfilled the 2019 EULAR/ACR SLE classification criteria and had ≥1 episode of thrombocytopenia (<100000/µL) between 2013 and 2023. Demographic and clinical data were collected.

Results: Table 1 shows demographic and clinical characteristics of the twenty-five patients included. The median (IQR) lowest platelet count was 57000 (61500)/µL with a median SLEDAI of 5 (4). Five (20%) patients had episodes of thrombocytopenia below 10000/µL. Nine (36%) had persistent thrombocytopenia (<150000/µL), of whom seven maintained values over 50000/µL and five required multiple lines of therapy. Six patients had relapsing thrombocytopenia (2-8 episodes), whilst ten had 1 episode.



TO 064 – Figure 1. Graphic 1 - Platelet count evolution per cycle of treatment

Ten patients (40%) underwent >1 cycles of treatment and in 3 patients (12%) no targeted therapy was started. No statistically significant differences were verified between these groups.

Considering the episodes requiring targeted therapy (n=40), GC were used the most (n=25). Five episodes required pulsed intravenous GC (methylprednisolone or dexamethasone) and in 25 the patients started or had their dose of oral prednisolone increased (1mg/Kg/day in 7, 10mg/day-1mg/Kg/day in 5 and ≤10mg/day in 13). Thirteen episodes were treated with IVIg (2g/Kg/cycle), of which six with a single cycle and the remaining up to 36 cycles (during 21±24.2 months). Other therapies chosen were azathioprine (n=10) and RTX (n=6; 1-3 cycles). Two patients with persistent thrombocytopenia despite treatment with medium to high doses of GC, IVIg and RTX or eltrombopag underwent splenectomy. No statistically significant differences were verified regarding the demographic and baseline clinical characteristics of the different treatment groups.

Bleeding dyscrasia happened in 28% of patients. The most common complications of treatment were infections (48%) and fragility fractures (20%).

At baseline of therapy, the average value of platelets was 68000 (66500)/µL. A statistically significant increase of platelet count was observed 1 month [120500 (104000)/µL; p<0.01] and 6 months [115500 (85000)/µL; p<0.01] after treatment.

Comparing the different treatments, the group treated with IVIg had a significantly lower value of platelets at baseline [2000-83000/µL, median of 16000 (60500)/µL vs 75000 (48000)/µL; p<0.01] and a significant increase in platelet count at day 7 (p=0.013). At the 6th month, there was a significant increase in platelet count in the added/increased GC group (p<0.01). No statistically significant differences were observed between treatments after 30 days, 3 and 12 months.

Conclusions: In SLE thrombocytopenia might be difficult to treat. Most moderate-severe episodes were treated with added/increased GC, which was more efficacious at 6 months. IVIg was used in cases with lower baseline values. RTX and splenectomy were rescue therapies. As a complication, a high percentage of infections was observed. The sample size, conditioned by the time interval and criteria heterogeneity, is a limitation of this report.

065 - PREGNANCY OUTCOMES IN SYSTEMIC LUPUS ERYTHEMATOSUS: A MULTICENTRIC EXPERIENCE

Inês Santos[1], Rafaela Nicolau[1], Tiago Beirão[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Santos[4], Mariana Rodrigues[4], Anabela Rocha[5], Sofia Bessa[5], Joana Aidos[6], Joana Santos[6], Paulo Monteiro[1], Nádia Martins[1], Iva Brito[4]
[1]Rheumatology Department, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [2]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [3]Paediatric department, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Paediatric and Young Adult Rheumatology Unit, Centro Hospitalar Universitário de São João, Porto, Portugal, [5]Obstetrics department, Centro Hospitalar Universitário de São João, Porto, Portugal, [6]Obstetrics department, Centro Hospitalar Tondela-Viseu, Viseu, Portugal

Purpose: Pregnancy in women with Systemic Lupus Erythematosus (SLE) carries a higher materno-fetal risk compared to healthy women. The purpose of this study was the evaluation of pregnancy outcomes in SLE patients.

Methods: Retrospective study including pregnant women with SLE followed in two Portuguese Rheumatology departments. Demographic and clinical data were collected including pregnancy risk factors, disease activity according to SLEDAI-2K and materno-fetal outcomes.

Results: Thirty pregnancies of 27 women were included. The average maternal age was 33.9 years, and the average disease duration was 81.6 months. Most of the patients (n=21; 77.8%) received preconception counselling, with 89.7% (n=26) presenting low or no disease activity at conception. Lupus nephritis was pres-

ent in 20.0% (n=6) and antiphospholipid syndrome in 13.3% (n=4). In 7 pregnancies (23.3%) the patients were positive for anti-SSA antibodies, meeting criteria for Sjögren's syndrome in 3 (10.0%). 36.7% (n=11) had antiphospholipid antibodies. 23.3% (n=7) were overweight and 16.7% (n=5) obese. In 23.3% (n=7) there was prior hypertension. Hydroxychloroquine was used in nearly all gestations (n=28; 93.3%) with two exceptional cases due to toxidermia and to low ophthalmic functional reserve. Azathioprine was used in 50% (n=15), tacrolimus in 3.3% (n=1) and corticosteroids in 60% (n=18). One pregnancy (3.3%) occurred under methotrexate suspended afterwards. Acetylsalicylic acid was used in 86.7% (n=26) and enoxaparin in 23.3% (n=7). Two patients (6.7%) with known lupus nephritis had a disease flare during pregnancy. 82.1% (n=23) of the pregnancies resulted in live births, the majority at full term (n=17; 73.9%) and non-instrumental vaginal deliveries (n=10; 43.5%). The average gestational age at birth was 37.6 weeks and the neonates' average weight was 2866g. A miscarriage rate of 16.7% (n=5) and a preterm delivery rate of 26.1% (n=6) were found. Fetal growth restriction (FGR) complicated 30.4% (n=7) of gestations and two serious maternal infections were reported. There were no cases of maternal, fetal or perinatal deaths, preeclampsia, neonatal lupus, fetal malformations, or postpartum disease flare.

Conclusions: Preconception counselling and management improvement have allowed SLE women to have better pregnancy outcomes similar to the general population, except for higher rates of FGR, prematurity and low birth weight.

066 - FATORES PREDITORES PARA INÍCIO DE TERAPÊUTICA BIOTECNOLÓGICA EM DOENTES COM ARTRITE PSORIÁTICA

Ana Margarida Correia[1], Paulo Pereira[1], Carla Campinho Ferreira[1], Emanuel Costa[1], Diogo Esperança Almeida[1], Joana Leite Silva[1], Joana Sousa-Neves[1], José Redondo[1], Ana Ribeiro[1], Marcos Cerqueira[1]

[1]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal

Introdução: A Artrite Psoriática (APso) é uma doença reumática inflamatória crónica que pode apresentar-se de uma forma muito heterogénea, estando disponíveis várias classes terapêuticas para o seu tratamento como são exemplo os fármacos biotecnológicos (bDMARD). Atualmente, são reconhecidos vários fatores de mau prognóstico na APso, nomeadamente o dano estrutural, a elevação de parâmetros inflamatórios, a presença de dactilite e a presença de psoríase ungueal. Contudo, ainda não está completamente estabelecido quais os fatores ao diagnóstico que predizem o início de terapêutica com bDMARD.

Objetivo: Determinar que fatores presentes ao diagnóstico são preditores para início de terapêutica biotecnológica numa população de doentes com Artrite Psoriática.

Métodos: Estudo unicêntrico, retrospetivo, que incluiu doentes seguidos na Consulta de Reumatologia com o diagnóstico de Artrite Psoriática. Foram colhidos dados sociodemográficos, clínicos e analíticos referentes às primeiras duas avaliações clínicas, assim como o início de bDMARD pela patologia articular em qualquer momento do seguimento. A análise estatística dos dados foi feita com recurso ao SPSS, utilizando as regressões logísticas uni e multivariada para identificar quais os fatores ao diagnóstico que se associaram ao posterior início de bDMARD. Foi considerado estatisticamente significativo um valor de $p<0,05$.

Resultados: Foram incluídos 220 doentes, 90 dos quais sob terapêutica com bDMARD à data de colheita dos dados, com um tempo médio de seguimento de 8,16±6,50 anos. A análise descritiva das variáveis incluídas no estudo está apresentada na tabela 1. Em comparação com o grupo de doentes que não iniciou bDMARD, aqueles que iniciaram foram mais frequentemente mulheres (p=0,015; OR 0,506; IC95%[0,292-0,877]), com diagnóstico em idade mais jovem (p<0,001; OR 0,959; IC95%[0,936-0,982]) e com doença do subtipo AR-like (p=0,002; OR 2,483; IC95%[1,409-4,377]). A proporção de doentes com subtipo AR-like foi superior nas mulheres (47,3% vs 27,1%). Valores de PCR mais elevados ao diagnóstico (p=0,041; OR 1,021; IC95%[1,001-1,042]) e necessidade de corticoterapia em dose intermédia nos primeiros 3 meses de doença (p=0,032; OR 3,629; IC95%[1,119-11,766]) foram também associados ao início de terapêutica com bDMARD. Por outro lado, a presença de hiperuricemia ao diagnóstico (p=0,004; OR 0,108; IC95%[0,024-0,491]) e o subtipo oligoarticular (p<0,001; OR 0,351; IC95%[0,197-0,624]) mostraram uma associação negativa ao início de bDMARD. Na análise multivariada, o valor mais elevado de PCR ao diagnóstico (p=0,019; OR 1,028; IC95%[1,004-1,052]) e a idade mais jovem ao diagnóstico (p= 0,012; OR 0,956; IC95%[0,923-0,990]) mantiveram-se como fatores preditores independentes para o início de terapêutica bDMARD.

Conclusões: No nosso estudo verificou-se que a presença de valores mais elevados de PCR se associa ao início de bDMARD, algo que já está definido como fator de mau prognóstico na APso. Para além disso, demonstrou-se que uma idade mais jovem ao diagnóstico se associa à necessidade de início de terapêutica com bDMARD em algum momento da evolução da doença. Este é um fator até à data não estabelecido, podendo-se especular se resulta de uma doença mais agressiva ou

TO 066 – TABELA 1. Distribuição das variáveis sociodemográficas e clínicas de acordo com o tratamento com bDMARD.

	Tratamento com bDMARD		p-value	OR [IC]
	Não % (N)/M(σ)	Sim % (N)/M(σ)		
Sexo (n=220) Feminino Masculino	34,6 (45) 65,4 (85)	51,1 (46) 48,9 (44)	0,015	ref 0,506 [0,292-0,877]
Profissão (n=195) Non-hard worker Hard worker	57,9 (70) 42,1 (51)	56,8 (42) 43,2 (32)	0,881	-
Tabagismo (n=87) Não-fumador Fumador	71,7 (38) 28,3 (15)	88,2 (30) 11,8 (4)	0,077	-
HTA (n=220) Sem HTA Com HTA	71,5 (93) 28,5 (37)	75,0 (72) 25,0 (18)	0,156	-
Dislipidemia (n=220) Sem dislipidemia Com dislipidemia	72,3 (94) 27,7 (36)	76,7 (69) 23,3 (21)	0,469	-
DM tipo 2 (n=220) Sem DM Com DM	90,0 (117) 10,0 (13)	93,3 (84) 6,7 (6)	0,390	-
Hiperuricemia (n=103) Sem hiperuricemia Com hiperuricemia	67,7 (42) 32,3 (20)	95,1 (39) 4,9 (2)	0,004	ref 0,108 [0,024-0,491]
Idade ao diagnóstico da APso (anos)	48,54 (12,750)	42,66 (10,497)	< 0,001	0,959 [0,936-0,982]
Idade no início dos sintomas (anos)	44,50 (12,817)	38,01 (10,658)	< 0,001	-
Tempo de evolução dos sintomas até ao diagnóstico da APso (meses)	48,18 (64,183)	49,22 (73,433)	0,918	-
Tempo de evolução da psoríase até ao diagnóstico da APso (meses)	188,31 (147,552)	133,29 (119,505)	0,053	-
Subtipo APso (n=220) AR-like Oligoarticular Distal Axial	26,9 (35) 52,3 (68) 3,8 (5) 16,9 (22)	47,8 (43) 27,8 (25) 7,8 (7) 17,8 (16)	0,002 < 0,001 0,216 0,869	2,483 [1,409-4,377] 0,351 [0,197-0,624] - -
Antecedentes (n=219) Dactilite Entesite Uveíte	20,8 (27) 11,5 (15) 1,5 (2)	21,3 (19) 7,9 (7) 2,2 (2)	0,918 0,377 0,702	- - -
Antecedentes de psoríase (n=218) Cutânea Ungueal	79,1 (102) 62,7 (22)	38,6 (64) 33,3 (11)	0,411 0,443	- -
HLA-B27 (n=107) Negativo Positivo	77,3 (51) 22,7 (15)	75,6 (31) 24,4 (10)	1,000	-
Fator Reumatóide (n=142) Negativo Positivo	95,3 (82) 4,7 (4)	100,0 (56) 0,0 (0)	0,153	-
Parâmetros inflamatórios ao diagnóstico VS (mm/h) PCR (mg/L)	23,85 (21,733) 10,91 (15,086)	26,00 (22,946) 17,68 (20,978)	0,566 0,041	- 1,021 [1,001-1,042]
Exame físico ao diagnóstico Nº Articulações Dolorosas (n=134) Nº Articulações Tumefactas (n=135) Nº dactilites (n=172) Nº entesites (n=176)	3,67 (5,954) 3,04 (5,823) 0,49 (1,708) 0,15 (0,494)	3,21 (2,824) 2,65 (2,448) 0,35 (1,116) 0,09 (0,282)	0,053 0,092 0,349 0,325	- - - -
Erosões ao diagnóstico (n=170) Não Sim	80,2 (89) 19,8 (22)	78,0 (46) 22,0 (13)	0,842	-
PDN ≥ 7,5 nos primeiros 3 meses (n=147) Não Sim	94,9 (93) 5,1 (5)	83,7 (41) 16,3 (8)	0,032	ref 3,629 [1,119-11,766]

de um viés de seleção (maior agressividade terapêutica em doentes jovens), e que poderá ser corroborado em estudos posteriores.

068 - CHARACTERIZATION OF ANXIETY AND DEPRESSION AND THEIR IMPACT ON DISEASE ACTIVITY, FATIGUE AND QUALITY OF LIFE IN SPONDYLARTHRITIS PATIENTS TREATED WITH ANTI-TUMOR NECROSIS FACTOR ALPHA AGENTS

Daniela Oliveira[1, 2], Ana Martins[1], Rafaela Nicolau[3], Carlos Vaz[1, 2, 4], Teresa Martins-Rocha[1, 4], Alexandra Bernardo[1], Miguel Bernardes[1, 4], Lúcia Costa[1]

[1]Rheumatology Department, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Center for Health Technology and Services Research (CINTESIS),, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, Porto, Portugal, [3]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [4]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal

Background: The psychological health of patients with spondylarthritis (SpA) influences their response to anti-tumor necrosis factor alpha (anti-TNF-α) therapy. However, little is known about the correlation between anxiety and depression symptoms and clinical outcomes over time. Hence based on clinical practice setting this study aimed to explore the impact of anxiety and depression symptoms on clinical outcomes in SpA patients treated with anti-TNF-α agents. **Methods:** An observational retrospective cohort study was conducted. Adult patients with diagnosis of SpA, according to ASAS classification criteria, who started their first anti-TNFα agent between 2002-2022 were included. Sociodemographic, clinical and laboratory data were obtained from the national register Reuma.pt at the baseline and after 12 months (M). Anxiety and depression symptoms were assessed by Hospital Anxiety and Depression Scale (HADS). A score ≥8 on the HADS-Anxiety and HADS-Depression indicates the presence of clinically significant anxiety and depression symptoms, respectively. Ankylosing Spondylitis (AS) Disease Activity Score with CRP (ASDAS-CRP) and Bath AS Disease Activity Index (BASDAI) were assessed to measure disease activity. Pain Visual-Analogue-Scale (VAS), Bath AS Functional Index (BASFI) and Bath AS Metrological Index (BASMI) were collected to assess pain severity and disability. To evaluate enthesitis, the Maastricht AS enthesitis score (MASES) was performed. Clinical response was evaluated by ASDAS response. Fatigue was evaluated using Functional Assessment of Chronic Illness Therapy (FACIT)-Fatigue (score≤39 indicates the presence of clinically significant fatigue) and health-related quality of life with EQ-5D. In order to correlate anxiety and depression symptoms with clinical outcomes, the Pearson coefficient was used. Linear regression model adjusted for age, gender and disease duration was used to assess the impact of anxiety and depression symptoms on clinical outcomes. **Results:** A total of 130 SpA patients (mean age 40.6±10.8 years; 85.4% female; median disease duration 7[4-14] years) were included. Nearly half (50.6%) of patients had anxiety, 33.8% depression and 30% had both symptoms. At the baseline, the median of anxiety and depression symptoms was 8[4.5-12] and 6[3-8], respectively. At the baseline, there were statistically significant correlations between anxiety and depression symptoms and pain-VAS at night (rp=0.47, p=0.03 and rp=0.6, p=0.004, respectively); between depression symptoms and BASMI (rp=-0.34, p=0.006) and between anxiety and depression symptoms and EQ-5D (rp=-0.56, p=0.04 and rp=-0.65, p=0.011, respectively). At 12M, there were statistically significant correlations between anxiety symptoms and BASDAI (rp=0.23, p=0.03), FACIT-Fatigue (rp=-0.68, p<0.001) and EQ-5D (rp=-0.51, p=0.001); and between depression (rp=-0.65, p<0.001) and EQ-5D (rp=-0.59, p<0.001). In the multivariable regression models, depression symptoms at baseline moment predicted VAS at night (β=0.6, p=0.06). At 12M anxiety symptoms predicted BASDAI (β=0.2, p=0.027), FACIT-Fatigue (β=-0.7, p<0.001) and EQ-5D (β=-0.5, p<0.002); depression symptoms also predicted EQ-5D (β=-0.6, p<0.001). **Conclusions:** Anxiety and depression are common conditions in SpA patients. After one year of anti-TNF-α therapy, both symptoms predicted worse quality of life and anxiety also predicted higher disease activity and fatigue. Our results encourage the assessment and monitoring of anxiety and depression symptoms over time in order to design more individualized multidisciplinary approaches.

069 - PREVALENCE OF NEUROPATHIC PAIN AND ITS ASSOCIATION WITH CLINICAL OUTCOMES IN PATIENTS WITH PRIMARY KNEE AND HIP OSTEOARTHRITIS

Daniela Oliveira[1, 2], Ana Martins[1], Joana Silveira[3], Neto FL[4, 5, 6], Lúcia Costa[1], Carlos Vaz[1, 2, 3], Joana Ferreira-Gomes [4, 5, 6]

[1]Rheumatology Department, Centro Hospitalar São João, Porto, Portugal, [2]Center for Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Porto, Portugal, Porto, Portugal, [3]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [4]Departamento de Biomedicina - Unidade de Biologia Experimental, Faculdade de Medicina do Porto, Universidade do Porto, Porto, Portugal, [5]Pain Group, Instituto de Biologia Molecular e Celular (IBMC), Porto, Portugal, [6]i3S - Instituto de Investigação e Inovação em Saúde, Universidade do Porto, Porto, Portugal

Background: Osteoarthritis (OA) is a common musculoskeletal disease associated with significant chronic pain and physical disability. The presence of pain with a neuropathic component may be associated with different phenotypes of this condition. Thus, this study aimed to explore the prevalence of neuropathic pain (NP) and its association with sociodemographic characteristics and clinical outcomes (pain severity and disability), in patients with primary knee and hip OA. **Methods:** A cross-sectional study involving adult patients with a primary knee and hip OA diagnosis according to American College of Rheumatology (ACR) was conducted. Sociodemographic variables, symptoms and disease duration and pain characteristics were obtained. Pain severity was assessed with Pain Visual Analog Scale (VAS) and functional disability with Health Assessment Questionnaire (HAQ). Western Ontario and McMaster Universities Arthritis Index (WOMAC) was used to assess pain, stiffness and physical function. The pain DETECT scale was used to evaluate NP in these patients. In order to correlate NP with sociodemographic, clinical and pain characteristics, a correlation coefficient was used- Pearson coefficient. Linear and logistic regression models adjusted for age, gender, symptom duration and diagnosis duration were applied to assess the main predictors of NP. **Results:** A total of 36 patients (mean age of 65.1±8.9 years old; 72.2% female) with primary knee OA (n=13, 36.1%), hip OA (n=4, 11.1%) and both (n=19, 52.8%) were included. Patients had a mean symptoms duration of 13.3±8.7 years and a median disease duration of 5 [3-13] years. The mean Pain VAS was 5.0±3.4, and the median HAQ of 1 [0.65-1.91]. The mean of WOMAC- pain, -stiffness, -physical function and -total were 11.9±4.2, 4.5±2.2, 37.5±15.4 and 53.8±20, respectively. Characteristics of NP were found in 28 (77.8%) patients and the mean of PAINDETECT scale was 31.1±11.9. None of the patients had treatment aimed at NP. There were positive correlations between NP and Pain-VAS (rp =0.63, p=0.001), HAQ (rp =0.33, p=0.005), WOMAC-physical function (rp =0.41, p=0.027) and WOMAC-total (rp =0.38, p=0.049). In the multivariable model, Pain-VAS predicted NP (β=0.76, p=0.014). **Conclusions:** Our study demonstrated that NP is common in patients with OA. The presence of this type of pain is associated with worse clinical outcomes, namely pain severity and functional disability. Hence, these findings encourage the screening of NP in all patients with knee and hip OA in order to implement more appropriated therapeutic strategies.

070 - ANTI-SSA RO52 AND ANTI-RO60 AUTOANTIBODIES: ASSOCIATION WITH CLINICAL PHENOTYPES

Carolina Mazeda[1, 2], Natacha Oliveira[3], Catarina Abreu[4], Vanessa Fraga[4], Ana Isabel Maduro[5], André Saraiva[5], Ines L[5, 6], Carla Campinho Ferreira[7], Ana Margarida Correia[7], Rafaela Nicolau[8], Filipa Farinha[9], Ingrid Villanueva[10], Diogo Jesus[6, 11], Pedro Abreu[12], Agna Neto[13], Joana Silva-Dinis[14], Anabela Barcelos[1, 2, 15]

[1]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal, [2]Rheumatology Department, Centro Hospitalar do Baixo Vouga, EPE, Aveiro, Aveiro, Portugal, [3]Department of Mathematics, Universidade de Aveiro, Aveiro, Portugal, [4]Reumatology Department, Hospital Garcia de Orta, EPE, Almada, Portugal, [5]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal, [6]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal, [7]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [8]Serviço de Reumatologia, Centro Hospitalar São João, Porto, Portugal, [9]Rheumatology Department, Hospital Distrital de Santarém, Santarém, Portugal, [10]Clinical Pathology Department, Hospital Distrital de Santarém, Santarém, Portugal, [11]Serviço de Reumatologia, Centro Hospitalar de Leiria, Leiria, Portugal, [12]Rheumatology Department, Unidade Local de Saúde de Castelo Branco, Castelo Branco, Portugal, [13]Serviço de Reumatologia, Hospital Central do Funchal, Funchal, Portugal, [14]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Central, Lisboa, Portugal, [15]Comprehensive Health Research Center (CHRC), NOVA Medical School, University of Lisbon, Lisboa, Portugal

Background: Anti-SSA autoantibodies can be differentiated according to their antigenic target proteins as anti-Ro60 (60 kDa) or anti-Ro52 (52 kDa). Anti-SSA(Ro60) are clearly associated with Connective Tissue Diseases (CTD), but the clinical significance of anti-SSA(Ro52) remains unclear.
Objective: To analyze the disease phenotype of patients with anti-Ro52 and/or anti-Ro60.
Methods: Multicenter, cross-sectional study of anti-Ro52 and/or Ro60 positive patients followed at 10 Rheumatology centers from January 2018 until December 2021. Patients were categorised into 3 groups: group 1 (Ro52+/Ro60-); group 2 (Ro52-/Ro60+); group 3 (Ro52+/Ro60+). Antinuclear antibodies were evaluated by indirect immunofluorescence assay and further screened for anti-extractable nuclear antigen (ENA) antibodies. Demographics and clinical data were compared between the 3 groups, by patients' medical chart review. Univariate analysis was performed using chi-square, Fisher's exact or Kruskall-Wallis test. Subsequently, the Bonferroni test was used to identify intergroup differences (level of significance: p<0.0167). Univariate logistic regression was used to calculate the odds ratio with a 95% confidence interval (CI).
Results: We included 776 patients [female: 83.1%; median age: 59 (46-71) years]. Groups 1, 2 and 3 com-

TO 070 – Table 1. Characteristics of the study population according to the groups of anti-SSA(Ro) positivity.

	Group 1 (n=241)	Group 2 (n=253)	Group 3 (n=282)	p
Age, median (IQR)	64 (52-76)	56 (44-67)	57 (44-69)	<0.001
Female, n (%)	185 (76.8)	214 (84.6)	246 (87.2)	0.005
Other anti-ENA, n (%)				
Anti-La	24 (10)	50 (19.8)	114 (40.4)	<0.001
Anti-RNP	11 (4.6)	23 (9.1)	17 (6.0)	0.115
Anti-Scl70	3 (1.2)	4 (1.6)	6 (2.1)	0.146
Anti-Jo1	7 (3)	1 (0.4)	3 (1.1)	0.070
Anti-Sm	1 (0.4)	7 (2.8)	6 (2.13)	0.098
Anti-dsDNA	11 (4.6)	39 (15.4)	37 (13.1)	<0.001
Anti-centromere	12 (5)	3 (1.2)	6 (2.1)	0.026
Lupus anticoagulant	10 (4.2)	32 (12.7)	23 (8.2)	0.008
Anti-cardiolipin	8 (3.3)	10 (3.9)	23 (8.2)	0.024
Anti-β2 glycoprotein 1	6 (2.5)	10 (3.9)	10 (3.6)	0.736
Rheumatoid Factor	46 (19.1)	44 (17.4)	81 (28.7)	0.001
Anti-CCP	11 (4.6)	15 (5.9)	19 (6.7)	0.327
Non-rheumatologic disease, n (%)	77 (32)	35 (13.8)	30 (10.6)	<0.001
Infections	11 (14.3)	2 (5.7)	1 (3.3)	0.192
Neoplasms	22 (28.6)	3 (8.6)	6 (20.0)	0.057
Interstitial lung disease	5 (6.5)	4 (11.4)	0	0.168
Other diseases	46 (59.7)	25 (71.4)	22 (73.3)	-
Immune-mediated rheumatologic disease, n (%)	164 (68.1)	218 (86.2)	252 (89.4)	<0.001
Sjögren syndrome	92 (56.1)	88 (40.3)	150 (59.5)	<0.001
Systemic lupus erythematosus	20 (12.2)	61 (28)	59 (23.4)	0.001
Systemic sclerosis	11 (6.7)	7 (3.2)	8 (3.2)	0.150
Inflammatory myositis	15 (9.2)	2 (0.9)	2 (0.8)	<0.001
Rheumatoid arthritis	18 (11)	17 (7.8)	16 (6.4)	0.234
Undifferentiated connective tissue disease	11 (6.7)	35 (16.1)	21 (8.3)	0.004
Mixed connective tissue disease	6 (3.7)	6 (2.8)	4 (1.6)	0.406
Other diseases	9 (5.5)	8 (3.7)	10 (4.0)	-

prised 31.1%, 32.6%, and 36.3% of the patients, respectively. Characteristics of the groups are presented in table 1. Anti-Ro52 alone is more frequently associated with non-rheumatic diseases, older age, and men (p<0.05). Among patients with CTD, the diagnosis of systemic lupus erythematosus is 3 and 2 times more prevalent in groups 2 and 3, respectively, than in group 1 [OR 2.8 (95% CI 1.60, 4.97),p=<0.001; OR 2.2 (95% CI 1.28, 3.86), p=0.007]. In group 2, the diagnosis of undifferentiated connective tissue disease is more frequent than in the other groups. The presence of isolated Ro52+ is more frequently associated with inflammatory myositis than in group 2 [OR 0.09 (95% CI 0.01, 0.33), p=<0.001] or group 3 [OR 0.08 (95% CI 0.01, 0.29), p=<0.001]. Group 1 was also more frequently associated with arthritis (p=0.006), interstitial lung disease (p=0.002), and myositis (p=0.009).

Conclusion: Anti-Ro52+ alone is frequently found in patients with non-rheumatic diseases. In addition, anti-Ro52+ is also prevalent in patients with CTD and associated with clinical phenotypes that are different from anti-Ro60+.

071 - THE REAL IMPACT OF AGE ON PSORIATIC DISEASE: A PORTUGUESE MULTICENTER STUDY

Carolina Mazeda[1, 2], Carolina Vilafanha[1, 2], Pedro Miguel Teixeira[1, 2], Catarina Abreu[3], Tiago Meirinhos[4], Rafaela Nicolau[5], Maria Pontes Ferreira[6], João Rovisco[7], Daniela Oliveira[8, 9, 10], Anabela Barcelos[1, 2, 11]

[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro,

Portugal, [3]Reumatology Department, Hospital Garcia de Orta, EPE, Almada, Portugal, [4]Rheumatology Unit, Centro Hospitalar do Tâmega e Sousa, Penafiel, Portugal, [5]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [6]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de

TO 071 – Table 1. Disease characteristics of patients stratified by the age at psoriatic arthritis onset.

	YOPsA (n=301)	LOPsA (n=242)	p
Current age, years (mean±SD)	48.2±9.3	68.4±7.7	<0.001
Sex (M/F), n	165/136	135/106	0.516
Age of diagnosis, years (mean±SD)	37.8±7.9	58.7±6.5	<0.001
Disease duration, years (mean±SD)	10.5±5.2	9.7±4.6	0.193
Comorbidities, n (%) Arterial Hypertension Dyslipidemia Diabetes Mellitus Obesity Hyperuricemia	 38 (12.6) 29 (9.6) 11 (3.7) 8 (2.7) 2 (0.7)	 69 (28.5) 61 (25.2) 38 (15.7) 9 (3.7) 10 (4.1)	 <0.001 <0.001 <0.001 0.468 0.007
Smoking status, n (%)	41 (13.6)	26 (10.7)	0.013
Psoriasis, n (%)	211 (70.1)	165 (68.2)	0.425
Nail involvement, n (%)	85 (28.2)	36 (14.9)	<0.001
Axial involvement, n (%)	76 (25.2)	59 (24.4)	0.919
Dactylitis (ever), n (%)	102 (33.9)	44 (18.2)	<0.001
Enthesitis (ever), n (%)	78 (25.9)	47 (19.4)	0.093
Positivity HLA-B27, n (%)	30 (10)	25 (10.3)	0.084
Family history, n (%)	55 (18.3)	42 (17.4)	0.909
Type of clinical pattern, n (%) Asymmetric oligoarthritis Symmetric polyarthritis Predominant distal interphalangeal joint Mutilans arthritis	 93 (31) 162 (53.8) 22 (7.3) 3 (1.0)	 69 (28.5) 154 (63.6) 1 (0.4) 4 (1.7)	0.001
DAS28-CRP at baseline, mean±SD	3.6±1.4	3.6±1.1-	0.909
DAS28-ESR at baseline, mean±SD	3.9±1.4	3.9±1.3	0.339
ESR at baseline, mean±SD	29.3±28.1	14.2±14.8	0.061
CRP at baseline, mean±SD	2.1±3.5	2.3±2.6	0.115
PGA at baseline, mean±SD	52.1±27.8	58.1±21.4	0.365
HAQ at baseline, mean±SD	0.6±0.7	0.7±0.6	0.531
DAS28-CRP, mean±SD*	2.4±1.1	2.4±1.1	0.339
DAS28-ESR, mean±SD*	2.5±1.4	2.8±1.3	0.006
ESR, mean±SD*	14.2±14.8	18.9±16.6	0.001
CRP, mean±SD*	0.7±1.5	0.9±3.55	0.038
PGA, mean±SD*	35.0±25.8	40.5±26.9	0.05
HAQ, mean±SD*	0.5±0.7	0.7±0.6	0.005
Therapeutic, n (%) cDMARDs bDMARDs	 202 (67.1) 84 (27.9)	 178 (73.5) 117 (48.3)	 0.084 <0.001

*after two years of follow-up

Lima, Portugal, [7]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [8]Rheumatology Department, Centro Hospitalar de São João, Porto, Portugal, [9]Department of Medicine, Faculty of Medicine, University of Porto, Porto, Portugal, [10]Center for Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Porto, Portugal, Porto, Portugal, [11]Comprehensive Health Research Center (CHRC), NOVA Medical School, University of Lisbon, Lisboa, Portugal

Background: Psoriatic Arthritis (PsA) is an immune-mediated disease that additionally to the predominant skin and musculoskeletal features, comorbidities such as metabolic syndrome are common. This type of comorbidities increases significantly the burden of the disease, reduces quality of life, and affects negatively the clinical response to the treatment. Ageing adds another variable to this composite as there appears to be evidence of clinical, genetic, and histopathological differences between early and late-onset PsA.
Objectives: To compare the socio-demographics and clinical data in PsA patients with a younger-onset (YOPsA) and late-onset (LOPsA) of disease and to evaluate disease activity and function at presentation and during a follow-up of two years.
Methods: A multicenter cohort study including adult PsA patients with peripheral involvement registered in the Rheumatic Diseases Portuguese Register (Reuma.pt) up to December 2022 was performed. Patients were divided into 2 groups according to the onset age of PsA: YOPsA (<50 years) and LOPsA (≥50 years). Univariate analysis was performed followed by multivariate analysis.
Results: A total of 543 [YOPsA, 301 (55.4%); LOPsO, 242 (44.6%)] patients with peripheral PsA were included. Demographic and clinical data is represented in table 1. Compared to patients with LOPsA, PsA patients with early onset showed more frequently dactylitis and nail involvement (p<0.001). Regarding the comorbidities, there was a higher frequency of arterial hypertension, dyslipidemia, diabetes mellitus and hyperuricemia in LOPsA patients (p<0.05). After 2 years of follow-up, LOPsA patients had a worse Disease Activity Score-28 with Erythrocyte Sedimentation Rate (DAS-ESR) (2.8 vs 2.5, p=0.006), higher Patient Global Assessment (PGA) (40.5 vs 35, p=0.05), higher levels of C -Reactive Protein (CRP) (0.9 vs 0.7, p=0.038) and ESR (18.9 vs 14.2, p=0.001) and higher Health Assessment Questionnaire (HAQ) (0.7 vs 0.5, p=0.005). LOPsA patients were treated with more biologic DMARDs (p<0.0001). Adjusting for age, the late onset of PsA was associated with the presence of traditional cardiovascular risk factors.
Conclusion: Our study suggests that the age at onset of PsA seems to be an important covariate that might affect the clinical and laboratory manifestations of the disease and its clinical outcomes.

072 - WHAT IS THE REAL IMPACT OF DEPRESSION ON CLINICAL RESPONSE TO THERAPY IN PATIENTS WITH PSORIATIC ARTHRITIS TREATED WITH BIOLOGIC DISEASE-MODIFYING ANTIRHEUMATIC DRUGS?

Ana Martins[1,2], Daniela Oliveira[1,2], Rafaela Nicolau[2,3], Teresa Martins-Rocha[1,2], Alexandra Bernardo[1], Sofia Pimenta[1,2], Miguel Bernardes[1,2], Lúcia Costa[1]
[1]Serviço de Reumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [3]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal

Background: The estimated prevalence of depression in patients with psoriatic arthritis (PsA) ranges from 20 to 30% and is higher than in the general population. The relationship between depression, systemic inflammation, disease activity and response to therapy in PsA patients remains unclear and under investigation. Patients with depression seems to have overexpression of pro-inflammatory cytokines involved in the pathogenesis of PsA. On the other hand, depression leads to pain sensitization and plays a pivotal role in the shaping of pain responses.
Objectives: To compare the response to therapy in PsA patients with or without depression, treated with biologic disease-modifying antirheumatic drug (bDMARD) for the first time.
Methods: A retrospective cohort study of patients with PsA (according to CASPAR criteria) registered in the national database (Reuma.pt) who started their first bDMARD was conducted. Demographic data, disease activity data, functional parameters and response to therapy 12 months after the start of bDMARD were collected.
Depression was defined according to Hospital Anxiety and Depression scale (HADS) or diagnosed previously by a psychiatrist. For patients with peripheral involvement, DAS28 (4V), CDAI and SDAI were collected at baseline. For patients with axial involvement, ASDAS-CRP was also collected. ACR response criteria, change in ASDAS(ΔASDAS), BASDAI50 response, change in HAQ(ΔHAQ) and EULAR response (good, moderate or no response) were calculated at month 12 (T12). Chi-square test and t-test were conducted and p-value <0.05 was considered statistically significant.
Results: A total of 129 PsA patients were included, 66(51.2%) females, with a mean age of 52.2±10.9 years and disease duration of 13.9±7.2 years. Regarding PsA

TO 072 – TABLE 1. Clinical response to therapy at T12 in psoriatic arthritis patients with and without depression.

	With depression (n=32)	Without depression (n=97)	P-value
With peripheral involvement	N=29	N=95	
ACR response, n (%) ACR20 ACR50 ACR70	 17 (58.6) 9 (31.0) 5 (17.2)	 82 (86.3) 60 (63.1) 49 (51.6)	 0.001 0.002 0.001
DDAS 28, mean ± ST	1.1 ± 1.3	2.1 ± 1.4	0.001
DHAQ, mean ± ST	-0.3 ± 0.6	-0.4 ± 0.6	0.69
EULAR response Good response Moderate response No response	 7 (24.1) 11 (37.9) 11 (37.9)	 57 (60.0) 24 (25.3) 14 (14.7)	 0.002
With axial involvement	N=25	N=72	
ASDAS response criteria Major improvement (DASDAS ≥ 2.0) Clinically important improvement (DASDAS ≥ 1.1) No improvement	 3 (12.0) 7 (28.0) 15 (60.0)	 26 (36.1) 22 (30.6) 24 (33.3)	 0.031
BASDAI50 response, n (%)	10 (13.3)	44 (61.1)	0.067
DASDAS, mean ± ST	-0.9 ± 1.2	-1.7 ± 1.1	0.006
All patients			
Switch due to inefficacy in the first year, n (%)	9 (28.1)	7 (7.2)	0.002

subtypes, 70 (54.3%) had symmetric polyarthritis, 28 (21.7%) asymmetric oligoarthritis, 28 (21.7%) predominant axial involvement, 2 (1.5%) distal interphalangeal predominant involvement and 1 (0.8%) arthritis mutilans. All patients were treated with bDMARD, 122 (94.6%) with anti-TNFα agents and 7 (5.4%) with anti-IL-17 agents. Thirty-two (24.8%) patients had depression and the mean value of HADS was 10.0±3.8. The majority of these patients were treated with antidepressants (n=25, 78.1%). Patients with and without depression had similar sociodemographic characteristics, except for gender (more females in the depression group, p<0.001). These 2 groups had similar disease activity scores at baseline. Patients with depression and peripheral involvement had a significantly lower ACR20/50/70 responses (p=0.001, p=0.002 and p=0.001 respectively) and poorer EULAR response (p=0.002) at T12. Furthermore, patients with depression and axial involvement had a lower Δ-ASDAS (p=0.03) and a lower BASDAI50 response (p=0.06) at T12. Switch due to ineffectiveness at T12 was higher in patients with depression (p=0.002).

Conclusion: In our study, depression was associated with poorer response to therapy and higher bDMARD discontinuation rates. Depression might be associated with a pro-inflammatory state but also has a close relationship with chronic widespread pain which can influence some composite disease activity measures. Clinical judgement of the degree of disease activity, using objective measures of disease activity, is important in patients with depression, in order to avoid inappropriate escalation of therapy. Furthermore, recognition and treatment of depression in patients with PsA are crucial, as this might improve their quality of life and their ability to manage their rheumatic disease.

073 - LUNG INVOLVEMENT IN RHEUMATOID ARTHRITIS: THE CURRENT PORTUGUESE PORTRAIT

Ana Catarina Duarte[1], Bruno Miguel Fernandes[2], Miguel Bernardes[2, 3], Catarina Tenazinha[4, 5], Patrícia Martins[4, 5], Carolina Mazeda[6], Beatriz Mendes[7], Francisca Guimarães[8], Laura Gago[9], Ana Margarida Correia[10], Ana Valido[11], Lígia Silva[12], Luisa Brites[13], Tiago Meirinhos[14], Filipe Araújo[15], Patrícia Nero[16], Margarida Cruz[17], Lídia Teixeira[18], Maria José Santos[5, 19]

[1]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [2]Serviço de Reumatologia, Centro

Hospitalar de São João, Porto, Portugal, [3]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [4]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [5]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [6]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [7]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [8]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [9]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [10]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [11]Serviço de Reumatologia, Unidade Local de Saúde do Litoral Alentejano, Santiago do Cacém, Portugal, [12]Serviço de Reumatologia, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal, [13]Serviço de Reumatologia, Centro Hospitalar de Leiria, Leiria, Portugal, [14]Rheumatology Unit, Centro Hospitalar do Tâmega e Sousa, Penafiel, Portugal, [15]Rheumatology and Osteopororis Unit, Hospital de Sant Ana, Lisboa, Portugal, [16]Rheumatology Department, Hospital CUF Descobertas, Lisboa, Portugal, [17]Serviço de Reumatologia, Centro Hospitalar de S. Francisco, Leiria, Portugal, [18]Serviço de Reumatologia, Hospital Central do Funchal, Funchal, Portugal, [19]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal

Introduction: Lung involvement is expected in 7-80% of rheumatoid arthritis (RA) patients and can affect almost all lung compartments. The lower prevalence of lung disease reported in Portugal and the fact that it remains an important cause of morbi-mortality in RA, makes it crucial to raise awareness for this complication. We aim to characterize lung involvement in a nationwide cohort of RA patients, identify factors associated with lung disease and analyze the current standard of care in interstitial lung disease (ILD) treatment.

Methods: Observational, retrospective, multicenter study of patients prospectively followed in the Rheumatic Diseases Portuguese Registry (Reuma.pt).Data were collected until May 2023. Lung involvement was defined by the presence of imagiological/histopathological changes described in the spectrum of RA-lung disease. Parametric and nonparametric tests were used for group comparison and logistic regression analysis to identify features independently associated with lung disease.

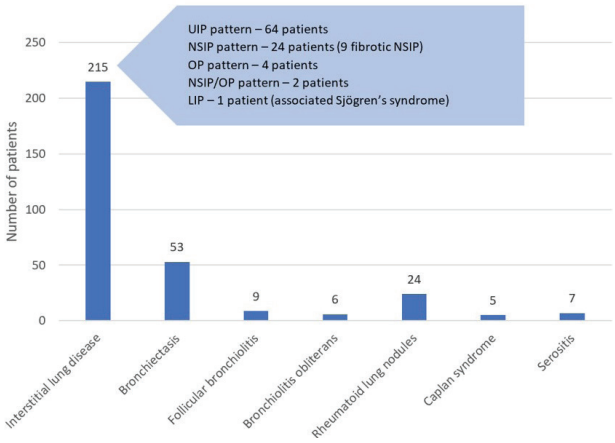
Results: In total 10403 patients with RA were included, 8221 (79%) female, with mean age at last visit of 62.2±13.5 years. Median disease duration was 12.5 [IQR 6.4-20.8] years. Lung disease was documented in 307 (2.95%) patients and the median interval between joint and pulmonary symptoms was 5.8 [IQR 1-15.3] years. Mean DAS-28-ESR at lung disease diagnosis (± 3 months) was 4.36±1.46 (85.3% missing data).

The distribution of the different types of lung involvement is represented in graphic 1.

TO073 – Table 1.

	With lung involvement (N=307)	Without lung involvement (N=10096)	Percentage of missing data	p-value
Male	93 (30.3%)	2089 (20.7%)	0%	< 0.001
Age at RA diagnosis	56.7±13.6	49.6±14.8	23.3%	< 0.001
RA duration	13.6 [IQR 7-23.3]	12.5 [IQR 6.3-20.7]	29.1%	0.018
BMI (kg/m^2)	23.4 [IQR 23.4-27.9]	26.2 [IQR 23.4-29.7]	59.2%	0.629
Ever smoking	102 (39.4%)	1566 (26.9%)	41.6%	< 0.001
Occupational exposure risk	43 (21.8%)	618 (16.3%)	61.7%	0.049
Years of education	4 [IQR 4-9]	6 [IQR 4-12]	60.6%	0.01
Positive RF	239 (82.1%)	5258 (70.3%)	25.3%	< 0.001
Positive ACPA	238 (85.6%)	4254 (68.5%)	37.6%	< 0.001
Erosive disease	168 (65.6%)	3425 (58.5%)	41.3%	0.027
SS	23 (7.5%)	344 (6.6%)	47.2%	0.556
Previous treatment with MTX	188 (71.5%)	6216 (82.4%)	25%	< 0.001
Previous treatment with TNFi	56 (21.3%)	3034 (40.2%)	25%	< 0.001

Legend: RA – rheumatoid arthritis; BMI – body mass index; RF – rheumatoid factor; ACPA - anti-citrullinated peptide antibody; SS – associated Sjögren's syndrome; MTX – methotrexate; TNFi – tumour necrosis factor inhibitor.
Continuous variables are expressed as mean ± S.D. if they had a normal distribution, or median with interquartile range (IQR) if not normally distributed. Categorical variables are presented as absolute values (n) and frequencies (%).



TO 073 – Figure 1. Graphic 1- Distribution of the different types of lung involvement in RA patients

The comparison of several variables between patients with and without lung involvement is illustrated in table 1.
Ever smoking (OR=2.01;[95%CI:1.41-2.88],p<0.001), positive anti-citrullinated peptide antibodies (ACPA) (OR=2.42;[95%CI:1.44-4.05], p<0.001), erosive disease (OR=1.46;[95%CI:1.04-2.03], p=0.028) and older age at RA diagnosis (OR=1.03 per year;[95%CI:1.02-1.04],p<0.001) were positively associated with lung disease, whereas previous treatment with methotrexate (MTX) (OR=0.35; [95%CI:0.25-0.5],p<0.001) and tumour necrosis factor inhibitors (TNFi) (OR=0.51;[95%CI:0.36-0.73],p<0.001) had a negative association.
From the 39 RA-ILD patients ever treated with biologics before ILD diagnosis, TNFi were the most prescribed (31; 79.5%), followed by tocilizumab (7 patients; 17.9%) and rituximab (RTX; 6 patients; 15.4%). After ILD diagnosis, RTX became the most prescribed biologic (70/100 patients; 70%), from which 47 were biologic-naĬve. Eight patients (5.6%) were started on abatacept after ILD diagnosis. Antifibrotics were used in 25 patients (19 nintedanib, 8 pirfenidone).
From the 16 patients with lung disease who died of a known cause, 2 were related to ILD progression and 10 to respiratory infection.
From the 16 patients with lung disease who died of a known cause, 2 were related to ILD progression and 10 to respiratory infection.
Conclusion: The prevalence of RA-associated lung disease in our Reuma.pt cohort was lower compared to other series in the literature, which might be explained by underreporting and because lung disease is mostly screened after development of respiratory symptoms. ILD, namely UIP, was the most prevalent type of lung disease.
Smoking, positive ACPA, presence of erosions and older age at RA diagnosis were positively associated with presence of lung disease, whereas previous treatment with MTX and TNFi seemed protective.
Patient education and clinician awareness, alongside a preclinical screening strategy (especially for those at higher risk of RA-associated lung disease) would allow for a timely diagnosis and treatment.

074 - GISCASPA – STUDY OF SUBCLINICAL GUT INVOLVEMENT IN AXIAL SPONDYLOARTHRITIS

Carolina Mazeda[1, 2], Sofia Ferreira Azevedo[1, 2], Susana P. Silva[1, 2], Joao Paulo Vilas-Boas[1, 2], C. Pinto Oliveira[1, 2], Bernardo Santos[1], Ana Rita Prata[1, 2], Gisela Eugénio[1, 2], Inês Cunha[1, 2], Anabela Barcelos[1, 2]
[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, EPE, Aveiro, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Introduction: Axial spondyloarthritis (axSpA) has a predilection for the axial skeleton and belong to the group of Spondyloarthritis, a set of inflammatory rheumatic diseases with common clinical, radiologic, and serologic features in which extraarticular manifestations are frequent, including inflammatory bowel disease (IBD). The link between SpA and IBD has been recognized for decades, with 5-10% of axial SpA patients suffering from concurrent IBD. But, emerging evidence suggests that subclinical gut inflammation in patients with SpA is an important pathophysiological event that plays a role in disease pathogenesis. However, it is difficult to detect subclinical gut inflammation since it is largely asymptomatic. Faecal calprotectin (FC) is a very sensitive marker for inflammation in the gastrointestinal tract. The role of FC in patients with SpA is not clearly defined, but some studies suggest that FC levels may predict the onset of IBD and may be related to disease activity.
Objective: To test the validity of FC as a marker of intestinal inflammation in patients with axSpA and to investigate factors associated with increased FC levels in patients with axSpA.
Methods: Patients involved fulfilled ASAS classification criteria for axSpA and were divided into two groups - ankylosing spondylitis (AS) or non-radiographic axSpA (nr-axSpA); a control group without rheumatologic disease or IBD was also included. All patients and controls answered a structured questionnaire about the presence of gastrointestinal symptoms - Red Flags Questionnaire for IBD and underwent FC measurement with enzyme-linked immunosorbent assay (≥50 mg/g was considered positive). Patients treated with oral NSAIDs should undergo adequate washout (at least 3 weeks). In

TO074 – TABLE 1. Characteristics of the population according to the groups of axSpA and controls.

	AS (n=34)	axSpA (n=30)	Controls (n=25)	P*
Age (years), mean±SD	55.5±14.7	44.9±13.3	40.2±14.0	0.004
Sex (F/M)	15/19	18/12	13/11	0.205
Age at disease onset (years), mean±SD	11.8±7.7	5.4±3.3	-	<0.001
Comorbidities, n (%)				
Arterial Hypertension	13(38.2)	10(33.3)	1(4)	0.166
Dyslipidemia	8(23.5)	7(23.3)	2(8)	0.985
Diabetes Mellitus	3(8.8)	2(6.7)	0	0.748
Obesity	1(2.9)	1(3.3)	0	
Smoking status, n (%)	4(11.8)	1(3.3)	1(4)	0.850
Peripheral involvement, n (%)	9(26.5)	5(16.7)	-	0.344
Dactylitis (ever), n (%)	0	1(3.3)	-	0.283
Enthesitis (ever), n (%)	1(2.9)	2(6.7)	-	0.482
Uveitis (ever), n (%)	9(26.5)	4(13.3)	-	0.192
Positivity HLA-B27, n (%)	25(73.5)	13(43.3)	-	0.014
BASDAI, mean±SD	3.4±2.5	3.8±2.4	-	0.589
BASFI, mean±SD	3.3±2.5	3.3±2.1	-	0.981
ASDAS-PCR, mean±SD	2.3±1.0	2.4±1.1	-	0.07
BASMI, mean±SD	4.2±1.8	3.1±1.7	-	0.226
Therapeutic, n (%)				
AINEs**	16 (47.1)	14 (46.7)	-	0.124
cDMARDs	2 (5.9)	2 (6.7)		0.897
bDMARDs	9 (26.5)	11 (36.7)		0.408

*Comparison between nr-axSpA and AS groups. ** washout 3 weeks before collecting stool.

axSpA patients, BASDAI, BASFI, ASDAS-PCR and BASMI were recorded. A general analysis was performed; p-value ≤0.05 was statistically significant.

Results: We included 64 patients (34 with AS and 30 with nr-axSpA) and 25 controls. Demographic, clinical data is represented in table 1. In AS group, 2.9% had nocturnal diarrhoea, first-degree relative with confirmed IBD and rectal urgency; in nr-axSpA, 6.7% had abdominal pain 30-45 minutes after meals and 3.3% chronic abdominal pain; all controls were asymptomatic. Elevated FC was observed in 32.4% of AS patients (129.2±404.0), 23.3% of nr-axSpA patients (70.9±114.4) and 4.2% of controls (21.9±18.4). FC was significantly higher in each axSpA subtype vs controls (p=0.03). There were no statistically significant differences in ASDAS, BASDAI, BASFI or BASMI scores between axSpA patients with normal vs high FC levels.

Conclusion: In patients with axSpA, gut inflammation measured by FC was higher than among controls and it was also higher in patients with AS than in nr-axSpA. We also found that high FC values were not associated with more pronounced gastrointestinal symptoms. Thus, studies with a larger population and invasive studies are needed to assess the impact that this marker may have on the management of this pathology.

076 - PREDICTORS OF ADVERSE FETAL OUTCOMES IN SYSTEMIC LUPUS ERYTHEMATOSUS

Inês Santos[1], Rafaela Nicolau[1], Tiago Beirão[2], Francisca Guimarães[3], Francisca Aguiar[4], Sara Santos[4], Mariana Rodrigues[4], Anabela Rocha[5], Sofia Bessa[5], Joana Aidos[6], Joana Santos[6], Paulo Monteiro[1], Nádia Martins[1], Iva Brito[4]
[1]Rheumatology Department, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [2]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [3]Paediatric department, Centro Hospitalar Entre Douro e Vouga, Santa Maria da Feira, Portugal, [4]Paediatric and Young Adult Rheumatology Unit, Centro Hospitalar Universitário de São João, Porto, Portugal, [5]Obstetrics department, Centro Hospitalar Universitário de São João, Porto, Portugal, [6]Obstetrics department, Centro Hospitalar Tondela-Viseu, Viseu, Portugal

Purpose: Systemic Lupus Erythematosus (SLE) mainly affects women of childbearing age and increases the

risk for materno-fetal complications during pregnancy. The aim of this study was the description of pregnancy outcomes as well as the identification of predictors of adverse events in pregnant SLE patients.

Methods: Retrospective and longitudinal study including pregnant women with SLE followed in two Portuguese rheumatology departments. Demographic and clinical data were collected. Disease activity was assessed using SLEDAI-2K. Statistical analysis was performed using R version 4.2.2 considering p-values lower than 0.05 as statistically significant.

Results: Thirty pregnancies of 27 women were included. The average maternal age was 33.9 years, and the average disease duration was 81.6 months. Most women (n=26; 89,7%) showed none or low disease activity at conception. Lupus nephritis (LN) was present in 20.0% (n=6) and antiphospholipid syndrome (APS) in 13.3% (n=4). In 7 pregnancies (23.3%) the patients were positive for anti-SSA antibodies, meeting criteria for Sjögren's syndrome in 3 (10.0%). 36.7% (n=11) had antiphospholipid antibodies (APLA). In 23.3% (n=7) there was prior hypertension (HTN). Two patients (6.7%) had a disease flare during pregnancy, both with renal activity in known LN. 82.1% (n=23) of the pregnancies resulted in live births, the majority at full term (n=17; 73.9%) and non-instrumental vaginal deliveries (n=10; 43.5%). The average gestational age at birth was 37.6 weeks and the neonates' average weight was 2866 g. A miscarriage rate of 10.0% (n=3) and a preterm delivery rate of 26.1% (n=6) were found. Fetal grow restriction (FGR) complicated 30.4% (n=7) of the gestations. The analysis of maternal factors on obstetric evolution demonstrated that miscarriage was more frequent in women with HTN (57.1% vs 4.3%, p=0.006), LN (66.7% vs 4.2%, p=0.003), and APLA with APS (75.0% vs 0.0%, p=0.024). No statistically significant difference was found regarding FGR, preterm birth, or low birth weight.

TO 076 – TABLE.

			Miscarriage		
			Yes	No	
Arterial hypertension	Yes	N (%)	5 (57.1%)	3 (42.9%)	p=0.006
	No	N (%)	1 (4.3%)	22 (95.7%)	
Lupus nephritis	Yes	N (%)	4 (66.7%)	2 (33.3%)	p=0.003
	No	N (%)	1 (4.2%)	23 (95.8%)	
Antiphospholipid antibodies with antiphospholipid syndrome	Yes	N (%)	3 (75.0%)	1 (25.0%)	p=0.024
	No	N (%)	0 (0.0%)	7 (100.0%)	

Table 1: Miscarriage rate in pregnant SLE women with arterial hypertension, lupus nephritis, and antiphospholipid antibodies with antiphospholipid syndrome

Conclusions: This study suggests the existence of an increased risk of miscarriage in SLE women with HTN, LN and APLA with APS, in line with existing literature. Even with management improvement, SLE still poses a significant risk for perinatal morbidity, which highlights the importance of a multidisciplinary follow-up.

081 - PREGNANCY IN IDIOPATHIC INFLAMMATORY MYOPATHIES – A CASE SERIES FROM A TERTIARY CENTRE

Ana Rita Lopes[1, 2], Ana Teodósio Chícharo[1, 2, 3], Ana Rita Cruz-Machado[1, 2], Sofia C Barreira[1, 2], Patrícia Martins[1, 2], Luísa Pinto[4], Susana Capela[1, 2]

[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, Faro, Portugal, [4]Departamento de Ginecologia, Obstetrícia e Medicina da Reprodução, Centro Hospitalar Universitário Lisboa Norte, CHULN, Lisboa, Portugal

Objectives: This study aims to describe maternal and perinatal outcomes in women with idiopathic inflammatory myopathies (IIM).

Methods: We report 5 pregnancies in women with IIM followed at our rheumatology-obstetric clinic from 2009 to 2022.

Results: A total of 5 pregnancies in 3 women with pre-existing IIM were identified, while 1 woman was diagnosed during pregnancy. Demographic and clinical data are summarized in Table 1.

Pregnancy 1: 29-year-old woman diagnosed with anti-synthetase syndrome associated with anti-Jo-1 antibodies, with pulmonary involvement - non-specific interstitial pneumonitis (NSIP), articular and cutaneous manifestations. The patient was receiving treatment with mycophenolic acid (MPA) 3g/day, pulse cyclophosphamide 1g/monthly (last administration 2 weeks before conception) and prednisolone (PDN) 20mg/day. Nevertheless, she still reported fatigue with minimal exertion and exhibited high Creatine Kinase (CK) levels (602 U/l). Despite medical advice against conceiving due to active disease and ongoing teratogenic treatment, the patient became pregnant. However, she experienced a spontaneous miscarriage at six weeks of gestation. Both active disease and concomitant use of MPA may have contributed to the pregnancy loss.

Pregnancy 2: 33-year-old woman diagnosed with dermatomyositis associated with anti-Ro-52 antibodies, with previous muscular, cutaneous, and microvascular

TO 081 – TABLE. Clinical background characteristics, treatments and pregnancy outcomes of pregnant patients with inflammatory myopathies.

Pregnancy number	Ethnicity	Age at conception	Obstetric history	Diagnosis	Disease duration, years	Disease status before conception	Pre-pregnancy treatments			Treatment agents during pregnancy				Flare in pregnancy	Flare in postpartum	Outcome	Gestational age at delivery, weeks	Mode of delivery	Birth weight of newborn, g	APO
							GC use	Mean GC dosage, mg/d	Immunosuppressants use	GC use	Mean GC dosage, mg/d	Increase in GC dosage	Immunosuppressants use							
1	Melanodermic	29	G1P0A0	Antisynthetase Syndrome	7	Active	1	20	MPA, Cyclophosphamide	1	20	0	MPA, Cyclophosphamide	0	N/A	Spontaneous abortion	N/A	N/A	N/A	N/A
2	Caucasian	33	G1P0A0	Dermatomyositis	2	Inactive	1	5	Chloroquine Phosphate	1	5	0	Chloroquine Phosphate	0	0	Live birth	39	Distocic	2920	0
3	Caucasian	25	G1P0A0	Necrotizing Myopathy	1	Active	0	0	0	1	5	0	0	0	1	Live birth	40+2	Cesarian	Missing	0
4	Melanodermic	32	G1P0A0	SS/PM Overlap Syndrome	5	Inactive	1	5	AZT, HCQ	1	5	0	AZT, HCQ	0	0	Live birth	37+5	Eutocic	2520	0
5	Melanodermic	33	G2P1A0	SS/PM Overlap Syndrome	5	Active	1	2.5	AZT, HCQ	1	2.5	1	AZT, HCQ	1	N/A	Spontaneous abortion	N/A	N/A	N/A	N/A

GC glucocorticoids , MPA mycophenolic acid , AZT azathioprine , HCQ hydroxychloroquine , APO adverse pregnancy outcomes , N/A not applicable

manifestations including Raynaud phenomenon. At the periconceptional period, under chloroquine phosphate 250 mg/day and PDN 5mg/day she exhibited low disease activity - mild heliotrope and periungual erythema. The treatment was maintained throughout pregnancy, which was uneventful – the disease remained quiescent, and she delivered a healthy baby at 39 weeks.

Pregnancy 3: 25-year-old woman diagnosed with anti-SRP necrotizing myopathy during her first pregnancy due to progressively worsening myalgias and elevated CK (759 U/L) and aldolase (11.3 U/L). The patient was treated with PDN 5mg/day throughout pregnancy. A healthy baby was born at 40 weeks. The patient experienced a disease flare characterized by myalgia and proximal weakness 5 weeks after delivery, requiring an increase of PDN to 10mg/day.

Pregnancy 4: 32-year-old woman diagnosed with Systemic Sclerosis/Polymyositis overlap syndrome associated with anti-PM/Scl-75 antibodies, with predominant clinical features of myositis. The patient got pregnant while taking azathioprine (AZT) 100mg/day, hydroxychloroquine (HCQ) 300mg/day and PDN 5mg/day. The disease remained quiescent throughout the pregnancy and the postpartum.

Pregnancy 5: the woman referred in the previous point became pregnant again at the age of 33. Prior to conception, she was under AZT 100mg/day, HCQ 300mg/day, and PDN 2.5mg/day with no symptoms associated with the disease. In the first trimester of pregnancy, she reported myalgias and muscle weakness, and the blood samples showed elevated levels of CK (2531 U/l) and aldolase (55 U/l). PDN was increased to 7.5mg/day. She experienced a miscarriage at 5 weeks.

No congenital abnormalities or neonatal infections were reported in pregnancy 2, 3 and 4.

Conclusion: Although containing a small sample, our case series on pregnant women with IIM suggests that these patients may face a higher risk of adverse pregnancy outcomes and of disease flares. Achieving disease remission before conception appears to offer a more favourable prognosis. These women should be closely monitored in high-risk pregnancy clinics by a multidisciplinary team, including rheumatologists and obstetricians.

084 - NAILFOLD CAPILLAROSCOPIC CHANGES IN PATIENTS WITH INFLAMMATORY MYOPATHIES

Daniel Carvalho[1, 2, 3], Eduardo Dourado[2, 3], Matilde Bandeira[2, 3], Ana Teresa Melo[2, 3], Bianca Paulo Correia[2, 3], Filipa Marques Costa[2, 3], Lídia Teixeira[1], JE Fonseca[2, 3], Raquel Campanilho-Marques[2, 3], Sofia C Barreira[2, 3]
[1]Rheumatology Department, Hospital Dr. Nélio Mendonça, Funchal, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Nailfold videocapillaroscopy (NVC) is a non-invasive imaging technique to assess microvascular abnormalities in connective tissue diseases. Microcirculation abnormalities have been mainly observed in patients with dermatomyositis (DM) among the idiopathic inflammatory myopathies (IIM), while NVC is usually normal in polymyositis. However, there is limited data on NVC abnormalities in other IIM subtypes with cutaneous and microcirculation involvement.

Objectives: To evaluate the NVC qualitative and semi-quantitative changes association with subtypes and clinical features in patients with IIM.

Methods: A single-center retrospective analysis was conducted, including adult patients diagnosed with IIM who underwent NVC at the discretion of the assistant rheumatologist between 2018 and 2023, includ-

TO 084 – TABLE. Clinical background characteristics, treatments and pregnancy outcomes of pregnant patients with inflammatory myopathies.

Baseline characteristics	All patients (n=40)	Dermatomyositis (n=25)	Antisynthetase syndrome (n=10)	MCTD (n=5)	p-value
Gender					0.713
Female, n (%)	29 (72.5)	17 (68.0)	8 (80.0)	4 (80.0)	
Male, n (%)	11 (27.5)	8 (32.0)	2 (20.0)	1 (20.0)	
Age, mean (S.D.), years	47.4 (17.2)	48.0 (18.3)	48.5 (15.3)	40.0 (17.3)	0.864
Disease duration (years), median (IQR)	4.0 (4.0)	4.0 (5.0)	4.0 (8.0)	4.0 (5.0)	0.796
Disease manifestations, n (%)					
Skin involvement	30 (75.0)	22 (88.0)	5 (50.0)	3 (60.0)	**0.045**
Gottron's papules	15 (37.5)	13 (52.0)	1 (10.0)	1 (20.0)	**0.047**
Raynaud's phenomenon	23 (57.5)	12 (48.0)	6 (60.0)	5 (100.0)	0.098
Arthritis	17 (42.5)	8 (32.0)	7 (70.0)	2 (40.0)	0.120
Muscle involvement	25 (62.5)	15 (60.0)	6 (60.0)	4 (80.0)	0.688
Calcinosis	2 (5.0)	2 (100.0)	0 (0.0)	0 (0.0)	0.532
Pulmonary involvement	13 (32.5)	6 (24.0)	7 (70.0)	0 (0.0)	0.074
Heart involvement	2 (5.0)	2 (8.0)	0 (0.0)	0 (0.0)	0.532
GERD	5 (12.5)	3 (12.0)	1 (10.0)	1 (20.0)	0.852
Dysphagia	6 (15.0)	4 (16.0)	2 (20.0)	0 (0.0)	0.578
Neoplasia	3 (7.5)	3 (12.0)	0 (0.0)	0 (0.0)	0.378
MSA and MAA, n (%)					**<0.001**
ASSD related (PL-7, Jo-1, PL-12, EJ)	8 (20.0)	0 (0.0)	8 (80.0)	0 (0.0)	
Mi-2	6 (15.0)	6 (24.0)	0 (0.0)	0 (0.0)	
MDA-5	3 (7.5)	3 (12.0)	0 (0.0)	0 (0.0)	
TIF-1y	2 (5.0)	2 (8.0)	0 (0.0)	0 (0.0)	
PM	2 (5.0)	2 (8.0)	0 (0.0)	0 (0.0)	
SAE	3 (7.5)	3 (12.0)	0 (0.0)	0 (0.0)	
Ku	6 (15.0)	6 (24.0)	0 (0.0)	0 (0.0)	
Ro52/Ro60	4 (10.0)	2 (8.0)	2 (20.0)	0 (0.0)	
U1 RNP	6 (15.0)	1 (4.0)	0 (0.0)	5 (100.0)	
ANA positivity, n (%)	24 (66.7)	12 (57.1)	7 (70.0)	5 (100.0)	0.182
NVC pattern, n (%)					0.191
Normal pattern	4 (10.0)	4 (16.0)	0 (0.0)	0 (0.0)	
Nonspecific changes	23 (57.5)	12 (48.0)	9 (90.0)	2 (40.0)	
Early scleroderma pattern	9 (22.5)	5 (20.0)	1 (10.0)	3 (60.0)	
Late scleroderma pattern	2 (5.0)	2 (8.0)	0 (0.0)	0 (0.0)	
''Scleroderma-like'' pattern	2 (5.0)	2 (8.0)	0 (0.0)	0 (0.0)	
NVC quantitative assessment, n (%)					
Capillary density					**0.044**
>7 capillaries/mm	29 (74.4)	17 (68.0)	9 (100.0)	3 (60.0)	
4-6 capillaries/mm	4 (10.3)	2 (8.0)	0 (0.0)	2 (40.0)	
<3 capillaries/mm	6 (15.4)	6 (24.0)	0 (0.0	0 (0.0)	
Capillary morphology					0.497
Normal	13 (33.3)	10 (40.0)	2 (22.2)	1 (20.0)	
Abnormal	26 (66.7)	15 (60.0)	7 (77.8)	4 (80.0)	
Capillary dimension					0.177
Normal (<20 μm)	6 (15.4)	6 (24.0)	0 (0.0)	0 (0.0)	
Dilated (20-50 μm)	21 (53.8)	12 (48.0)	7 (77.8)	2 (40.0)	
Giants (>50 μm)	12 (30.8)	7 (28.0)	2 (22.2)	3 (60.0)	
Microhaemorrhages	25 (62.5)	16 (64.0)	6 (60.0)	3 (60.0)	0.969

GERD: gastrointestinal esophageal reflux disease; MSA: myositis-specific autoantibodies; MAA: myositis-associated autoantibodies; ASSD: anti-synthetase syndrome; ANA: antinuclear antibodies; NVC: nailfold videocapillaroscopy; CK: creatine kinase; PL-7: anti-threonyl-tRNA synthetase; PL-12: alanyl-tRNA synthetase; EJ: glycyl-tRNA synthetase; Jo-1: histidyl-tRNA synthetase; Mi-2: nucleosome remodeling-deacetylase; MDA-5: melanoma differentiation-associated gene 5; TIF-1y: Antitranscriptional intermediary factor 1γ; PM: polymyositis; SAE: small ubiquitin-like modifier-1 activating enzyme; U1 RNP: U1 ribonucleoprotein.

ing: DM, antisynthetase syndrome (ASSD), and mixed connective tissue disease (MCTD). The NVC images were systematically assessed and analyzed using standardized definitions provided by the EULAR Study Group on Microcirculation in Rheumatic Diseases, incorporating semi-quantitative and qualitative evaluations. Demographic, clinical and immunologic features were collected and analyzed. Categorical variables are presented as frequencies, while continuous variables are reported as mean ± standard deviation or median [interquartile range (IQR)]. Differences between groups were assessed with chi-square, t-test, Mann-Whitney U test, ANOVA or Kruskal-Wallis test, as appropriate.

Results: This study included 40 patients (72.5% females) with a mean age at diagnosis of 47.4±17.2 years and a median disease duration of 4.0 (IQR 4.0) years. DM was the most prevalent diagnosis (n=25, 62.5%), followed by ASS (n=10, 25%), and MCTD (n=5, 12.5%). Typical skin involvement (including rash, photosensitive erythema, Gottron's, heliotrope rash, periungueal erythema) was present in 30 (75.0%) patients and Raynaud's phenomenon in 24 (57.5%). NVC abnormalities were observed in 90.0% (N=36) of the patients, with no statistically significant differences among the diagnostic subgroups (p=0.191). Nonspecific abnormalities were the most common finding in both DM (48%) and ASSD (90%) patients, while the early scleroderma pattern was the most frequent in DMTC (60%) patients (p=0.191). Very low capillary density (<3/mm) was only observed in patients with DM (24.0% vs 0.0%, p=0.044). The presence of morphologic abnormalities was associated with a shorter disease duration [2.0 (IQR 4.0) vs 5.0 (IQR 2.0), p=0.040]. Regarding specific clinical manifestations, very low capillary density was more frequent in patients with calcinosis (100% vs 10.8%, p=0.030). Furthermore, no other associations were found between NVC features and clinical or immunological subsets.

Conclusion: NVC abnormalities are highly prevalent in patients with DM, ASSD and DMTC, although a selection bias has to be considered. In our cohort, there were no significant differences in the qualitative pattern among different IMM subtypes. Regarding semi-quantitative changes, very low capillary density was associated with both DM and the presence of calcinosis. A larger prospective and longitudinal study has to be conducted to better assess the associations between NVC abnormalities and IIM features as well as their change over time.

092 - NEUTROPHILE TO LYMPHOCYTE AND PLATELET TO LYMPHOCYTE RATIOS PREDICT CLINICAL RESPONSE TO BDMARD IN NAÏVE SPONDYLOARTHRITIS PATIENTS

Frederico Rajão Martins[1], Miguel Bernardes[2, 3], Graça Sequeira[1], Lúcia Costa[2], Pedro David Carvalho[4, 5]
[1]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, [2]Rheumatology Department, Centro Hospitalar Universitário de São João, Porto, Portugal, [3]Department of Medicine, Faculty of Medicine, University of Porto, Porto, Portugal, [4]Hospital de Gambelas, Hospital Particular do Algarve, Faro, Portugal, [5]Centro Académico de Medicina de Lisboa, Portugal, Lisboa, Portugal

Introduction: Spondyloarthritis (SpA) constitute a group of heterogeneous diseases characterized by inflammatory symptoms involving the axial and/or peripheral joints. COVID19 challenged monitoring of disease activity in a significant proportion of patients, stressing the need for objective markers of disease activity. Neutrophil to lymphocyte (NLR) and platelet to lymphocyte (PLR) ratios have been previously associated with active rheumatoid arthritis and lower therapeutic response. Studies in SpA are lacking.

We aim to study associations between these ratios and clinical and sociodemographic variables, and secondarily to assess NLR and PLR predictive value in bDMARD response.

Methods: A bicentric prospective open cohort study was conducted. Patients classified as having SpA under bDMARD registered in Reuma.pt/Spondyloarthritis until December 2021 were included in the analysis. Sociodemographic, clinical and laboratorial data were assessed on bDMARD initiation (t0), 6 months (t6), 12 months (t12), 18 months (t18) and 24 months (t24). Associations between NLR and PLR, and clinical and sociodemographic variables were assessed through generalized estimation equations (GEE) with linear, gamma-link and logistic binary models as needed. Predictive models for response to treatment were constructed using logistic regression (LR). Results for GEE analysis are presented as B value (95% confidence interval), results for LR are displayed as odds ratio [(OR), 95% confidence interval]. A p-value under 0.05 was considered significant. Sensitivity analysis were conducted.

Results: A total of 170 patients were included in this analysis. Most were male (54.7%), non-smokers (68.9%), non-drinkers (81.6%), with an average body mass index of 27.1 (±10.9) kg/m2. There was a predominantly axial phenotype (n=144; 84.7%), with a mean age at bDMARD introduction of 41.8 (±12.2) years, after an average delay until bDMARD treatment of 15.9 (±11.3) years. Adalimumab was the most frequently administered bDMARD (40.6%). Regarding the disease activity models constructed, there was a significant association between NLR [B=1.55 (1.38; 1.74)] and PLR [(B=1.16 (1.09; 1.24)] with ASDAS-CRP (p<0.001), as well for ASDAS-ESR [B=1.48 (1.33; 1.64); B=1.14

TO 092 – TABLE: Predictive value of N/L and P/L ratios in clinically important improvement (Δ ASDAS ≥ 1.1)

	T6*		T12*		T18*		T24*	
	ASDAS-CRP	AS-DAS-ESR	AS-DAS-CRP	AS-DAS-ESR	AS-DAS-CRP	AS-DAS-ESR	ASDAS-CRP	ASDAS-ESR
N/L	2.20 (1.21; 4.00), 0.01	2.77 (1.43; 5.36), <0.01	2.62 (1.02; 6.73), <0.05		2.44 (1.59; 3.76), <0.001			
P/L	1.02 (1.01; 1.04), <0.01	1.03 (1.01; 1.05), <0.001	1.02 (1.00; 1.03), 0.02	1.02 (1.00;1.04), <0.05	1.02 (1.01; 1.03), <0.001	1.01 (1.01; 1.02), <0.01	1.01 (1.00; 1.02), <0.01	1.01 (1.00; 1.02), 0.01
Baseline Covariates								
	T12						T18	
	ASDAS-CRP					ASDAS-ESR	ASDAS-CRP	
	N/L		P/L			P/L	N/L	
Age at bDMARD	0.87 (0.78; 0.98), 0.02		0.91 (0.85; 0.98), 0.02					
Years until bDMARD	1.06 (0.96; 1.17), 0.24		1.03 (0.96; 1.10), 0.45					
BASMI						1.04 (0.60; 1.82), 0.88	1.14 (0.76; 1.071), 0.53	
BASFI	1.07 (0.74; 1.56), 0.71					1.06 (0.73; 1.53), 0.76	0.98 (0.70; 1.37), 0.91	
MASES						0.96 (0.65; 1.44), 0.85	1.23 (0.99; 1.02), 0.10	
SPARCC	1.83 (1.05; 3.19), 0.03							
Physician VAS						1.08 (1.04; 1.13), <0.001	1.04 (1.01; 1.06), <0.01	
Nocturnal pain VAS	1.05 (1.01; 1.08), <0.01		1.04 (1.02;1.07), <0.001				1.00 (0.98; 1.03), 0.84	

*Results shown as odds-ratio (95% confidence interval), p-value; Red filling corresponds to absence of significance in univariate analysis. Grey filling corresponds to absence of inclusion in a multivariate model. In the absence of description of N/L and P/L covariates for a certain model and time set, ratio values reflect univariate results. Covariate section only shows variables included in multivariate analysis in which ratio significance was observed. Abbreviatures: ASDAS: Ankylosing Spondylitis Disease Activity Score; BASMI: Bath Ankylosing Spondylitis Metrology Index; BASFI: Bath Ankylosing Spondylitis Function Index; bDMARD: biotechnological disease modifying antirheumatic drug; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; MASES: Maastricht Ankylosing Spondylitis Enthesitis Score; N/L: neutrophile to lymphocyte ratio; P/L: platelet to lymphocyte ratio; SPARCC: Spondyloarthritis Research Consortium of Canada Enthesitis Index; VAS: visual analogue scale.

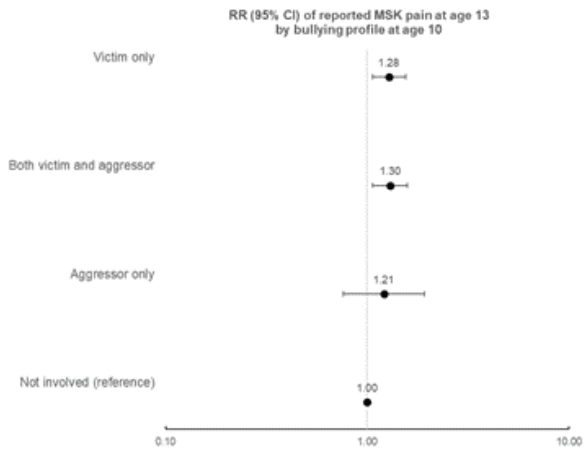
(1.08; 1.21); respectively, both p<0.001]. Predictive analysis revealed a negative predictive value of PLR for ASDAS-ESR [B=-0.23 (-0.50, -0.02); p=0.03] and AS-DAS-CRP [B=-0.35 (-0.52, -0.09); p<0.01] at 6 months of treatment. Regarding clinically important improvement (Δ ASDAS ≥ 1.1; Table), both baseline ratios were predictive of therapeutic response at 6 months, with results for ASDAS-CRP models as follows [NLR OR =2.20 (1.21, 4.00), p=0.01; PLR OR = 1.02 (1.01, 1.04), p<0.01)], and PLR was a significant predictor in all time sets evaluated [12M OR=1.02 (1.00, 1.03), p=0.02; 18M OR=1.02 (1.01, 1.03), p<0.001; 24M OR=1.01 (1.01, 1.02), p<0.01]. Multivariate models were possible at different time sets (Table), but significant results were only seen at 12 months for age at bDMARD introduction (p=0.02); SPARCC (p=0.03); nocturnal pain VAS (p<0.01 ; p<0.001); physician VAS (p<0.001) and at 18 months of treatment for physician VAS (p <0.01).

Conclusion: NLR and PLR seem to be significant predictors of therapeutic response to bDMARD in naïve SpA patients. Such easily available serum parameters could prove useful on the assessment of disease activity in patients with low consultation adherence or living far from hospital centres.

093 - CHILDHOOD BULLYING AND MUSCULOSKELETAL PAIN IN ADOLESCENCE: A PROSPECTIVE STUDY OF REPORTED PAIN HISTORY AND QUANTITATIVE SENSORY TESTING

Raquel Lucas[1], Sara Soares[1], Sílvia Fraga[1], Makram Talih[1]
[1]Departamento de Epidemiologia, ISPUP, Porto, Portugal

Psychosocial circumstances are strongly associated with the onset and prognosis of chronic musculoskeletal (MSK) pain. However, it is unknown they precede, coexist with or follow MSK pain conditions. Bullying during youth is a particularly damaging but modifiable social experience that is known to interfere with



TO 093 – Figure 1. Relative risk of musculoskeletal pain at age 13 by bullying profile at age 10

health status, potentially including pain memories and responses to noxious stimuli.

We aimed to quantify the longitudinal associations between bullying profiles and musculoskeletal pain (reported history and pressure pain sensitivity) in a large population-based cohort of adolescents followed since birth.

We used data from the 10 and 13 years waves of the Generation XXI birth cohort study based in Porto, Portugal.

Data on youth pain history were collected during face to face interviews from parents (age 10) and adolescents (age 13) using the Luebeck pain screening questionnaire. MSK pain was considered present when the participant had recurrent pain in one of the following sites: back, neck, shoulders, upper/lower limbs, hips, or generalized MSK pain. A subsample of the cohort underwent quantitative sensory testing using computerized deep tissue cuff pressure algometry in the legs, which allowed estimating pain detection/tolerance thresholds, and measuring pain intensity ratings. Bullying profiles were computed for the 10 and 13 years waves based on responses to the Bully Scale Survey and participants were classified as "victim only", "both victim and aggressor", "aggressor only", or "not involved". Cross-sectional and longitudinal associations between bullying profiles and reported pain were quantified using relative risks (RR) and 95% confidence intervals (95% CI) obtained using Poisson regression. Associations between bullying and pain sensitivity were estimated using linear regression coefficients and 95% CI. Estimates were adjusted for sex and number of Adverse Childhood Experiences adapted from the questionnaire by Felitti et al (1998).

Among the 4049 adolescents examined, bullying profiles at age 10 were associated with MSK pain at age 13, whereas there were no associations between MSK pain at age 10 and bullying profiles at 13. Cross-sectional associations between bullying and MSK pain history were present at 10 and 13 years. When compared to adolescents "not involved" in bullying, those who reported being victims at age 10 had higher risk of reporting MSK pain at age 13: RR (95% CI) 1.28 (1.06, 1.55) for "victims only" and 1.30 (1.06, 1.58) among "both victims and aggressors" (Figure below). "Aggressors only" did not show clear differences in terms of pain history. Among the 1727 adolescents who underwent cuff pressure algometry, those who were "victims only" at age 10 had lower average pain detection and tolerance thresholds at 13 years [linear regression coefficients (95% CI): -1.81 (-3.29, -0.33) for detection and -2.73 (-5.17, -0.29) for tolerance], as well as higher pain intensity ratings [0.37 (0.07, 0.68) and 0.39 (0.06, 0.72)], when compared with adolescents not involved in bullying. No differences were seen for the remaining bullying profiles.

Our study provides prospective evidence that bullying victimization is more likely to lead to negative reported MSK pain experiences than the reverse. Bullying may have long-term influence on the risk of chronic musculoskeletal pain and may interfere with somatosensory responses to painful stimuli.

094 - COMPARATIVE EFFECTIVENESS OF COMBINED RHEUMATOID ARTHRITIS THERAPIES ON PHYSICAL FUNCTION AND BIOLOGIC DRUG SURVIVAL IN PATIENTS STARTING THEIR FIRST BDMARD: DATA FROM THE REUMA.PT REGISTRY

João Lagoas Gomes[1], Rita Cravo[2], Sophee Blanthornhazell[3], Marília Antunes[2], Miguel Bernardes[4], Maria Seabra Rato[4], Augusto Silva[5, 6], Ana Margarida Duarte-Monteiro[5, 6], Cátia Duarte[7], Jorge Pestana Lopes[8], Maria Manuela Costa[9], José Tavares-Costa[10], João Madruga Dias[11], Couto M[12], P Pinto[13], Gisela Eugénio[14], Paula Valente[15], Teresa Novoa[16], Maria José Santos[6, 8], Rodrigues AM[17, 18, 19]

[1]Rheumatology Unit, Centro Hospitalar do Tâmega e Sousa, Penafiel, Portugal, [2]., Lisboa, Portugal, [3]Medical, AbbVie, Portugal, Lisboa, Portugal, [4]Serviço de Reumatologia, Centro Hospitalar de São João, Porto, Portugal, [5]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Hospital de Santa Maria, CHLN, Lisboa, Portugal, [6]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [7]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [8]Rheumatology Department, Hospital Garcia de

Orta, Almada, Portugal, [9]Serviço de Reumatologia, Centro Hospitalar Lisboa Ocidental | Hospital Egas Moniz, Lisboa, Portugal, [10]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [11]Serviço de Reumatologia, Centro Hospitalar Médio Tejo, Torres Novas, Portugal, [12]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [13]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [14]Rheumatology Department, Centro Hospitalar Entre Douro e Vouga, Aveiro, Portugal, [15]Rheumatology, Centro Hospitalar Entre o Douro e Vouga, Santa Maria da Feira, Portugal, [16]Serviço de Reumatologia, Hospital do Divino Espírito Santo, Ponta Delgada, Portugal, [17]EpiDoC Unit, CEDOC, NOVA Medical School, NOVA University, Lisboa, Portugal, [18]Rheumatology, Hospital Lusíadas, Lisboa, Portugal, [19]Reuma.pt, Sociedade Portuguesa de Reumatologia, Lisboa, Portugal

Introduction: Combining Rheumatoid Arthritis treatments is a widely used therapeutic strategy. However, there is uncertainty surrounding the most effective regimen in terms of the number and type of treatments. The aim of the present study is to evaluate if the number of concomitant RA-related therapies is associated with differences in physical function, pain VAS (PVAS) and patient global assessment (PtGA) variation in RA patients starting their first biologic DMARD (bDMARD). We also aim to determine the impact of the number of concomitant RA-related therapies on bDMARD survival.

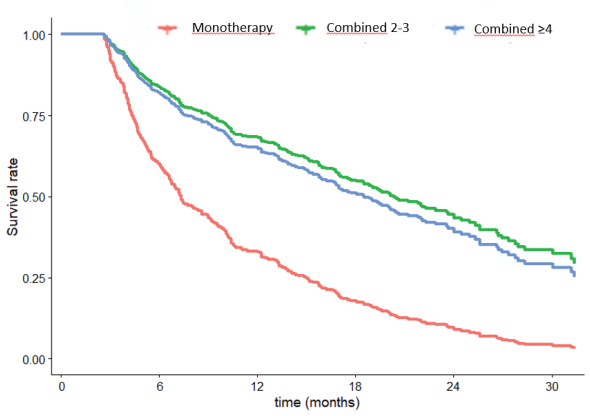
Methods: This multicentric, prospective observational cohort study included adult RA patients registered in Reuma.pt, who were divided into 3 groups according to the number of RA therapies, which included bDMARDS, csDAMRDs, corticosteroids (GCs), non-steroidal anti-inflammatory drugs (NSAIDs) and other analgesics: bDMARD monotherapy; group 2-3 concomitant RA therapies and group ≥4 concomitant RA therapies. At baseline, which was the date of 1st line bDMARDs initiation, several demographic and clinical characteristics were described.

The primary outcome was change from baseline in HAQ score at 12 months of follow-up. An ANCOVA model was created to adjust for potential confounders, with HAQ variation as the dependent variable, the concomitant RA therapy group as a fixed factor and HAQ at baseline, age, gender, disease duration, age at diagnosis and number of comorbidities as covariates. Similar models were created for HAQ at 6, 24 and 36 months and for PVAS and PtGA.

Results: Overall, 659 patients were included, 27 in the bDMARD monotherapy group, 207 in group 2-3 and 425 in group ≥4. At baseline (table 1), monotherapy group presented significantly fewer extra-articular manifestations (7% vs 28% and 16%) and lower baseline mean HAQ (1vs1.4). The most frequently prescribed bDMARD was etanercept in all groups [monotherapy: n=15 (55.6%); group2-3: n=79 (38.1%); group ≥4: n=184 (43.3%)]. The most common therapeutic combinations in group 2-3, in addition to bDMARD, were: methotrexate (MTX) + GCs (N=95) and MTX (N=69). In group ≥4, the most common combinations were: MTX + GCs + NSAIDs (N=126) and leflunomide + glucocorticoids + NSAIDs (n=53).

Mean HAQ at 12 months decreased in all three therapeutic groups, but this reduction was significantly lower in the monotherapy group (0.26±0.69) compared with the other 2 groups (0.4±0.62 and 0.46±0.59). No differences were found between 2-3 and ≥4 concomitant therapies. Regarding other time points, the difference in HAQ variation was similar to T12, but when adjusting for confounders this difference was not statistically significant. Mean PtGA decrease was significantly lower in monotherapy patients compared with the other 2 groups at 6 months (16.1±11.3 vs 26.7±19.5 and 25.03±18.4) and 36 months (17.1±12.3 vs 24.9±17.1 and 27.6±19.3). Also mean pain VAS (PVAS) decrease was lower in the monotherapy group at 6 months (10.1±6.3 vs 25.7±19.1 and 25.2±18.7) and 36 months (19±9.3 vs 24.3±18.1 and 25.5±19.7), after adjusting for potential confounders. The risk of bDMARD discontinuation was higher in monotherapy group when compared with the other two groups (fig. 1).

Conclusion: This study showed that RA patients starting their first bDMARD in combination with other RA-related therapies had significantly greater HAQ reduction at 12 months and lower discontinuation rates than on monotherapy. No differences were found between combined treatment regimens (2-3 vs ≥4).



TO 094 – Figure 1. Cox regression for bDMARD discontinuation, adjusting for the number of concomitant RA therapies, age, sex, age at diagnosis, disease duration, smoking status, disease activity and number of comorbidities.

TO 094 – TABLE 1. Baseline demographic characteristics and clinical variable of the study population, stratified by the number of concomitant RA therapies

	bDMARD monotherapy N=27	2-3 RA concomitant therapies N=207	≥ 4 RA concomitant therapies N=425
Age* (in years; mean ± SD)	53.2 ± 14.2	55.2 ± 11.3	53.6 ± 12.4
Female gender (N; %)	23 (85%)	176 (85%)	365 (86%)
Age at diagnosis, in years (mean ± SD) (N=622)	42.1 ± 17.6	45.9 ± 13.2	45 ± 12.4
Smoking status (N=563) Current Ex Never	2 (12%) 4 (24%) 11 (65%)	20 (12%) 25 (14%) 129 (73%)	49 (13%) 50 (13%) 273 (73%)
Alcohol (N; %) (N=532) Current Ex Never	1 (6%) 1 (6%) 16 (88%)	9 (6%) 11 (7%) 144 (87%)	30 (9%) 11 (3%) 309 (88%)
BMI (N=233) (mean ± SD)	23.7 ± 4.2	27.2 ± 5.1	26.1 ± 4.4
Working status (N= 422) (N; %) Full-time Part-time Retired Sick leave > 1 month Unemployed	7 (58%) 0 4 (33%) 0 1 (8%)	46 (37%) 4 (3%) 52 (42%) 10 (8%) 11 (9%)	114 (40%) 13 (5%) 118 (41%) 21 (7%) 21 (7%)
Number of comorbidities (N;%) 0 1 2 ≥3	23 (85%) 3 (11%) 1 (4%) 0	131 (63%) 57 (27%) 14 (7%) 5 (2%)	264 (62%) 99 (23%) 49 (3%) 13 (1%)
RF positive (N= 593) (N; %)	16 (89%)	152 (81%)	295 (76%)
ACPA positive (N=489) (N; %)	16 (94%)	126 (77%)	228 (74%)
Erosive disease (N=460) (N; %)	10 (63%)	112 (75%)	220 (75%)
Presence of extra-articular manifestations (N=566) (N; %)	2 (1%)	47 (28%)	62 (16%)
Type of bDMARD therapy (N;%): TNF inhibitors Etanercept Adalimumab Golimumab Infliximab Certolizumab Tocilizumab Rituximab Anakinra	21 (78%) 15 1 0 1 4 5 (19%) 1 (4%) 0	154 (74%) 79 30 28 11 6 38 (18%) 14 (7%) 1 (0.5%)	368 (86%) 184 81 61 35 7 32(8%) 25 (6%) 0
Concomitant csDMARDs (N; %)*	0	162 (78.3%)	424 (99.8%)
Concomitant Methotrexate (N; %)	0	126 (60.9%)	347 (81.7%)
Concomitant Glucocorticoids (N;%)	0	115 (55.6%)	399 (93.9%)
Concomitant NSAIDs (N;%)	0	54 (26%)	340 (80%)
DAS 28 ESR (N=641) (mean ± SD)	5.2 ± 1.5	5.2 ± 1.5	5.4 ± 1.3
Patient global assessment (N= 653) (0-100) (mean ± SD)	54 ± 23.2	59.4 ± 25.9	61.2 ± 24.3
Pain VAS (N= 614) (0-100) (mean ± SD)	50.2 ± 25.2	57.3 ± 26.7	60.8 ± 24.2
Physician global assessment (N= 593) (0-100) (mean ± SD)	46.5 ± 21.5	50.5 ± 23.3	52.5 ± 19.2
HAQ (0-3) (mean ± SD)	1 ± 0.7	1.4 ± 0.7	1.4 ± 0.6
EuroQol 5D (N=181) (0-1) (mean ± SD)	0.4 ± 0.2	0.4 ± 0.2	0.3 ± 0.3

*Age at start of bDMARD therapy; ** Included csDMARDs: Methotrexate, Leflunomide, Sulfasalazine, Hydroxychloroquine, Cyclosporine and Azathioprine; ACPA-anti-citrullinated protein antibodies; BMI- body mass index; ESR- erythrocyte sedimentation rate; EQ5D- European; HAQ- health assessment questionnaire; RA-rheumatoid arthritis; RF- rheumatoid factor; SD-standard deviation; TNF- tumor necrosis factor; VAS- visual analogue scale.

095 - CAN AXIAL SPONDYLOARTHRITIS UNEQUIVOCALLY BE DIAGNOSED BY RHEUMATOLOGISTS IN PATIENTS WITH CHRONIC BACK PAIN OF LESS THAN TWO YEARS DURATION? THE PRIMARY OUTCOME OF THE TWO-YEAR SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

Mary Lucy Marques[1, 2], Sofia Ramiro[2, 3], Miranda van Lunteren[2], Rosalinde Stal[2], Robert Landewé[3, 4], MGH van de Sande[4], Inger Jorid Berg[5], Karen Minde Fagerli[5], Maikel van Oosterhout[6], Sofia Exarchou[7], Roberta Ramonda[8], Désirée van der Heijde[2], F vanGaalen[2]

[1]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [3]Rheumatology, Zuyderland Medical Center, Heerlen, Netherlands, [4]Clinical Immunology & Rheumatology, Academic Medical Center, University of Amsterdam, Amsterdam, Netherlands, [5]Department of Rheumatology, Diakonhjemmet Hospital, Oslo, Norway, [6]Rheumatology, Groene Hart Ziekenhuis, Gouda, Netherlands, [7]Clinical Sciences, Section of Rheumatology, Lund University, Malmo, Sweden, [8]Division of Rheumatology, University of Padova, Padova, Italy

Background: Unacceptable diagnostic delay in axial Spondyloarthritis (axSpA) remains an issue. In 2008, the longitudinal SPondyloArthritis Caught Early (SPACE)-cohort started to assess the prevalence of axSpA and the reliability of an early diagnosis in patients with chronic back (CBP) of unknown origin. Here we present the primary outcomes of SPACE.

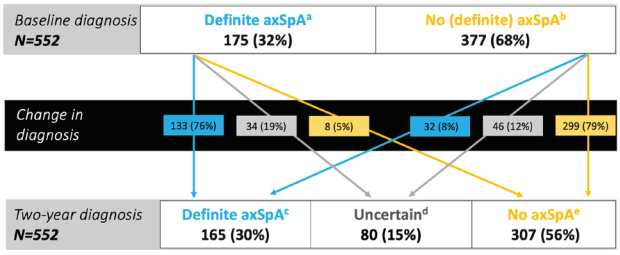
Objectives: To assess the two-year (2y) prevalence of an axSpA diagnosis in patients with CBP referred to the rheumatologist; The sustainability of a baseline (BL) diagnosis of axSpA when reviewed after 2y; And to explore BL patient-differences of those with and without an axSpA diagnosis at 2y.

Methods: We analysed the 2y data from SPACE, a European inception cohort of patients (age <45y) with CBP of recent onset (≥3 months, ≤2y) and unknown origin. The full diagnostic work-up included clinical SpA features, acute phase reactants, HLA-B27, radiographs and MRI of the sacroiliac joints (SI-CR and SI-MRI) and spine (data not shown). Patients with an increased likelihood of having axSpA (≥1 major or ≥2 minor prespecified SpA features) were eligible for follow-up. The clinical diagnosis at 2y was the main outcome of this study. At each visit, the treating rheumatologist judged the presence or absence of axSpA (axSpA or no-axSpA) with a level of confidence (LoC) on a numeric rating scale (0: not confident at all to 10: very confident). The main outcome was the presence of 'definite axSpA' at 2y, defined by a clinical diagnosis of axSpA with LoC ≥7 at 2y. 'No axSpA' was defined as not having axSpA at 2y (LoC ≥7; or if LoC <7, plus an alternative diagnosis for CBP reported by the rheumatologist). All other patients were considered to have an 'uncertain' diagnosis (Figure 1). If the 2y visit was missing, the last observation carried forward

TO 095 – TABLE. Baseline characteristics by 2y diagnosis of patients with chronic back pain duration of ≥3 months but ≤2y, < 45y of age

	Definite axSpA **N=165**	*Uncertain* **N=80**	*No axSpA* **N=307**
Age at inclusion, years	29.8 (7.6)	30.9 (8.7)	31.0 (8.4)
Male	52%	38%	26%
Symptom duration, months	12.8 (7.0)	13.4 (7.2)	13.4 (7.2)
HLA-B27 +	81%	60%	15%
Family history of SpA	48%	61%	39%
Inflammatory back pain§	76%	63%	55%
Good response to NSAIDs	43%	40%	24%
Peripheral arthritis#	16%	16%	7%
Dactylitis#	7%	9%	3%
Heel pain#	19%	15%	8%
Anterior uveitis#	13%	13%	3%
Inflammatory bowel disease#	7%	6%	7%
Psoriasis#	11%	9%	8%
Increased acute phase reactants§	37%	20%	19%
Sacroiliitis on radiographs§	23%	9%	0%
Sacroiliitis on MRI§	67%	26%	6%

§ local data; # currently present or past presence (if confirmed/reported by a physician); axSpA: axial Spondyloarthritis; MRI: magnetic resonance imaging.



[a] patients diagnosed at BL with axSpA with a Level of Confidence (LoC) ≥7; [b] patients diagnosed at BL with axSpA (LoC <7) or with no axSpA (any LoC); [c] patients diagnosed with axSpA with a LoC ≥7 at 2y (if complete follow-up) or at the last two available visits (if missing the 2y visit); [d] patients diagnosed with axSpA with a LoC <7 at 2y (or LoC ≥7, if only baseline observation available), and patients with axSpA with LoC <7 at the last available observation and no consistent diagnosis at the last two observations nor alternative no axSpA diagnosis given at the last observation; [e] patients with no axSpA at the last observation over 2y with LoC ≥7 (or if <7, plus an alternative no axSpA diagnosis reported, e.g. fibromyalgia or aspecific back pain). **axSpA:** axial Spondyloarthritis.

TO 095 – Figure 1. DIagnosis course from baseline (BL) to the two-year {2y} anchor visit.

approach was used (details provided in Figure 1). ASAS classification criteria were computed using sacroiliitis local reading in definite axSpA patients only. We assessed the prevalence of definite axSpA at 2y as well as changes in diagnosis over time, and descriptively summarised BL characteristics by 2y diagnostic groups.

Results: We included 552 CBP patients (Leiden n=383, Oslo n=94, Amsterdam n=48, and Gouda n=27). A diagnosis of definite axSpA was given to 175 (32%) patients at BL and 165 (30%) at 2y (Figure 1). The mean (SD) LoC were 8.1 (2.0) and 8.7 (1.0), with 155/175 (89%) and 145/165 (87%) fulfilling ASAS classification criteria, respectively. BL diagnostic judgments were relatively unequivocal and remained rather stable: At 2y, 5% of the BL diagnoses of definite axSpA were refuted; and -vice versa: 8% of those who did not obtain a BL diagnosis of axSpA 'gained' one at 2y. Diagnostic uncertainty remained in 15% of CBP-patients. Expectedly, BL SpA features were more prevalent in the 2y definite axSpA group (Table 1). HLA-B27 status and (presence or absence of) imaging-detected sacroiliitis at BL appeared the best discriminators between definite axSpA and no axSpA at 2y.

Conclusion: One-third of patients with CBP of recent onset referred to the rheumatologist has definite axSpA. Most patients can be unequivocally and reliably diagnosed at their first assessment, though residual diagnostic uncertainty persisted after 2y. None of the many SpA features suffices alone, but HLA-B27 positivity and sacroiliitis on imaging discriminate best 2y diagnostic groups.

096 - THE YIELD OF REPEATED ASSESSMENTS IN CHRONIC BACK PAIN PATIENTS SUSPECTED OF EARLY AXIAL SPONDYLOARTHRITIS: TWO-YEAR DATA FROM THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

Mary Lucy Marques[1, 2], Sofia Ramiro[2, 3], Miranda van Lunteren[2], Rosalinde Stal[2], Robert Landewé[3, 4], MGH van de Sande[4], Karen Minde Fagerli[5], Inger Jorid Berg[5], Maikel van Oosterhout[6], Sofia Exarchou[7], Roberta Ramonda[8], Désirée van der Heijde[2], F vanGaalen[2]

[1]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [3]Rheumatology Department, Zuyderland Medical Center, Heerlen, Netherlands, [4]Clinical Immunology & Rheumatology, Academic Medical Center, University of Amsterdam, Amsterdam, Netherlands, [5]Department of Rheumatology, Diakonhjemmet Hospital, Oslo, Norway, [6]Rheumatology, Groene Hart Ziekenhuis, Gouda, Netherlands, [7]Clinical Sciences, Section of Rheumatology, Lund University, Malmo, Sweden, [8]Division of Rheumatology, University of Padova, Padova, Italy

Background: We have shown in the SPACE cohort that a diagnosis of early axial spondyloarthritis (axSpA) can be made in patients with chronic back pain (CBP) of less than two years (2y). However, diagnostic uncertainty can be an obstacle to initiating appropriate treatment. The value of repeated assessments of SpA features for a definite diagnosis is yet to be determined.

Objectives: To assess the yield of repeated assessments of SpA features over 2y to make a definite axSpA diagnosis in patients with recent onset CBP referred to the rheumatologist, and to describe the characteristics of patients who change to definite axSpA over time.

Methods: We used the 2y data from the SPACE cohort, a European multicentre inception cohort of patients (age <45y) with CBP of recent onset (≥3 months, ≤2y) included from 2008 to 2016. The diagnostic work-up consisted of patient history, physical exam, acute phase reactants (APR) and HLA-B27 testing, radiographs, and MRI of the sacroiliac joints (SI-CR and SI-MRI) and of the spine (not shown). In patients with ≥1 major or ≥2 minor prespecified SpA features, clinical assessments, APR, and imaging were repeated at 3 months, 1y and 2y visits. At each visit, the rheumatologist reported a clinical diagnosis of axSpA or no axSpA together with the level of confidence (LoC; numeric rating scale from 0 (not confident at all) to 10 (very confident)). Herein, we categorized patients by diagnosis likelihood. At baseline (BL), two categories were defined: 'Definite axSpA/no axSpA' when the diagnosis was given with LoC ≥7 and 'Uncertain axSpA/no axSpA' if LoC <7. At 2y, the following categories were defined: 'definite, most likely and possible axSpA' and 'possible, most likely and definite no axSpA' (definitions in Figure 1). The ASAS classification criteria were applied using local reading. We explored the diagnostic course over 2y. In patients with a new diagnosis of definite axSpA at 2y, SpA fea-

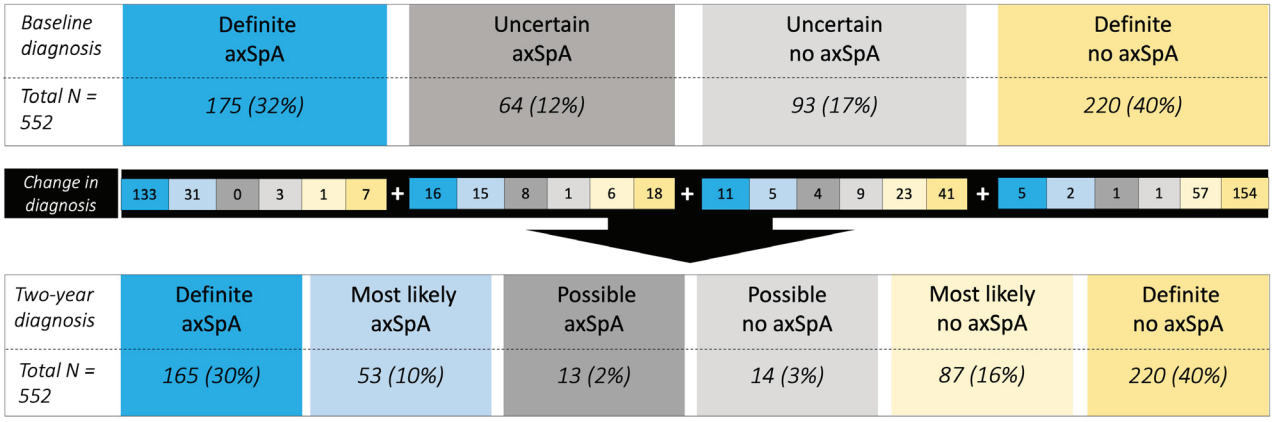
tures were investigated over time.

Results: We included 552 patients (Leiden n=383, Oslo n=94, Amsterdam n=48, and Gouda n=27). Definite axSpA was attributed to 175 (32%) patients at BL and 165 (30%) at 2y (Figure 1), 155/175 (89%) and 145/165 (87%) fulfilled ASAS classification criteria, respectively. Of the 175 patients with definite axSpA at BL, 133 (76%) retained the diagnosis, and only 11 (6%) changed to no axSpA at 2y. Although still considered as axSpA by the rheumatologist, 31/175 (17%) definite axSpA patients at baseline were no longer definite axSpA at 2y, due to a decrease in LoC <7 (n=14/31) or incomplete follow-up (n=17/31). Overall, the diagnosis changed to definite axSpA over 2y in 32 patients

TO 096 – TABLE. Characteristics of 32 patients changing to definite axSpA with newly developed SpA features over 2 years

Baseline diagnosis	Uncertain axSpA at BL N=16		Uncertain no axSpA at BL N=12		Definite no axSpA at BL N=5	
	BL	2Y#	BL	2Y#	BL	2Y#
Age at inclusion, years	30.3 (8.6)	-	35.1 (7.9)	-	25.8 (6.1)	-
Male	50%	-	73%	-	40%	-
Symptom duration, months	12.7 (6.8)	-	12.5 (6.8)	-	12.0 (4.7)	-
HLA-B27 +	81%	-	55%	-	80%	-
Family history of SpA	8	9	3	4	3	4
Inflammatory back pain	14	15	6	7	5	5
Good response to NSAIDs	4	10	5	7	3	4
Peripheral manifestations	4	8	1	3	1	2
Extra-musculoskeletal manifestations	4	6	3	4	0	1
Increased acute phase reactants	4	4	1	3	2	2
Sacroiliitis on radiographs§	0	0	1	2	0	1
Sacroiliitis on MRI§	3	8	3	5	0	1
Total nr of SpA features^	3.4 (1.1)	4.6 (1.4)	2.6 (1.0)	3.7 (1.3)	3.6 (0.5)	4.8 (0.8)
Nr of new SpA-features over follow-up^$	-	1.3 (1.0)	-	1.1 (0.9)	-	1.2 (0.8)
ASAS classification criteria	-	81%	-	58%	-	80%

Data presented as mean (SD), % or n of patients. §local readings; ^including HLA-B27 and imaging; #Cumulative numbers over the two-year follow-up; $24 (75%) patients developed new SpA features over time. BL – baseline; MRI –magnetic resonance imaging.



Diagnosis definitions at baseline - Definite axSpA/no axSpA: 'axSpA' or 'no axSpA' at baseline (LoC ≥7); **Uncertain axSpA/no axSpA:** 'axSpA' or 'no axSpA' at baseline (LoC <7).
Diagnosis definitions at two years (last observation carried forward approach if two-year visit data was missing) - Definite axSpA: 'axSpA' (LoC ≥7) at two years (complete follow-up) or at the two last available visits (missing at the two-year visit); **Most likely axSpA:** 'axSpA' (LOC <7) at two years, plus a consistent diagnosis of 'axSpA' in the two last visits (complete follow-up) or 'axSpA' (LoC ≥7) at the last visit only (missing at the two-year visit); **Possible axSpA:** 'axSpA' (LOC <7) at two years, plus no consistent diagnosis of 'axSpA' in the last two visits (complete follow-up) or 'axSpA' (LoC <7) at the last visit (missing at the two-year visit); **Possible no axSpA:** 'no axSpA' (LOC <7), plus no consistent diagnosis of 'no axSpA' in the last two visits (complete follow-up) or 'no axSpA' (LoC <7) at the last visit and no alternative diagnosis reported (missing at the two-year visit); **Most likely no axSpA:** 'no axSpA' (LOC <7), plus a consistent diagnosis of 'no axSpA' in the last two available visits (complete follow-up) or 'no axSpA' (LoC ≥7) at the last visit only or if LoC <7, plus an alternative diagnosis reported (missing at the two-year visit); **Definite no axSpA:** 'no axSpA' (LoC ≥7) at two years (complete follow-up) or at the two last available visits (missing at the two-year visit). **axSpA** – axial Spondyloarthritis; **LoC** – level of confidence.

TO 096 – Figure 1. Course of diagnosis over two years in patients with recent onset chronic back pain of unknown origin

(BL: 16 uncertain axSpA, 11 uncertain no axSpA, and 5 definite no axSpA); on average, 3 to 4 SpA features were already present at BL and 1 new SpA feature developed over 2y (Table 1), with response to NSAIDs (9/24 patients) and MRI sacroiliitis (8/24 patients) being the most frequently developed over time. Of the 8 patients with new MRI sacroiliitis over time, 7 (88%) were HLA-B27+ and 5 (63%) were male.

Conclusion: The yield of repeated assessments of SpA features in patients with CBP suspected of axSpA was modest for the increase of new definite axSpA diagnosis at 2y. Most SpA features were already present at BL, with sacroiliitis on MRI and response to NSAIDs being the most frequently incident SpA features potentially adding to a definite axSpA diagnosis over time. The usefulness of repeating MRI in terms of diagnostic yield is low but can be considered in HLA-B27+ patients, especially if male.

099 - MANIFESTATIONS AND PREDICTORS OF NEUROLOGIC INVOLVEMENT IN BEHÇET'S DISEASE: RESULTS FROM A MONOCENTRIC STUDY

Margarida Lucas Rocha[1, 2, 3], Roberto Costa[1, 2], Ana Teodósio Chícharo[1, 2, 3], Martins-Martinho J [1, 2], Carla Macieira[1], Cristina Ponte[1, 2], Nikita Khmelinskii[1, 2]

[1]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Rheumatology Research Unit, Instituto de Medicina Molecular João Lobo Antunes, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal, [3]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal

Background/Purpose: Behçet's disease (BD) is a multisystem inflammatory disorder primarily affecting mucocutaneous tissues. Central nervous system (CNS) involvement, Neuro-BD (NBD), is a potentially severe manifestation of BD with a variable prevalence (1-59%) depending on the diagnostic criteria and ethnicity of the patients. NBD is classified according to the type of involvement into parenchymal (brainstem, hemisphere and spinal cord) and non-parenchymal (meningitis, intracranial hypertension and cerebral vascular thrombosis) disease. We aim to characterize BD patients with CNS involvement and to identify predictors of this clinical subtype.

Methods: Single-centre observational retrospective study using data from patients with a diagnosis of BD registered at the Rheumatic Diseases Portuguese Registry (Reuma.pt). NBD was defined according to International Consensus Recommendation Criteria for NBD diagnosis as having neurological and/or psychiatric symptoms with compatible abnormalities in MRI and/or cerebral spinal fluid (CSF) analysis, in the absence of an alternative diagnosis. Data regarding the demographics and clinical manifestations were collected.

Results: We included 160 patients with BD, 42/160 (26%) males, 129/147 (88%) Caucasian with a median [IQR] age at diagnosis of 31.9 [16.5] years and a median follow-up of 11.0 [12] years. Diagnostic delay was lower in non-Caucasian vs Caucasian patients (time between BD symptoms onset and BD diagnosis: 0.84 vs 3.0 years; p=0.045). NBD was diagnosed in 24/160 (15%) patients, in 2/160 (1%) at disease onset. The median age at NBD diagnosis was 31.0 [13] years; 3.8 [5.9] years after BD onset. Fifteen (63%) patients had parenchymal involvement, eight (33%) had non-parenchymal involvement, and one (4%) had mixed involvement. Imaging abnormalities were found in 22/22 (100%) patients and CSF abnormalities in 6/11 (55%) patients. Headache was the most frequent symptom (12/17; 71%) followed by nausea and diplopia (6/17; 35% each), pyramidal symptoms (5/17; 29%) and cerebellar symptoms (4/17; 24%). Most of the patients (19/24; 79%) had a single episode; 4/24 (17%) had a progressive form with relapses and 1/24 (4%) had a relapsing remitting form.

Table 1 shows the difference between patients with and

TO 099 – TABLE. Comparison of demographic and disease characteristics between Benhçet's disease patients with without central nervous system involvement.

	All patients (N= 160)	NBD patients (N=24)	Patients without CNS manifestation (N=136)	*p*-value*
Demographics				
Age at symptom onset[θ], years (median, IQR)	24.6 (19.1)	27.0 (13.6)	24.1 (19.8)	0.220
Age at BD diagnosis[ϕ], years (median, IQR)	31.9 (16.5)	31.0 (12.3)	32.3 (17.7)	0.913
Diagnostic delay[β], years (median, IQR)	3.0 (10.9)	1.8 (9.2)	3.0 (11.9)	0.095
Symptom duration[ϕ], years (median, IQR)	11.0 (12.0)	11.5 (18.0)	11.0 (11.0)	0.979
Male gender (n, %)	42 (26)	6 (25)	36 (26)	0.880
Caucasian ethnicity[β] (n, %)	129 (88)	16 (73)	113 (90)	**0.031**
Comorbidities (n, %)				
Arterial hypertension[θ]	34 (22)	4 (17)	30 (23)	0,497
Current smokers[β]	26 (21)	6 (32)	20 (20)	0.240
Diabetes mellitus[θ]	11 (7)	2 (8)	9 (7)	0.680
Dyslipidemia[θ]	26 (17)	4 (17)	22 (17)	1.000
Manifestations at disease onset (n, %)				
Oral ulcers[θ]	138 (88)	20 (91)	118 (88)	1.000
Genital ulcers[θ]	59 (38)	15 (68)	44 (33)	**0.002**
Ocular manifestations[1θ]	22 (14)	6 (27)	16 (12)	0.090
Cutaneous manifestations[2θ]	39 (25)	6 (27)	33 (25)	0.699
Articular manifestations[3θ]	18 (12)	1 (5)	17 (13)	0.470
Vascular manifestations[4θ]	6 (4)	2 (10)	4 (3)	0.187
Gastrointestinal manifestations[5θ]	1 (1)	0 (0)	1 (1)	1.000
Constitutional symptoms[θ]	11 (7)	0 (0)	11 (8)	0.362
Manifestations at disease onset and during disease follow up (n, %)				
Oral ulcer, ever	156 (96)	23 (96)	131 (96)	1.000
Genital ulcer, ever	130 (80)	19 (79)	109 (80)	1.000
Ocular manifestations[1θ], ever	60 (38)	13 (57)	47 (35)	**0.047**
Cutaneous manifestations[2θ], ever	115 (73)	15 (65)	100 (74)	0.378
Articular manifestations[3θ], ever	71 (45)	11 (46)	60 (44)	0.900
Vascular manifestations[4], ever	24 (15)	5 (21)	19 (14)	0.364
Gastrointestinal manifestations[5], ever	17 (11)	2 (8)	15 (11)	1.000
Constitutional symptoms	23 (14)	7 (29)	16 (12)	0.051
Positive pathergy test∇	32 (35)	8 (62)	24 (30)	0.055
Positive HLA-B51 haplotype∇	36 (45)	6 (50)	30 (44)	0.706
ISG, 1990 Criteria fulfillment	128 (80)	20 (83)	108 (79)	0.787
Treatment (n, %)				
Glucocorticoids[θ]	128 (81)	23 (100)	105 (78)	**0.008**
csDMARDs[θ]	102 (65)	18 (86)	84 (62)	**0.035**
bDMARDs[θ]	22 (14)	6 (29)	16 (12)	0.085
Cyclophosphamide[θ]	12 (8)	10 (48)	2 (1)	**<0.001**
Prognosis (n, %)				
Mortality	6 (4)	4 (17)	2 (1)	**0.005**

θ Missing data <10%; ϕ Missing data 10-20%; β Missing data 20-25%; ∇ Missing data 25-50%.
* Independent samples t-test for continuous variables and Chi2 for categorical variables.
NBD – neuro-Behçet's disease; CNS – central nervous system; ISG – International Study Group; csDMARD: conventional synthetic disease modifying antirheumatic drugs; bDMARD: biologic disease modifying antirheumatic drugs.

[1]Ocular manifestations included anterior and/or posterior uveitis, retinal vasculitis, and central retinal vein or artery occlusion; [2]Cutaneous manifestations included erythema nodosum, pseudofoliculitis, and papulopustular or acneiform lesions; [3]Articular manifestations included inflammatory arthralgia or arthritis; [4]Vascular manifestations included superficial phlebitis, deep vein thrombosis, large vein thrombosis, and arterial thrombosis or aneurysm; [5] Gastrointestinal manifestations included abdominal pain, diarrhea, bowel obstruction, and bowel perforation.

without NBD. Patients with NBD were more frequently non-Caucasian (27% vs 10%, p=0.031), had more genital ulcers at disease onset (68% vs 33%, p=0.002), more ocular manifestations (57% vs 35%, p=0.047) during the disease course, were more frequently treated with systemic glucocorticoids (p=0.008), csDMARDs (p=0.035) and cyclophosphamide (p<0.001) and had a higher mortality rate (17% vs 1%, p=0.005) than patients without NBD. On multivariate analysis, genital ulcers at presentation (OR 3.36, 95%: 1.20-9.43) and constitutional symptoms during the disease course (OR 3.41, 95%: 1.02-11.32) were independent predictors of CNS involvement, irrespective of sex, ethnicity and age at symptom onset.

Conclusion: In our cohort, CNS involvement occurred in 15% of patients with BD, most commonly with a parenchymal phenotype. Non-Caucasian ethnicity, genital ulcers at presentation and constitutional and ocular manifestations during the disease course were associated with an increased risk of developing NBD. Given the high mortality rate verified in NBD (17%), a potential tailored treatment approach in patients with these disease characteristics may be justified during follow-up.

105 - MANIFESTATIONS AND PREDICTORS OF VASCULAR INVOLVEMENT IN BEHÇET'S DISEASE

Martins-Martinho J [1, 2], Margarida Lucas Rocha[1, 2, 3], Roberto Costa[1, 2], Ana Teodósio Chícharo[1, 2, 3], Carla Macieira[1], Cristina Ponte[1, 2], Nikita Khmelinskii[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar e Universitário do Algarve, Faro, Portugal

Introduction: Behçet's disease (BD) is a multisystem variable vessel vasculitis primarily affecting mucocutaneous tissues. Vascular manifestations can occur in up to 40% of patients and involve veins and arteries of all sizes, contributing to substantial morbidity and mortality. We aim to characterize BD patients with vascular involvement and to identify predictors of this clinical subtype.

Methods: Single-centre observational retrospective study using data from patients with a diagnosis of BD registered at the Rheumatic Diseases Portuguese Registry (Reuma.pt). Vascular involvement was considered when clinically suspected venous or arterial events were confirmed by imaging. Data regarding the demographics and clinical manifestations was collected.

Results: We included 160 patients with BD, 42/160 (26.3%) males, 129/147 (87.8%) Caucasian, with a median [IQR] age at diagnosis of 31.7 [17.1] years, and a median [IQR] follow-up time of 11.2 [12.3] years. Vascular involvement was diagnosed in 24/160 (15.0%) patients and in 5/24 (20.8%) at disease presentation. The vessels affected were more frequently veins than arteries (20/24, 83.3% vs. 10/24, 41.7%) and were more commonly multiple than isolated (13/24, 54.2% vs. 11/24, 45.8%). Regarding the symptoms of vascular involvement, patients presented with limb pain in 12/16 (75.0%) and oedema in 7/14 (50.0%), thoracic pain in 4/22 (18.2%), haemorrhage in 3/24 (12.5%), abdominal pain in 2/23 (8.7%), and haemoptysis in 1/22 (4.5%) of cases. Concerning venous involvement, patients presented with deep vein thrombosis (VT) in 12/24 (50.0%) and superficial VT in 10/24 (41.7%). The venous segments affected were veins of the lower limbs in 19/21 (90.5%) and inferior vena cava in 1/24 (4.2%) of patients. Concerning arterial involvement, aneurysmatic dilatation was seen in 6/24 (25.0%), pseudoaneurysm in 3/24 (12.5%) and pulmonary thrombosis in 2/24 (8.3%) of cases. The arterial segments affected were the pulmonary artery in 4/24 (16.7%), arteries of the lower limb in 3/24 (12.5%), abdominal aorta in 2/24 (8.3%), mesenteric artery in 2/24 (8.3%), and thoracic aorta in 1/24 (4.2%) of patients. Caucasian patients presented with more venous and less arterial involvement than non-Caucasians (94.7% vs. 25.0%, p=0.009, and 26.3% vs 100%, p=0.014; respectively). Table 1 shows the differences between patients with and without vascular involvement. Patients with vascular involvement were more frequently male (p=0.004), presented with ocular manifestations at disease onset (p=0.022), and cutaneous involvement at disease onset (p=0.040) and during follow-up (p=0.023) compared to patients without vascular involvement. On multivariate analysis, male sex (OR 3.29, 95%CI: 1.29-8.39) and ocular involvement at presentation (OR 3.41, 95%CI: 1.13-10.27) were independent predictors of vascular involvement, irrespective of age at diagnosis and cutaneous involvement at disease onset. Patients with vascular involvement were more frequently treated with cyclophosphamide (p=0.018) and anticoagulants (p<0.001) than patients without this involvement.

Conclusions: In our cohort, vascular involvement occurred in 15% of patients with BD during the disease course. Caucasians were more likely to present with venous and non-Caucasians with arterial involvement. Male sex, ocular involvement at disease onset, and cutaneous involvement at disease onset and during follow-up were associated with an increased risk of vascular manifestations in BD.

TO 105 – TABLE 1. Clinical and demographic differences between patients with BD with and without vascular involvement.

	With vascular involvement (N=24)	Without vascular involvement (N=136)	p-value
Demographics			
Male sex	12 (50%)	30/136 (22.1%)	0.004
Caucasians	19/23 (82.6%)	110/124 (88.7%)	0.486
Age at disease onset (median [IQR])	26.3 [16.1]	24.3 [20.0]	0.853
Age at diagnosis (median [IQR])	35.3 [13.6]	31.0 [18.1]	0.291
Comorbidities			
Arterial hypertension	9/24 (37.5%)	25/131 (19.1%)	0.045
Diabetes mellitus	2/24 (8.3%)	9/131 (6.9%)	0.680
Dyslipidaemia	4/24 (16.7%)	23/130 (17.7%)	1.000
Manifestations at disease onset			
Oral ulcers	21/24 (87.5%)	117/133 (88.0%)	1.000
Genital ulcers	8/24 (33.3%)	51/133 (38.3%)	0.641
Ocular manifestations[1]	7/24 (29.2%)	14/133 (10.5%)	0.022
Cutaneous manifestations[2]	10/24 (41.7%)	29/132 (22.0%)	0.040
Articular manifestations[3]	2/24 (8.3%)	16/132 (12.1%)	0.742
Gastrointestinal manifestations[4]	0/24 (0%)	1/132 (0.8%)	1.000
Neurologic manifestations[5]	0/24 (0%)	1/133 (0.8%)	1.000
Constitutional symptoms	1/24 (4.2%)	10/132 (7.6%)	1.000
Manifestations at disease onset and during disease follow-up			
Oral ulcers	24/24 (100%)	129/135 (95.6%)	0.592
Genital ulcers	20/24 (83.3%)	108/136 (79.4%)	0.787
Ocular manifestations[1]	13/24 (54.2%)	46/133 (34.6%)	0.068
Cutaneous manifestations[2]	22/24 (91.7%)	92/133 (69.2%)	0.023
Articular manifestations[3]	8/24 (33.3%)	63/133 (47.4%)	0.204
Peripheral neuropathy	0/24 (0%)	1/135 (0.7%)	1.000
Gastrointestinal manifestations[4]	2/24 (8.3%)	15/135 (11.1%)	1.000
Neurologic manifestations[5]	5/24 (20.8%)	19/134 (14.2%)	0.370
Constitutional symptoms	6/24 (25.0%)	17/135 (12.6%)	0.121
Positive pathergy test	5/13 (38.5%)	28/79 (35.4%)	1.000
Presence of HLAB51	4/11 (36.4%)	32/69 (46.4%)	0.746
Mortality	2/24 (8.3%)	4/136 (2.9%)	0.222
Treatment			
Glucocorticoids	20/23 (87.0%)	108/135 (80.0%)	0.571
csDMARD	17/23 (73.9%)	85/133 (63.9%)	0.478
bDMARD	5/23 (21.7%)	17/131 (13.0%)	0.329
Cyclophosphamide	5/23 (21.7%)	7/132 (5.3%)	0.018
Anticoagulation	7/22 (31.8%)	3/134 (2.2%)	<0.001

csDMARD: conventional synthetic disease modifying antirheumatic drugs; bDMARD: biologic disease modifying antirheumatic drugs. 1Ocular manifestations included anterior and/or posterior uveitis, retinal vasculitis, and central retinal vein or artery occlusion; 2Cutaneous manifestations included erythema nodosum, pseudofoliculitis, and papulopustular or acneiform lesions; 3Articular manifestations included inflammatory arthralgia or arthritis; 4Gastrointestinal manifestations included abdominal pain, diarrhea, bowel obstruction, and bowel perforation; 5Neurologic manifestations included headache, pyramidal signs, behavioral and psychiatric alterations, meningeal signs, cerebellar signs, hearing impairment, hemichorea, memory impairment, gait disturbances, seizures, diplopia, and ataxia.

107 - CHILDREN WITH EXTENDED OLIGOARTICULAR AND POLYARTICULAR JUVENILE IDIOPATHIC ARTHRITIS HAVE ALTERATIONS IN B AND T FOLLICULAR CELL SUBSETS IN PERIPHERAL BLOOD AND A CYTOKINE PROFILE SUSTAINING B CELL ACTIVATION

Catarina Tomé[1], Filipa Oliveira Ramos [1, 2], Raquel Campanilho-Marques[1, 2], Ana Filipa Mourão[3], Sandra Sousa[4], Cláudia Marques[1], Ana Teresa Melo[1, 2], Rui Lourenço Teixeira[1, 2], Ana P. Martins[5], Sofia Moeda[6], Patrícia Costa Reis[1, 6], Rita Pinheiro Torres [3], Matilde Bandeira[1, 2], Helena Fonseca[6], Miroslava Gonçalves[5], Maria José Santos[1, 4], L Graca[1], JE Fonseca[1, 2], Rita A. Moura[1]

[1]Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Rheumatology Department, Centro Hospitalar Universitário Lisboa Norte, EPE, Hospital de Santa Maria, Lisboa, Portugal, [3]Rheumatology Department, Centro Hospitalar Lisboa Ocidental, EPE, Hospital de São Francisco Xavier, Lisboa, Portugal, [4]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [5]Pediatric Surgery Department, Centro Hospitalar Universitário Lisboa Norte, EPE, Hospital de Santa Maria, Lisboa, Portugal, [6]Pediatric Department, Centro Hospitalar Universitário Lisboa Norte, EPE, Hospital de Santa Maria, Lisboa, Portugal

Background: Juvenile idiopathic arthritis (JIA) is the most common rheumatic disease in children. Our group has recently demonstrated that extended oligoarticular and polyarticular JIA mostly evolve to a rheumatoid arthritis (RA)-like phenotype in adulthood. Disturbances in B cells, T follicular helper (Tfh) and T follicular regulatory (Tfr) cell immune responses are associated with RA pathogenesis, but their exact role in JIA development is poorly understood.
Objectives: The main goal of this study was to characterize the frequency and phenotype of B, Tfh and Tfr cells in peripheral blood and the cytokine environment present in circulation in children with with extended oligoarticular JIA (eoJIA) and polyarticular JIA (pJIA) when compared to healthy controls, children with persistent oligoarticular JIA (poJIA) and adult JIA patients.
Methods: Blood samples were collected from 105 JIA patients (children and adults) and 50 age-matched healthy individuals. Peripheral blood mononuclear cells were isolated and the frequency and phenotype of B, Tfh and Tfr cells were evaluated by flow cytometry. Serum levels of APRIL, BAFF, IL-1β, IL-2, IL-4, IL-6, IL-10, IL-17A, IL-21, IL-22, IFN-γ, PD-1, PD-L1, sCD40L, CXCL13 and TNF were measured by multiplex bead-based immunoassay and/ or ELISA in all groups included.
Results: The frequency of B, Tfh and Tfr cells was similar between JIA patients and controls. Children with eoJIA and pJIA, but not poJIA, had significantly lower frequencies of plasmablasts, regulatory T cells and higher levels of Th17-like Tfh cells in circulation when compared to controls. Furthermore, APRIL, BAFF, IL-6 and IL-17A serum levels were significantly higher in pediatric eoJIA and pJIA patients when compared to controls. These immunological alterations were not found in adult JIA patients in comparison to controls.
Conclusions: Our results suggest a potential role and/ or activation profile of B and Th17-like Tfh cells in the pathogenesis of eoJIA and pJIA, but not poJIA.

109 - MULTIFACTORIAL EXPLANATORY MODEL OF SLEEP DISTURBANCE IN PATIENTS WITH RHEUMATOID ARTHRITIS: A STRUCTURAL EQUATION APPROACH

Maria João Cadório[1], Eduardo Santos[1, 2, 3], João Alexandre Oliveira[1], Ricardo J. O. Ferreira[1, 3, 4], JAP da Silva[1, 5], Cátia Duarte[1, 5]

[1]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal, [2]Instituto de Ciências Biomédicas Abel Salazar, Porto, Portugal, [3]Health Sciences Research Unit: Nursing (UICiSA:E), Coimbra, Portugal, [4]Centro de Investigação, Inovação e Desenvolvimento em Enfermagem de Lisboa (CIDNUR), Escola Superior de Enfermagem de Lisboa (ESEL), Lisboa, Portugal, [5]Coimbra Institute for Clinical and Biomedical Research (iCBR) - Faculty of Medicine, University of Coimbra, Coimbra, Portugal

Background: Around 38% of people with Rheumatoid Arthritis (RA) have sleep disturbances, a number that increases with disease duration. However, their origin is still poorly understood, and its management frequently neglected.
Objective: To foster the understanding of sleep disturbances in patients with RA through a multifactorial explanatory model incorporating the influence of disease activity, perceived disease impact, personality traits and comorbidities, namely depression and anxiety.
Methods: This is a cross-sectional analysis in a cohort from an outpatient clinic in a tertiary Portuguese hospital using a structural equation modelling estimation to analyse the associations between sleep disturbance and disease activity, perceived disease impact, personality traits, and depression and anxiety dimensions.
Sleep disturbance was assessed using the Sleep Disturbance item (0-10, NRS) from the Rheumatoid Arthritis Impact of Disease (RAID) questionnaire; disease activity through the Disease Activity Score 28 joints in its three variables (3v) and C-reactive protein (CRP) variant (DAS28CRP3v); disease impact by the individual domains of RAID.7 questionnaire; depressive and anxiety

TO 109 – TABLE 1. Clinical and serological features of patients with and without interstitial lung disease

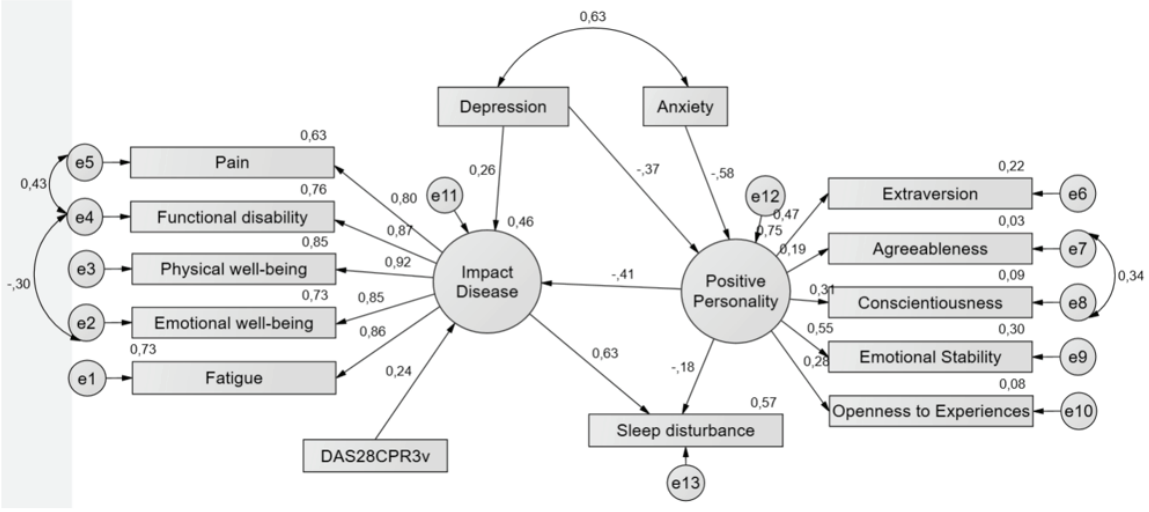
	Patients with ILD (n = 64)	Patients without ILD (n = 136)	Univariate analysis (p-value)
Age at diagnosis, median (IQR)	50.5 (25.8)	46.5 (33.5)	p = 0.165
Disease duration (in years), median (IQR)	4.0 (3.0)	5.0 (8.0)	p = 0.072
Female, n (%)	42 (65.6)	106 (77.9)	p = 0.064
Mortality, n (%)	1 (1.6)	7 (5.1)	p = 0.440
Diagnosis	ILD subtypes		
Antisynthetase syndrome (N = 34)	NSIP, n (%): 26 (76.5) UIP, n (%): 5 (14.7) OP, n (%): 1 (2.9) OP/NSIP overlap, n (%): 1 (2.9) LIP, n (%): 1 (2.9)	NA	NA
Dermatomyositis (N = 13)	NSIP, n (%): 8 (61.5) OP, n (%): 3 (23.1) OP/NSIP overlap, n (%): 1 (7.7) AIP, n (%): 1 (7.7)	NA	NA
Polymyositis (N = 2)	NSIP, n (%): 1 (50.0) DIP, n (%): 1 (50.0)	NA	NA
Overlap syndrome (N = 10)	NSIP, n (%): 6 (60.0) UIP, n (%): 3 (30.0) OP, n (%): 1 (10.0)	NA	NA
Undifferentiated connective tissue disease (N = 3)	NSIP, n (%): 3 (100.0)	NA	NA
Mixed connective tissue disease (N = 2)	NSIP, n (%): 2 (100.0)	NA	NA
Musculoskeletal involvement			
MMT8 last value, median (IQR)	150.0 (8.0)	150.0 (16.0)	
Maximum	150.0	150.0	p = 0.301
Minimum	100.0	86.0	
Arthritis, n/N (%)	39/62 (62.9)	37/135 (27.4)	p < 0.001
Skin involvement			
Highest modified DAS skin, median (IQR)	0.0 (2.0)	0.0 (2.0)	p = 0.855
Calcinosis, n/N (%)	3/63 (4.8)	12/135 (8.9)	p = 0.396
Mechanic's hands, n/N (%)	19/63 (30.2)	15/135 (11.1)	p < 0.001
Raynaud's phenomenon, n/N (%)	29/63 (46.0)	40/135 (29.6)	p = 0.024
Internal organ involvement			
Heart involvement	2/64 (3.1)	4/136 (2.9)	p = 1.000
Esophageal involvement, n/N (%)	11/63 (17.5)	6/135 (4.4)	p = 0.002
Neoplasia			
Diagnosis of neoplasia, n/N (%)	5/64 (7.8)	12/136 (8.8)	p = 0.811
Antibodies	N = 60	N = 98	
Anti-Ro52, n (%)	4 (6.7)	9 (9.2)	p = 0.768
Anti-Ro60, n (%)	1 (1.7)	1 (1.0)	p = 1.000
Anti-Jo1, n (%)	25 (41.7)	4 (4.1)	p < 0.001

continues on the next page

TO 109 – TABLE 1. Continuation

	Patients with ILD (n = 64)	Patients without ILD (n = 136)	Univariate analysis (p-value)
Anti-PL7, n (%)	4 (6.7)	3 (3.1)	p = 0.428
Anti-PL12, n (%)	6 (10.0)	1 (1.0)	p = 0.012
Anti-EJ, n (%)	1 (1.7)	0 (0.0)	p = 0.380
Anti-PM/Scl100, n (%)	1 (1.7)	3 (3.1)	p = 1.000
Anti-PM/Scl75, n (%)	5 (8.3)	4 (4.1)	p = 0.302
Anti-Ku, n (%)	1 (1.7)	7 (7.1)	p = 0.157
Anti-U1-RNP, n (%)	1 (1.7)	14 (14.3)	p = 0.009
Anti-Mi2a, n (%)	0 (0.0)	7 (7.1)	p = 0.045
Anti-Mi2b, n (%)	0 (0.0)	13 (13.3)	p = 0.002
Anti-TIF1γ, n (%)	2 (3.3)	10 (10.2)	p = 0.134
Anti-MDA5, n (%)	7 (11.7)	3 (3.1)	p = 0.043
Anti-NXP2, n (%)	0 (0.0)	4 (4.1)	p = 0.298
Anti-SAE1, n (%)	1 (1.7)	5 (5.1)	p = 0.409
Anti-SRP, n (%)	0 (0.0)	6 (6.1)	p = 0.083
ANA, n/N (%)	47/60 (78.3)	66/115 (57.4)	p = 0.006

ILD: interstitial lung disease; n: number of patients positive for the variable of interest; N: number of patients without missing information regarding the variable of interest; NA: not applicable; NSIP: non-specific interstitial pneumonia; OP: organizing pneumonia; UIP: usual interstitial pneumonia; AIP: acute interstitial pneumonia; LIP: lymphocytic interstitial pneumonia; DIP: desquamative interstitial pneumonia. MMT8, manual muscle testing; DAS, disease activity score; Jo1, histidyl tRNA synthetase; PL7, threonyl tRNA synthetase; PL12, anti-alanyl tRNA synthetase; EJ, glycyl tRNA synthetase; Pm/Scl, polymyositis/scleroderma; RNP, ribonucleoprotein; TIF1g, transcription intermediary factor 1-gamma; MDA5, melanoma differentiation-associated gene 5; NXP2, nuclear matrix protein 2; SAE, small ubiquitin-like modifier activating enzyRo52/me; SRP, signal recognition particle; ANA, anti- nuclear antibodies.
All statistically significant differences between groups are shown in bold.



DAS28CRP3v= Disease Activity Score using 28 joints and C-reactive protein 3 variables. Circles represent latent factors. Squares represent measured variables (the scale scores). Arrows connecting circles and rectangles in one direction show a hypothesized direct relationship between the two variables. Circles with the letter "e" written in it represent the associated error.

TO 109 – Figure 1. Estimated standardised direct effects for the proposed model.

symptoms were evaluated using the Hospital Anxiety and Depression Scale (HADS); and personality by the Ten Item Personality Inventory (TIPI). We designated the latent factor derived from TIPI as "Positive" personality, to denote the predominantly adaptive nature of the represented dimensions.
Descriptive and correlational analyses were performed using SPSS®, v. 29 (IBM, Armonk NY, USA) software. Statistically significant effects were assumed for $p<0.05$.
Results: A total of 302 RA patients were included in this analysis. The results obtained in the structural equation model indicated a good fit to the data, explaining 57% of the variance of the sleep disturbance (R2=0.57). "Positive" personality and disease activity explained 46% of the variance of perceived impact of disease (R2=0.46). Furthermore, the results corroborate a direct association between disease activity [DAS-28CRP3v] and impact of disease (β=0.24; $p<0.001$) which, in turn, showed a significant positive direct relation with sleep disturbance (β=0.63; $p<0.001$). "Positive" personality traits had a total effect of β=-0.44 on sleep disturbance, including a direct effect of β=-0.18 ($p=0.006$) and an indirect effect of β=-0.26 ($p=0.001$) through impact of disease, indicating a mediating influence in this association. The model also shows a direct negative relation between "positive" personality traits and impact of disease (β=-0.41; $p<0.001$). Depression showed a significant positive direct relation with impact of disease (β=0.26; $p=0.005$) and a significant negative direct relation with "positive" personality (β=-0.37; $p<0.001$). Anxiety showed a significant negative direct relation with "positive" personality (β=-0.37; $p<0.001$) (Figure 1).
Conclusion: Positive personality traits and perceived impact of disease seem to have a major role in explaining sleep disturbances in patients with RA. In contrast, disease activity and comorbidities such as depression and anxiety play a secondary role. This evidence reinforces the need to consider a multidisciplinary approach to people living with RA, that goes beyond a treat-to-target strategy focused on disease activity.

112 - CLINICAL CHARACTERISTICS AND BURDEN OF PORTUGUESE AXIAL SPONDYLOARTHRITIS PATIENTS: RESULTS OF THE REAL-LIFE PROOF STUDY

Helena Santos[1], Sofia Pimenta[2], P Pinto[3], Fernando Pimentel-Santos[4], Anabela Barcelos[5], Rita Mateus[6], Elsa Vieira-Sousa[7, 8]

[1]Instituto Português de Reumatologia, Lisboa, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/ Espinho, Vila Nova de Gaia, Portugal, [4]Serviço de Reumatologia, Centro Hospitalar Lisboa Ocidental | Hospital Egas Moniz, Lisboa, Portugal, [5]Serviço de Reumatologia, Centro Hospitalar do Baixo Vouga, E.P.E., Aveiro, Portugal, [6]Medical, AbbVie, Portugal, Lisboa, Portugal, [7]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Hospital de Santa Maria, CHULN, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [8]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background: Axial spondyloarthritis (axSpA) is a chronic rheumatic musculoskeletal disease with diverse manifestations, often with inflammatory back pain (IBP). It comprises a spectrum of radiographic (r-axSpA) and non-radiographic axSpA (nr-axSpA) patients.1 This study aimed to assess epidemiological and clinical characteristics, and the burden of axSpA, and to compare baseline characteristics of r-axSpA and nr-axSpA, in the sample of Portuguese patients included in the observational prospective multi-country PROOF study.2 Baseline characteristics of European patients included in the PROOF study are also described to contextualize the Portuguese results.
Methods: From December 2014 to June 2015, adults with undiagnosed chronic back pain for ≥3 months and onset before 45 years old were enrolled and screened for axSpA based on the ASAS criteria. Those classified as axSpA were characterized at baseline and followed yearly for 5 years, including disease activity measures [BASDAI, Patient global assessment (PtGA)], function (BASFI), quality of life (QoL, assessed by SF12), and work productivity and activity impairment (WPAI). Classification of r-axSpA and nr-SpA, and of active sacroiliitis on MRI were performed by the physician. Clinical characteristics were summarized at baseline and r-axSpA and nr-axSpA patients were compared through chi-square or Fisher's exact test (categorical variables), or t-test or non-parametrical Mann-Whitney test (numerical variables). Generalized Estimating Equations (GEE) were performed to estimate statistical significance of changes from baseline during follow-up (α=0.05), regarding disease activity, QoL and WPAI.
Results: Of 76 patients screened in 6 Portuguese centers, 47 were classified as axSpA [nr-axSpA: 27 (57.4%)] - Table. Mean age was 38.0 ± 12.0 years and symptom duration 5.9 ± 8.4 years, 36.2% of patients were men, 14.9% had SpA family history, and 63.8% were referred by a general practitioner. Peripheral and extra-musculoskeletal manifestations affecting ≥10% of patients were enthesitis (29.8%), peripheral arthritis (23.4%), and psoriasis (12.8%). Active inflammation on MRI was reported in 46.3% of patients. Most patients (85.1%)

TO 112 – TABLE. Clinical characteristics, SpA features and treatment at baseline, in patients with axSpA

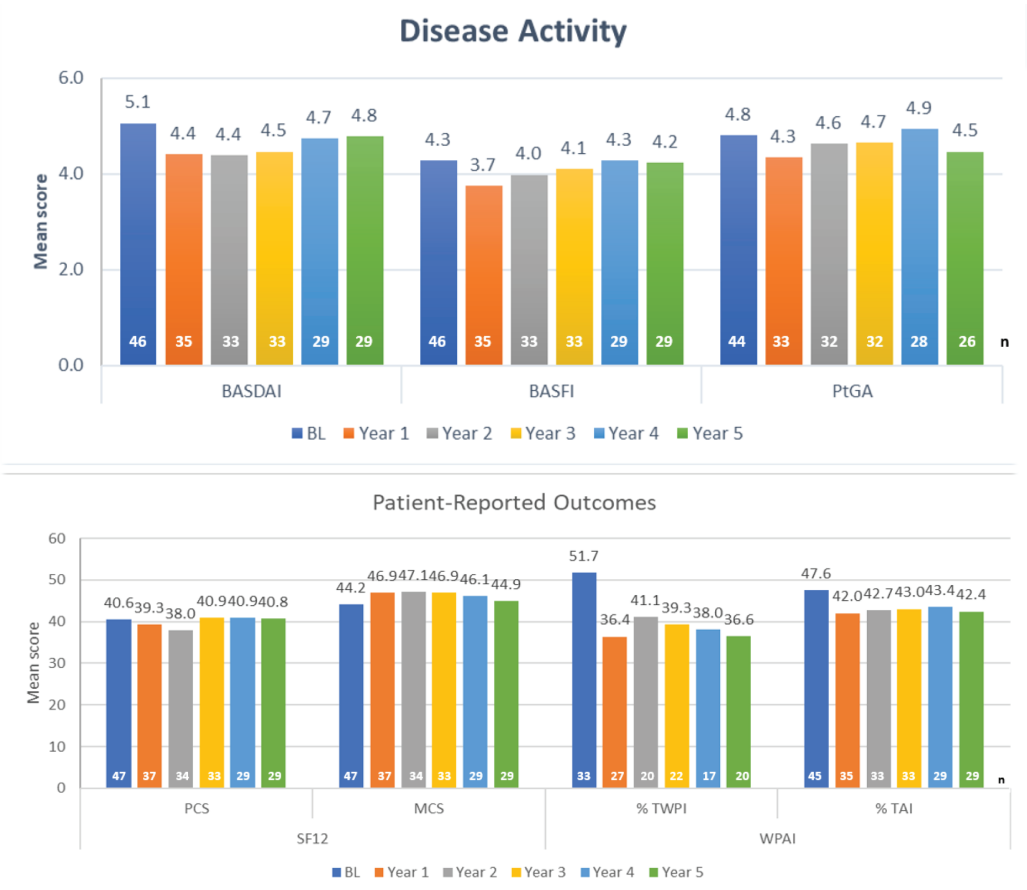
	Europe Region [a]	Portuguese Results			
	Overall N=958	Overall n = 47	nr-axSpA n = 27	r-axSpA n = 20	p-value [d]
Age, years, mean ± SD	36.3 ± 10.5	38.0 ± 12.0	34.9 ± 7.5	42.1 ± 15.5	0.228 MW
Male, n (%)	570 (59.5%)	17 (36.2%)	5 (18.5%)	12 (60.0%)	0.003 CS
Current smokers, n (%)	NR	11 (23.4%)	3 (11.1%)	8 (40.0%)	0.021 FE
CBP duration, years, mean ± SD	5.1 ± 7.3 [n=944]	5.9 ± 8.4	5.4 ± 6.3	6.5 ± 10.8)	0.821 MW
Referred from specialist, n (%)	NR				--
Dermatologist		2 (4.3%)	0 (0.0%)	2 (10.0%)	
Gastroenterologist		2 (4.3%)	1 (3.7%)	1 (5.0%)	
General Practitioner		30 (63.8%)	17 (63.0%)	13 (65.0%)	
Orthopedist		5 (10.6%)	4 (14.8%)	1 (5.0%)	
Other		4 (8.5%)	1 (3.7%)	3 (15.0%)	
Patient initiative		4 (8.5%)	4 (14.8%)	0 (0.0%)	
Sacroiliitis on imaging, n (%)	NR	[n=41]	[n=21]	[n=20]	<0.001 CS
Active sacroiliitis on MRI		19 (46.3%)	17 (81.0%)	2 (10.0%)	
Definite radiographic sacroiliitis – modified New York criteria		22 (53.7%)	4 (19.0%)	18 (90.0%)	
SpA features[b], mean ± SD	3.7 ± 1.4 [n=776]	3.5 ± 1.3	3.3 ± 1.0	3.8 ± 1.7	
SpA features [b], n (%)					
Inflammatory back pain	908 (94.8%)	43 (91.5%)	27 (100.0%)	16 (80.0%)	0.054 FE
Peripheral arthritis	286 (29.9%)	11 (23.4%)	6 (22.2%)	5 (25.0%)	>0.999 FE
Enthesitis (heel)	374 (39.0%)	14 (29.8%)	9 (33.3%)	5 (25.0%)	0.537 CS
Psoriasis	82 (8.6%)	6 (12.8%)	2 (7.4%)	4 (20.0%)	--
Crohn's disease or ulcerative colitis	28 (2.9%)	3 (6.4%)	2 (7.4%)	1 (5.0%)	--
Uveitis	92 (9.6%)	3 (6.4%)	1 (3.7%)	2 (10.0%)	--
Dactylitis	55 (5.7%)	3(6.4%)	0 (0.0%)	3 (15.0%)	--
Family history for SpA	190 (19.8%)	7 (14.9%)	4 (14.8%)	3 (15.0%)	>0.999 FE
Good response to NSAIDs	614 (64.1%)	25 (53.2%)	14 (51.9%)	11 (55.0%)	0.831 CS
Elevated C-reactive protein	453 (47.3%)	24 (51.1%)	10 (37.0%)	14 (70.0%)	0.025 CS
HLA-B27 positive	447 (57.6% [n=776]	26 (68.4%) [n=38]	15 (71.4%)[n=21]	11 (64.7% [n=17]	0.658 CS
Current axSpA treatment, n (%)					
NSAIDs	764 (79.7%)	40 (85.1%)	22 (81.5%)	18 (90.0%)	0.704 FE
csDMARDs	255 (26.6%)	14 (29.8%)	8 (29.6%)	6 (30.0%)	0.978 CS
methotrexate	50 (5.2%)	3 (6.4%)	1 (3.7%)	2 (10.0%)	--
sulfasalazine	193 (20.1%)	11 (23.4%)	7 (25.9%)	4 (20.0%)	--
csDMARDs, other	18 (1.9%)	1 (2.1%)	0 (0.0%)	1 (5.0%)	--
Systemic corticosteroids	89 (9.3%)	4 (8.5%)	2 (7.4%)	2 (10.0%)	--
Analgesics	191 (19.9%)	9 (19.1%)	4 (14.8%)	5 (25.0%)	0.611 FE
TNF inhibitors	121 (12.6%)	2 (4.3%)	2 (7.4%)	0 (0.0%)	--
Disease activity/PRO measures, mean ± SD					
C-reactive protein level, mg/L	15.0 ± 22.2 [n=868]	23.5 ± 40.9 [n=41]	22.5 ± 38.4 [n=24]	24.9 ± 45.5 [n=17]	0.721 MW
BASDAI	4.8 ± 2.3 [n=949]	5.1 ± 2.2 [n=46]	5.3 ± 2.2 [n=27]	4.7 ± 2.1 [n=19]	0.292 TT
BASFI	3.8 ± 2.5 [n=948]	4.3 ± 2.6 [n=46]	4.1 ± 2.8 [n=27]	4.6 ± 2.4 [n=19]	0.475 MW

continues on the next page

TO 112 – TABLE. Continuation.

	Europe Region [a]	Portuguese Results			
	Overall N=958	Overall n = 47	nr-axSpA n = 27	r-axSpA n = 20	p-value [d]
PtGA	5.2 ± 2.8 [n=917]	4.8 ± 2.8 [n=44]	5.0 ± 3.0 [n=26]	4.5 ± 2.5 [n=18]	0.569 TT
SF-12 v2 PCS	40.1 ± 8.7 [n=943]	40.6 ± 8.7 [n=47]	40.0 ± 8.7 [n=27]	41.4 ± 8.8 [n=20]	0.570 TT
SF-12 v2 MCS	43.8 ± 10.5 [n=943]	44.2 ± 10.2 [n=47]	45.5 ± 9.7 [n=27]	42.5 ± 10.9 [n=20]	0.327 TT
WPAI-SHP % TWPI [c]	41.7 ± 29.9 [n=477]	51.7 ± 33.6 [n=33]	59.8 ± 28.5 [n=21]	37.5 ± 38.2 [n=12]	0.060 MW
WPAI-SHP % TAI	47.1 ± 28.0 [n=936]	47.6 ± 30.8 [n=45]	50.4 ± 30.9 [n=27]	43.3 ± 30.9 [n=18]	0.462 MW

SD, standard deviation. SpA, SpondyloArthritis. axSpA, axial SpA. nr-axSpA, non-radiographic axSpA. r-axSpA, radiographic axSpA. CBP, chronic back pain. NSAIDs, non-steroidal anti-inflammatory drugs. csDMARDs, conventional synthetic disease modifying antirheumatic drugs. TNF, tumor necrosis factor. HLA-B27, human leukocyte antigen B27. MRI, Magnetic resonance imaging. PRO, patient-reported outcomes. BASDAI, Bath Ankylosing Spondylitis Disease Activity Index. BASFI, Bath Ankylosing Spondylitis Functional Index. PtGA, Patient Global Assessment. MCS, mental component summary. PCS, physical component summary. SF-12v2, 12-item Short Form Health Survey version 2. TAI, total activity impairment. TWPI, total work productivity impairment. WPAI-SHP, Work Productivity and Activity Impairment Questionnaire–Specific Health Problem. NR, not reported.
a) as reported by Poddubnyy D et al. Characteristics of patients with axial spondyloarthritis by geographic regions: PROOF multicountry observational study baseline results. Rheumatology 2022;61:3299–3308.
b) SpA features included in the ASAS classification criteria for axSpA excluding imaging.
c) Only for patients currently employed and with information.
d) p-value for the comparison between r-axSpA and nr-SpA, based on chi-square (CS), Fisher's exact (FE), Mann-Whitney (MW), or t-test (TT). Bivariate analysis of categorical variables with less than 10% of individuals by category was not applicable (--).



Legend: BL, baseline. BASDAI, Bath Ankylosing Spondylitis Disease Activity Index. BASFI, Bath Ankylosing Spondylitis Functional Index. PtGA, Patient Global Assessment. SF12, 12-item Short Form Health Survey. MCS, mental component summary. PCS, physical component summary. WPAI, Work Productivity and Activity Impairment Questionnaire. TWPI, total work productivity impairment. TAI, total activity impairment.

TO 112 – Figure 1. Disease Activity and Patient-Reported Outcomes at baseline and during follow-up

received NSAIDs at baseline, 29.8% csDMARDs, and 8.5% corticosteroids. Physical function, QoL, and activity impairment at baseline were similar between the nr-axSpA and r-axSpA subgroups. Overall, the characteristics of Portuguese patients overlap those of the PROOF European subgroup, although baseline disease severity seems to be higher in the Portuguese patients (e.g., BASDAI mean score of 5.1 compared to 4.8 in the European region). Statistically significant reductions of BASDAI were observed from baseline to year 1 (mean change: -0.59 [95%CI: -1.15, -0.04]; p=0.035) and to year 2 (-0.99 [96%CI: -1.77; -0.20]; p=0.013) - Figure. Work productivity impairment (TWPI) also reduced from baseline to year 1 (mean change: -14.4% [95%CI: -26.7%, -2.1%]; p=0.022). No other statistically significant changes were observed during follow-up.

Conclusion: Our results suggest that r-axSpA and nr-axSpA patients in Portugal have similar clinical presentation and burden of disease, and that overall baseline characteristics are aligned with those reported for European patients included in the PROOF study. Despite an improvement of BASDAI and TWPI impairment after one year of follow-up, disease activity and patient-reported outcomes remained stable over 5 years.

113 - SYSTEMIC VASCULITIDES IN PORTUGAL AND BRAZIL: PRELIMINARY RESULTS FROM THE REUMA.PT/ VASCULITIS REGISTRY

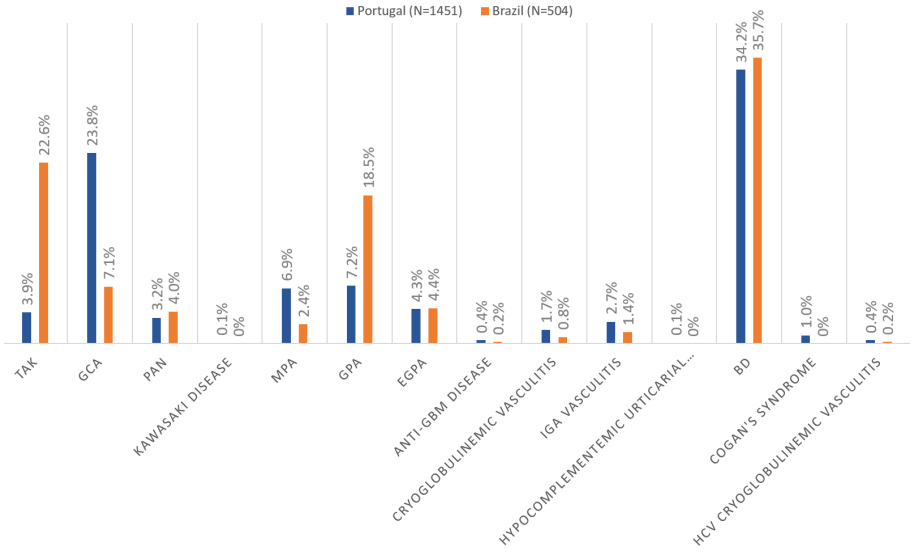
Mariana Aguiar[1], Alexandre Souza[1], Martins-Martinho J [2, 3], Adriana Carones[4], Ana Freitas[5], Ana Catarina Duarte[6], Ana Filipa Rocha Águeda[7], Camila Elias[8], Catarina Cortesão[9], Catarina Soares[10], Carla Macieira[2], Carlos Teixeira[11], Daniela Peixoto[10], Duarte Pereira Vinha[2, 3], Estela Nogueira[12], Fabricia Simil[13], Francisca Guimarães[10], Frederico Rajão Martins[14], Gilda Ferreira[13], Helena Assunção[4], Heloisa Tiemi Arume Rulff[8], Jorge Pestana Lopes[6], José Costa[12], Julia Brito de Medeiros[15], Lilian Santuza Santos Porto[16], Luiz Felipe Adsuara de Sousa[15], Manuella Lima Gomes Ochtrop[8], Maria João Gonçalves[17], Mariana Diz-Lopes [18], Matheus Vieira[1], Nikita Khmelinskii[2, 3], Rita Pinheiro Torres [17], Sarah Abati Curi Neaime[1], Vítor Teixeira[14], Zoraida Sachetto[11], Cristina Ponte[2, 3]

[1]Serviço de Reumatologia, Universidade Federal de São Paulo, Sao Paulo, Brazil, [2]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [4]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [5]Serviço de Reumatologia, Hospital de Barretos, Sao Paulo, Brazil, [6]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [7]Rheumatology Department, Centro Hospitalar e Universitário Cova da Beira, Covilhã, Portugal, [8]Serviço de Reumatologia, Universidade Estadual do Rio de Janeiro, Rio de Janeiro, Brazil, [9]Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal, [10]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [11]Serviço de Reumatologia, Universidade Estadual de Campinas, Campinas, Brazil, [12]Serviço de Nefrologia e Transplantação Renal, Centro Hospitalar e Universitário Lisboa Norte, Lisboa, Portugal, [13]Serviço de Reumatologia, Universidade Federal de Minas Gerais, Belo Horizonte, Brazil, [14]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, [15]Serviço de Reumatologia, Santa Casa de Misericórdia de São Paulo, Sao Paulo, Brazil, [16]Serviço de Reumatologia, Santa Casa de Misericórdia de Belo Horizonte, Belo Horizonte, Brazil, [17]Rheumatology Department, Centro Hospitalar Lisboa Ocidental, Lisboa, Portugal, [18]Rheumatology Department, Centro Hospitalar de São João, Porto, Portugal

Background: The epidemiology of vasculitis varies widely across different geographic areas of the world which may be due to different ethnic and environmental factors. Brazil has a heterogeneous population with influences from Indigenous, African, Asian, and European countries, while Portugal has a very ethnically homogeneous background. This study aims to assess the main differences in the profile of systemic vasculitides between Portugal and Brazil.

Methods: Collaborative project between the Portuguese and the Brazilian Societies of Rheumatology in which centres from both countries were invited to register data in the vasculitis module of the Rheumatic Diseases Portuguese Register, Reuma.pt/vasculitis. A cross-sectional analysis was performed comparing demographic, ethnic and diagnostic information between Brazilian and Portuguese centres.

Results: A total of 1955 patients were analysed: 74.2% from 30 Portuguese centres and 25.8% from 7 Brazilian centres. Portuguese patients were predominantly European White (89.2%) and in Brazil the most common ethnic groups with vasculitis were the non-European White (48.3%) and Mestizos (40.1%); 5.4% of all participants were born in other countries. Brazilian patients were younger at the onset of symptoms [35.2 (24.1-46.6) vs. 48.1 (27.4-70.1) years; p<0.05] and diagnosis of vasculitis [37.0 (27.6-48.3) vs. 50.8 (32.7-70.4) years; p<0.05] than Portuguese patients, respectively. When analysing individual forms of vasculitis, Brazilian patients with giant cell arteritis (GCA), Takayasu arteritis (TAK), polyarteritis nodosa (PAN) and granulomatosis with polyangiitis (GPA) were significantly younger than Portuguese patients at diagnosis (p<0.05). The



TO 113 – Figure 1. Comparison of the prevalence of different vasculitis subtypes between Portugal and Brazil.
TAK: Takayasu arteritis; GCA: Giant cell arteritis; PAN: Polyarteritis nodosa; MPA: Microscopic polyangiitis (MPA); GPA: Granulomatosis with polyangiitis; EGPA: Eosinophilic granulomatosis with polyangiitis;GBP; Glomerular basement membrane; Hypocomplementemic urticarial vasculitis; BD: Behçet's disease; HCV: Hepatitis C Virus.

TO 113 – TABLE 1.

Variables	Portugal	Brazil	p
Behçet's disease			
Females, %	75.5	67.2	0.033*
Age at diagnosis, years	33.3 (25.0-42.3)	33.3 (27.0-40.7)	0.878
Time between symptoms and diagnosis, years	4.00 (1.00-12.00)	0.99 (0.32-4.00)	< 0.0001*
Giant cell arteritis			
Females, %	66.0	80.6	0.076
Age at diagnosis, years	75.3 (69.4-80.4)	68.6 (61.7-75.4)	< 0.0001*
Time between symptoms and diagnosis, years	0.16 (0.05-0.37)	0.43 (0.08-1.83)	0.014*
Takayasu arteritis			
Females, %	85.2	93.9	0.066
Age at diagnosis, years	37.0 (24.3-49.6)	30.3 (23.3-40.4)	0.020*
Time elapsed between symptoms and diagnosis, years	1.16 (0.58-4.61)	1.08 (0.41-4.37)	0.692
Polyarteritis nodosa			
Females, %	56.8	70.0	0.316
Age at diagnosis, years	43.1 ± 17.1	32.3 ± 14.4	0.027*
Time between symptoms and diagnosis, years	1.00 (0.49-2.87)	0.48 (0.29-3.83)	0.463
Granulomatosis with polyangiitis			
Females, %	58.6	60.2	0.818
Age at diagnosis, years	51.5 ± 15.8	43.9 ± 14.2	0,001*
Time elapsed between symptoms and diagnosis, years	0.67 (0.24-3.00)	0.99 (0.29-2.08)	0.600
Microscopic polyangiitis			
Females, %	57.3	75.0	0.239
Age at diagnosis, years	64.3 (54.8-74.1)	58.2 (38.2-74.5)	0.352
Time elapsed between symptoms and diagnosis, years	0.42 (0.10-0.91)	0.33 (0.23-1.58)	0.738
Eosinophilic granulomatosis with polyangiitis			
Females, %	61.0	54.5	0.598
Age at diagnosis, years	54.7 ± 15.8	49.6 ± 11.1	0.176
Time elapsed between symptoms and diagnosis, years	1.56 (0.26-6.50)	2.00 (0.83-4.00)	0.480

Continuous data are presented as median and interquartile range or as mean and standard deviation; * - Flags significant results.

proportion of females was higher in Portuguese patients with Behçet's disease (BD) than in Brazilian patients (p=0.03). No differences regarding the proportion of females were observed for other vasculitides (Table 1). The most common form of vasculitis in both countries was BD followed by GCA in Portugal and by TAK in Brazil. Regarding ANCA-associated vasculitis, GPA was more common in Brazil and microscopic polyangiitis (MPA) in Portugal. Both countries had similar proportions of patients with PAN and eosinophilic granulomatosis with polyangiitis (EGPA) (Figure 1). Time elapsed between the onset of symptoms and the diagnosis of GCA was higher in Brazil than in Portugal (p=0.014) while Portugal had a longer interval between the onset of BD symptoms and its diagnosis compared to Brazil (p<0.05) (Table 1).

Conclusion: In this large multicentre binational study, Portugal and Brazil had a different profile of systemic vasculitis concerning the proportion of GCA and TAK patients, as well as GPA and MPA patients. In addition, both countries had differences in the age of onset, female gender, and ethnicity of patients with systemic vasculitis.

114 - HIP INVOLVEMENT IN AXIAL SPONDYLARTHRITIS PATIENTS: COMPARISSON BETWEEN ANKYLOSING SPONDYLITIS AND NON-RADIOGRAPHIC AXIAL SPONDYLARTHRITIS - A RETROSPECTIVE STUDY

Sofia Ferreira Azevedo[1, 2], Carolina Mazeda[1, 2], Susana P. Silva[1, 2], Anabela Barcelos[1, 2]

[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, EPE, Aveiro, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Introduction: Hip involvement is common in axial spondylarthritis (axSpA) and represents a negative impact on the quality of life and daily functionality of these patients.

The purpose of our study is to compare hip involvement between AS and Nr-axSpA patients and to evaluate predictors of hip involvement in axSpA.

Methods: Retrospective study including patients with axSpA (all patients fulfilled ASAS criteria) followed in a Portuguese Rheumatology Center. Patients were divided into two groups: patients with AS and patients with Nr-axSpA. Sociodemographic, clinical, radiographic and laboratory data were collected. Hip involvement was evaluated by radiographic imaging and defined by a Bath ankylosing spondylitis radiology index-hip (BASRI-h) >1. Hip radiographs were analyzed by two independent and blinded observers. In case of disagreement, an evaluation by a third blinded observer was conducted. Axial radiographic involvement was evaluated by modified Stoke Ankylosing Spondylitis Spine Score (mSASSS). The grade of agreement between the two observers was accessed using Kappa and weighted Kappa coefficients.

Clinical, laboratory, and radiological findings were compared between patients with AS and Nr-axSpA using parametric and non-parametric tests. A multivariate analysis, with a stepwise backward binary and multinominal logistic regression, was performed to identify risk factors independently associated with hip involvement.

Results: 103 patients were enrolled in the study, 54 (52.4%) with AS and 49 (47.6%) with Nr-axSpA (mean age was 57.15±13.09 years, with 46.3 % of females in AS group, and 43.22±11.34 years, with 65.3% females in Nr-axSpA group). AS patients were older, had longer disease duration, higher mSASSS score, and Bath Ankylosing Spondylitis Metrology Index (BASMI) (p<0.001). There were no differences regarding the presence of extra-articular manifestations, HLA-B27 positivity, inflammation markers, disease activity scores, treatment, or comorbidities between groups.

Of all patients, 53.4% had hip involvement, 41.7% had bilateral involvement, and 26.2% were symptomatic. We found a strong grade of agreement between observers for different BASRI-h scores (weighted kappa 0.802 and 0.820, for right and left hip, respectively; p<0.001), and, an overall strong agreement when considering only the presence or absence of hip involvement (Kappa 0.801; p<0.001). Hip involvement was significantly more prevalent among AS patients (74.1% Vs 30.6%, p<0.001), and higher BASRI-h scores were encountered in AS patients [median 2 (IQR1) Vs median 1 (IQR1); p<0.001). Patients with hip involvement were older (p<0.001), had longer disease duration (p=0.03), higher mSASSS, BASMI, and BASFI scores (p<0.001), and higher frequency of dyslipidemia (p=0.01) and high blood pressure (p=0.001). On multivariate analysis, older age and higher mSASSS score were found as independently associated predictors of hip involvement in axSpA [age: OR1.08 CI(1.03-1.13), p=0.003; mSASSS:OR1.12 CI(1.03-1.22), p=0.008;].

Conclusion: Our study suggests that hip involvement is frequent in axSpA, mainly in those with AS, and that more than one-fourth of the patients are symptomatic. Older age and more severe radiographic spine involvement were potential independent risk factors for hip involvement, reinforcing the importance of closely monitoring this subset of patients, thus potentially minimizing its negative impact on patient's quality of life and functionality.

116 - THE ROLE OF OBESITY IN AXIAL SPONDYLOARTHRITIS

Sofia Ferreira Azevedo[1, 2], Carolina Mazeda[1, 2], Susana P. Silva[1, 2], C. Pinto Oliveira[1, 2], Anabela Barcelos[1, 2]
[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Introduction: Axial spondyloarthritis (axSpA) is a chronic inflammatory condition primarily affecting the axial skeleton, characterized by sacroiliitis and inflammatory back pain. While the etiology of axSpA remains multifactorial, there is growing interest in understanding the influence of obesity on disease activity, functionality, and other outcomes in this population.
The purpose of this study is to investigate the association between obesity and disease activity, radiographic progression, and functional impact in patients with axSpA.
Materials and Methods: We conducted a single-center, retrospective study of patients with axSpA who fulfilled ASAS criteria, followed at a Portuguese Rheumatology center. Patients were divided into three groups according to their Body Mass Index (BMI): Normal weight (BMI < 25 kg/m²); Overweight (BMI 25-29.9 kg/m²); Obese (BMI ≥ 30 kg/m²).
Sociodemographic, clinical, and laboratory data were collected. Disease activity scores assessed included the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) and Ankylosing Spondylitis Disease Activity Score (ASDAS). Radiographic axial damage was evaluated by the modified Stoke Ankylosing Spondylitis Spine Score (mSASSS), and the Bath Ankylosing Spondylitis Radiology Index-Hip (BASRI-h) was assessed for hip involvement. Lastly, the Bath Ankylosing Spondylitis Metrology Index (BASMI) was also collected.
Clinical, laboratory, and radiological findings were compared between different groups using parametric and non-parametric tests. The Spearman coefficient was calculated to evaluate the correlation between BMI and different variables.
Significance was set at a p-value ≤ 0.05.
Results: A total of 103 patients were enrolled in the study, of whom 57 (55.3%) were women, with no statistical differences between groups. Among all patients, 31.1% were obese, 37.8% were overweight, and 31.1% had a normal weight.
Obese patients were older compared to the other two groups (mean age 54.8 ± 13.9 vs 48.6 ± 13.9 years; p=0.006), had a higher BASMI index (p < 0.001), and had a more frequent bilateral radiographic hip involvement (p < 0.001). Finally, obese patients more frequently used "on-demand" non-steroidal anti-inflammatory drugs (NSAIDs) (p = 0.005).
We found a fair correlation between BMI and BASMI scores ($\rho = 0.32$; $p = 0.001$) and between BMI and C-reactive protein levels ($\rho = 0.30$; $p = 0.003$).
On multivariate analysis, the need for NSAIDs and the presence of bilateral hip involvement were independently associated with obesity in axSpA [NSAIDs OR 4.8 (CI 1.6-14.7); bilateral hip involvement OR 8.9 (CI 2.9-27.1)].
Conclusion: These findings underscore the importance of considering obesity as a significant comorbidity in axSpA. The more frequent need for analgesic treatments, impaired spinal mobility, and increased hip involvement observed in obese patients highlight the need for tailored management strategies for this population.

119 - LYMPHOPENIA FLUCTUATION PATTERNS DETERMINE THE TRAJECTORY OF THE DISEASE IN SJOGREN'S PATIENTS WITH HEMATOLOGICAL INVOLVEMENT

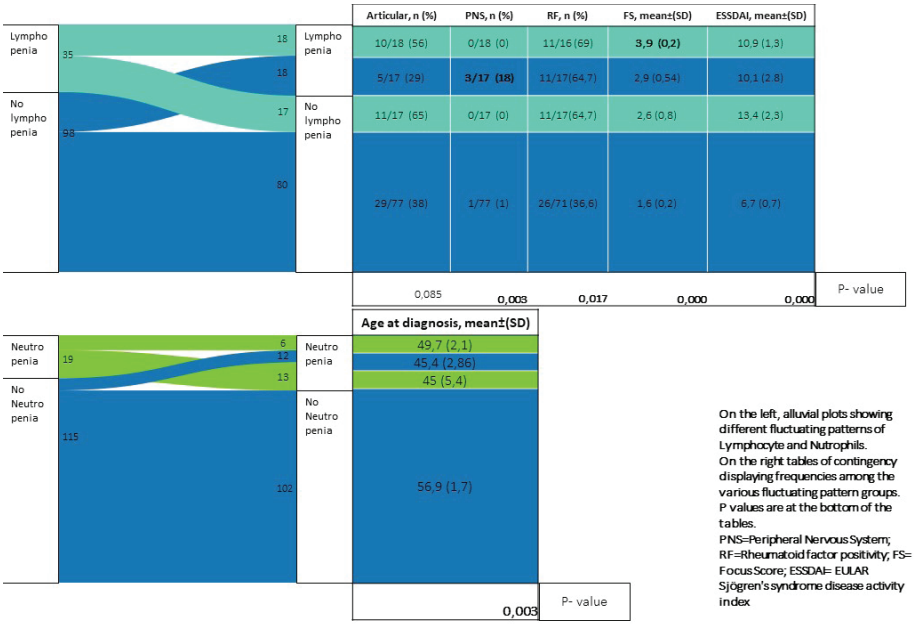
G. Fulvio[1], Matilde Bandeira[2], Augusto Silva[2], Manuel Silvério-António[2], G. La Rocca[1], S. Fonzetti[1], Nikita Khmelinskii[2], F. Ferro[1], M. Mosca[1], C. Baldini[1], Vasco C Romão[2]
[1]Rheumatology Unit, University of Pisa, Firenze, Italy, [2]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background/Purpose: Sjogren's disease (SjD) is characterized by a moderate prevalence of cytopenia, mainly neutropenia and lymphocytopenia with a typical fluctuating pattern. The associations between clinical manifestations and cumulative hematological involvement are well described. However, the prognostic value of white blood cell counts over time is still not fully understood. Our aim was to determine the prevalence of neutropenia and lymphopenia in patients with SjD, their variations over time and the corresponding clinical associations.
Methods: This is a multicentric study conducted at the University of Pisa and Lisbon Academic Medical Centre. Data of SjD patients (ACR/EULAR 2016 criteria) with hematological involvement were retrospectively collected. Patients were divided into groups accordingly to the presence of lymphopenia and neutropenia throughout their disease course: no cytopenia, cytopenia only present at baseline, cytopenia only present at the last follow up and persistent cytopenia. Demographic and cumulative clinical features, such as serological markers, organ involvement (assessed by ESSDAI), and salivary gland biopsy findings were analyzed. Univariate and multivariate logistic regression analysis was performed.
Results: We included 143 patients, 92.3 % were female and the mean follow up was 8.46±0.76. Symptomatic treatment, hydroxychloroquine or short course glucocorticoids were the most common therapies in our pa-

TO 119 – TABLE.

	All pts n=143	Never Lymphopenia N=75	Lymphopenia N=61	p-value	Never neutropenia n= 98	Neutropenia n=35	p-value
Age at diagnosis, mean±(SD)	50,57 (1,26)	53,1 (1,6)	48,3 (1,9)	0,06	53,4 (1,4)	45,3 (2,5)	**0,00**
Age last visit, mean±(SD)	59,12 (1,27)	60 (1,7)	58 (2)	0,45	60,7 (1,4)	55,1 (2,8)	0,06
Follow up, mean±(SD)	8,46 (0,76)	6,9 (0,7)	9,7 (1,1)	**0,03**	7,3 (0,7)	9,8 (1,4)	0,09
Gender, n (%)	11/143 (7,7)	7\75 (9)	4\61 (7)	0,75	11\98 (11)	0\35 (0)	0,07
Focus Score, mean±(SD)	2,23 (0,23)	1,7 (0,2)	3 (0,5)	**0,01**	2,4 (0,3)	1,6 (0,3)	0,20
ANA, n (%)	133/136 (97,8)	73\75 (97)	60\61 (98)	1,00	96\98 (98)	34\35 (97)	1,00
SSA, n (%)	123/143 (86)	62\75 (83)	54\61 (89)	0,47	84\98 (86)	29\35 (83)	0,78
SBB, n (%)	61/143 (42,7)	23\75 (31)	32\61 (52)	**0,01**	37\98 (38)	15\35 (43)	0,69
Rheumatoid Factor, n (%)	66/131 (50,4)	23\66 (35)	39\58 (67)	**0,00**	48\90 (53)	11\31 (35)	0,10
Low C3, n (%)	36/133 (27,1)	15\69 (22)	21\58 (36)	0,08	26\93 (28)	9\32 (28)	1,00
Low C4, n (%)	21/132 (15,9)	7\68 (10)	14\58 (24)	0,05	17\93 (18)	3\31 (10)	0,40
Cryoglobulinemia, n (%)	5/83 (6)	0\45 (0)	5\35 (14)	**0,01**	5\63 (8)	0\16 (0)	0,58
ESSDAI, mean±(SD)	8,27 (7,57)	6,6 (0,7)	10,8 (1,2)	**0,00**	9,4 (0,8)	6,6 (0,9)	0,07
Constitutional domain, n (%)	23/139 (16,6)	11\72 (15)	11\60 (18)	0,65	18\96 (19)	4\33 (12)	0,59
Lhymphadenopaty domain, n (%)	32/139 (23)	15\72 (21)	16\60 (27)	0,54	26\96 (27)	5\33 (15)	0,24
Glandular domain, n (%)	25/139 (18)	9\72 (13)	14\60 (23)	0,11	17\96 (18)	6\33 (18)	1,00
Articular domain, n (%)	57/139(41)	25\72 (35)	32\60 (53)	**0,04**	43\96 (45)	12\33 (36)	0,42
Cutaneous domain, n (%)	20/139 (14,4)	7\72 (10)	12\60 (20)	0,13	16\96 (17)	3\33 (9)	0,40
Pulmonary domain, n (%)	14/139 (10,1)	8\72 (11)	6\60 (10)	1,00	13\96 (14)	1\33 (3)	0,11
Renal domain, n (%)	5/139 (3,6)	1\72 (1)	4\60 (7)	0,18	5\96 (5)	0\33 (0)	0,33
Muscular domain, n (%)	3/139 (2,1)	1\72 (1)	2\60 (3)	0,59	3\96 (3)	0\33 (0)	0,57
PNS domain, n (%)	4/139 (2,9)	0\72 (0)	4\60 (7)	**0,04**	3\96 (3)	1\33 (3)	1,00
CNS domain, n (%)	1/139 (0,7)	1\72 (1)	0\60 (0)	1,00	1\96 (1)	0\33 (0)	1,00
Biological domain, n (%)	81/139 (58,3)	38\72 (53)	41\60 (68)	0,08	57\96 (59)	21\33 (64)	0,84
Hematological Neoplasia, n (%)	7/132 (5,3)	1\72 (1)	6\60 (10)	**0,05**	7\96 (7)	0\33 (0)	0,19
LAB TEST AT BASELINE							
Hemoglobin, mean±(SD)	12,52 (0,12)	12,6 (0,2)	12,5 (0,2)	0,77	12,5 (0,2)	12,6 (0,2)	0,65
Platelets, mean±(SD)	231314,81 (8353)	242467,7 (12930,1)	216282,6 (8682,2)	0,12	238797,6 (10046,7)	205125 (12155,3)	0,09
Monocytes, mean±(SD)	414,91 (29,20)	505,7 (42,9)	289,4 (23,7)	**0,00**	428,4 (34,6)	367,8 (50,9)	0,39
VES, mean±(SD)	38,01 (3)	38,2 (4)	37,8 (4,6)	0,96	39,8 (3,6)	31,1 (5,6)	0,23
IgG levels, mean±(SD)	1602,16 (71)	1576,4 (87,9)	1634,9 (118,6)	0,69	1583,5 (76,7)	1700,1 (196,7)	0,55
IgM levels, mean±(SD)	123,03 (13)	99,3 (9,8)	151 (27,5)	0,06	125,7 (16,6)	110,9 (18,9)	0,69
IgA levels, mean±(SD)	261,13 (19)	272,9 (26,3)	247,3 (27,7)	0,51	265,3 (19,5)	242,2 (59,3)	0,64
Free light chain K, mean±(SD)	659 (84,44)	676,2 (145,7)	646,1 (105,2)	0,87	701,2 (100)	479,5 (98,1)	0,31
Free light chain L, mean±(SD)	336,43 (46)	329,9 (76,2)	341,3 (59,8)	0,91	367,9 (53,5)	202,5 (43)	0,16
Ratio K/L, mean±(SD)	2,08 (0,09)	2,1 (0,2)	1,9 (0,1)	0,38	1,9 (0,1)	2,4 (0,2)	**0,05**
B2 microglobulinemia, mean±(SD)	2437 (111)	2448,8 (188,3)	2431,9 (142,8)	0,95	2504,4 (135,2)	2236,7 (179,4)	0,31
LDH, mean±(SD)	222,84 (9,7)	210,6 (10,6)	237 (16,9)	0,18	221,6 (10,5)	230 (26,8)	0,76
C-Reactive Protein, mean±(SD)	0,61 (0,16)	0,7 (0,2)	0,5 (0,2)	0,55	0,7 (0,2)	0,5 (0,3)	0,62
C3 levels, mean±(SD)	102,7 (2,5)	103,5 (4,2)	101,9 (2,8)	0,75	105,3 (2,8)	90,3 (6)	**0,03**
C4 levels, mean±(SD)	18,3 (0,8)	18,8 (1)	17,8 (1,3)	0,55	18,4 (0,9)	18,2 (1,5)	0,95

Table1
In the upper part of the tabel cumulative manifestations of Sjogren's Syndrome, in the lower part laboratory test collected at baseline. PNS= peripheral nervous system; CNS=central nervous system;ESSDAI= EULAR Sjögren's syndrome disease activity index



TO 119 – Figure 1. On the left Alluvial plots showing different fluctuating patterns, on the right tables of contingency

tients. During the disease course, lymphopenia and neutropenia were present in 61/136 pts (44.9%) and 32/133 (26.31%), respectively. Lymphopenia was associated with a longer follow-up, serological features (SSB positivity, RF positivity and cryoglobulinemia), higher focus score, higher disease activity as assessed by ESSDAI, more common involvement of the peripheral nervous system, articular and muscular systems, and a higher risk of lymphoma development. Neutropenia was associated with a younger age at diagnosis and lower C3 levels at baseline. A trend was observed for sex (0/35 vs 11/98, p=0.07), as male patients did not have neutropenia (Table 1). In multivariate analysis, lymphopenia was independently associated with focus score (OR 1.312 C.I. 1.02-1.68, p= 0.034), whereas neutropenia remained associated with both age and C3 (OR 0.95, C.I 0.92-0.99, p=0,014; OR 0.95, C.I. 0.92-0.99, respectively). Patients presented different features depending on variations of blood cell levels: persistent lymphopenia tended to be associated with higher focus score, whereas the onset of lymphopenia during the disease course was associated with peripheral nervous involvement (p=0.003) Variations in neutropenia were not associated with any specific SjD clinical manifestations (Fig.1).

Conclusions: Our study highlights the associations of lymphopenia with key histological, serological and clinical features of SjD. Interestingly, lymphocytopenia may show different pattern of variations, ultimately associated with higher disease activity and tissue infiltration. This is consistent with the hypothesis of glandular tissue homing of circulating lymphocytes in the pathogenesis of SjD and should be further explored. We did not specifically investigate the impact of treatments on lymphopenia and neutropenia and future studies should address this point.

124 - DESCRIPTION OF NAILFOLD CAPILLAROSCOPY CHANGES IN PATIENTS WITH SYSTEMIC SCLEROSIS AT DIAGNOSIS

Ana Martins[1, 2], Daniela Oliveira[1, 2], Raquel Miriam Ferreira[1,2], Georgina Terroso[1], Miguel Bernardes[1, 2], Sofia Pimenta[1, 2], Lúcia Costa[1]

[1]Serviço de Reumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal

Background: Capillaroscopy is a simple and non-invasive method that allows direct observation of the microcirculation of the nail bed. This exam allows the evaluation of the number, shape and architecture of the nail bed capillaries. Capillaroscopy, a pivotal exam in patients with Raynaud's phenomenon, was considered as part of the EULAR/ACR criteria for systemic sclerosis (SSc). Several microvascular abnormalities were described in patients with SSc, which led to the identification of scleroderma patterns that reflect the different stages of SSc microangiopathy. Capillaroscopy may also give useful information about prognosis in patients with SSc since microvascular abnormalities seem to be correlated with disease severity.

Objectives: To describe the nailfold capillaroscopy changes in patients with systemic sclerosis at diagnosis

Methods: A monocentric retrospective cohort study of patients with SSc (according to 2013 ACR/EULAR criteria), followed in a tertiary hospital was conducted. Only patients with a capillaroscopy performed in the first 6 months after diagnosis were included. Demographic data was collected from Rheumatic Diseases Portuguese Registry (Reuma.pt) and medical records. Capillaroscopy was performed by the same physician and the second to fifth fingers were examined bilaterally in each patient. Features from the capillaroscopy, namely capillary organization, capillary density, avascular areas, presence of abnormal capillary shapes, giant capillaries, and hemorrhages were collected. Capillaroscopy patterns were described as early, active, late or non-scleroderma pattern, according to the EULAR Study Group on Microcirculation in Rheumatic Diseases standardized capillaroscopy evaluation chart (Smith et al. 2020).

Results: Eighty-six patients with SSc were included. The majority of these patients were female (n=71, 82.6%), with a mean age of onset of 46.4 ± 15.5 years and a mean age of diagnosis of 52.6 ± 15.9 years. Seventy-four (86.1%) patients had a limited cutaneous disease, 5 (5.8%) had diffuse cutaneous disease and 7 (8.1%) had SSc sine scleroderma.

Seventy-nine (91.9%) patients had a scleroderma pattern: 29 (33.7%) early pattern, 37 (43.0%) active pattern and 13 (15.1%) late pattern. Seven (8.1%) patients had nonspecific alterations. Fourteen (16.3%) patients had avascular areas and 16 (18.6%) had abnormal capillary shapes. Giant capillaries were present in 69 (80.2%) patients with a mean number of involved fingers of 3.8 ± 2.8. Hemorrhages were present in 62 (72.1%) patients with a mean number of involved fingers of 2.6 ± 2.4.

The majority of patients without skin involvement had a scleroderma pattern (6 of 7 patients, 85.7%). Also, the majority of patients without specific autoantibodies had a scleroderma pattern (12 out of 14 patients, 85.7%).

Conclusion: A significant percentage of patients had a late scleroderma pattern at onset, which can be related to a higher destruction of microcirculation in the early stage of the disease in some patients or to a longer disease duration prior to the diagnosis in others.

Only a small percentage of patients had a non-scleroderma pattern at diagnosis which reinforces the importance of capillaroscopy as a diagnostic tool in patients with SSc, especially if some specific manifestations, namely skin involvement and specific autoantibodies, are absent.

125 - SYSTEMIC SCLEROSIS AND PULMONARY HYPERTENSION – A SINGLE-CENTER CASE SERIES

Marcelo Neto[1], Fernando Albuquerque[1], André Saraiva[1], Ana Isabel Maduro[1], Tânia Santiago[1, 2], Maria João Salvador[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal

Background: Pulmonary hypertension (PH) is a major cause of mortality in systemic sclerosis (SSc). The recognition and appropriate clinical management of PH can significantly improve the prognosis of affected patients. As screening protocols emerge, SSc-specific follow-up and treatment are unmet needs.
Methods: Retrospective study of all available records of patients fulfilling the ACR/EULAR 2013 Classification Criteria for SSc, followed in our clinic between January 1998 and May 2023, with a diagnosis of PH according to the 2022 ESC/ERS criteria, regardless of PH type. Analyzed variables included sex, age at SSc and PH diagnosis, SSc subset, positivity for specific SSc antibodies, cumulative organ involvement, chest CT-scan and lung function test data at SSc and PH diagnosis, echocardiography data at SSc and PH diagnosis, serum BNP, NT-proBNP and urate at SSc and PH diagnosis, right heart catheterization data at PH diagnosis, and the occurrence of death (irrespective of cause).
Results: Of the 170 screened records of SSc patients, 11 were found to have a diagnosis PH based on right heart catheterization. Ninety-one percent were females with a mean ± SD age at SSc and PH diagnosis of 60.0 ± 11.7 and 64.5 ± 10.5 years, respectively, and a median follow-up time of 8.4 (IQR 4.6-11.1) years. SSc subtype and autoimmunity profile are summarized in Table 1.
Most patients had a limited SSc subtype (n=7) and anti-centromere antibodies (n=7). All patients had pre-capillary PH, with one exception which had combined PH. Most patients (n=7) were classified with clinical type 1 (pulmonary arterial hypertension), 3 had PH associated with lung disease and one had multifactorial PH (heart + lung disease). Five patients (45,5%) had deceased at the time of data collection.
Patients with an estimated pulmonary artery systolic pressure (ePASP) >60 mmHg on echocardiogram at PH diagnosis had higher serum urate concentration at that time (8.55 vs. 5.03 mg/dL, p=0.004). No other associations were found between the studied variables. Sensitivity or multivariate analysis was not performed owing to the low number of cases.
Conclusion: Our cohort of SSc-associated PH was composed of mostly female patients with limited cutaneous SSc subtype and anticentromere antibodies, corroborating data existent in the literature. As expected, most patients had type 1, pre-capillary PH. With the caveat of a small sample and no sensitivity analysis, we found that patients with a very high ePASP had higher urate levels at PH diagnosis. Larger nationwide studies are needed to better understand the clinical course of SSc-associated PH.

TO 125 – TABLE 1. Demographic and clinic characteristics of included patients.

Female gender, N (%)	10 (90,9)
Age at SSc diagnosis, mean ± SD (years)	60,0±11,7
Age at PH diagnosis, mean ±SD (years)	64,5±10,5
Follow-up duration, median (IQR) (years)	8,4 (4,6-11,1)
SSc subtype	
Limited, N (%)	7 (63,6)
Diffuse, N (%)	2 (18,2)
Sine scleroderma, N (%)	2 (18,2)
Autoimmunity profile	
ANA positivity, N (%)	11 (100)
anti-centromere, N (%)	7 (63,6)
anti Scl-70, N (%)	2 (18,2)
anti-RNA polymerase III, N (%)	1 (9,1)
non-specific, N (%)	1 (9,1)
PH – hemodynamic classification	
Pre-capillary, N (%)	10 (90,9)
Combined post- and pre-capillary, N (%)	1 (9,1)
PH – clinical classification	3 (42,9)
Type 1, N (%)	7 (63,6)
Type 3, N (%)	3 (27,3)
Type 5, N (%)	1 (9,1)
Occurrence of death	5 (45,5)
Time from SSc diagnosis to death, mean ± SD (years)	9,4±5,0
Time from PH diagnosis to death, mean ± SD (years)	5,4±3,5

SSc – systemic sclerosis; PH – pulmonary hypertension, SD – standard deviation; IQR – interquartile range

REFERENCES:

Vonk, M.C., E. Vandecasteele, and A.P. van Dijk, Pulmonary hypertension in connective tissue diseases, new evidence and challenges. Eur J Clin Invest, 2021. 51(4): p. e13453.
Humbert, M., et al., 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J, 2022. 43(38): p. 3618-3731.

132 - LEFLUNOMIDE IS A SAFE AND EFFECTIVE DRUG IN PATIENTS WITH SJÖGREN'S SYNDROME WITH ACTIVE SYSTEMIC DISEASE

Elisa Fiorentini[1], Matilde Bandeira[2, 3], Manuel Silvério-António[2, 3], Arianna Damiani[1], Nikita Khmelinskii[2, 3], Vasco C Romão[2, 3]

[1]Department of Experimental and Clinical Medicine, University of Florence, and Division of Rheumatology AOUC, Firenze, Italy, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: To date, there are no approved treatments for Sjögren's syndrome (SS). Leflunomide (LEF) was recently shown to be promising for the treatment of systemic manifestations of SS (1). It has a pleiotropic immunosuppressive effect, suppressing T and B-cell ex-

TO 132 – TABLE 1. Clinical and demographic features of Sjögren's Syndrome patients treated with leflunomide.

	n=14		
Age, years (mean ± sd)	49.2 ± 14.7		
Female (%)	14 (100)		
Disease duration, years (mean ± sd)	5.6 ± 5.3		
Xerostomia, n (%)	13 (92.9)		
Positive salivary gland biopsy, n (%)	9 (64.3)		
Xeropthalmia, n (%)	13 (92.9)		
Positive Schirmer's test, n (%)	5 (35.7)		
ANA+, n (%)	14 (100)		
Anti-Ro/SSA+, n (%)	14 (100)		
Anti-LA/SSB+, n (%)	8 (57.1)		
Rheumatoid Factor+, n (%)	12 (85.7)		
Main Indication for LEF start, n (%) - Glandular activity - Articular activity - Skin disease - Constitutional symptoms - Biological activity	 5 (35.7) 5 (35.7) 2 (14.3) 1 (7.1) 1 (7.1)		
LEF dose at BL / FU, n (%) - 10mg/d - 15mg/d - 20mg/d	 13 (92.9) / 5 (35.7) 1 (7.1) / 3 (21.4) 0 (0) / 6 (42.9)		
Concomitant therapy, n (%) - Hydroxychloroquine - Prednisolone - Pilocarpine	 8 (57.1) 4 (28.6) 9 (64.3)		
	Baseline	FU	p-value
ESSDAI (mean ± sd)	6.21 ± 4.41	4.07 ± 5.24	0.059
ESSPRI (mean ± sd)	6.28 ± 1.86	5.78 ± 2.61	0.210
ESSPRI dryness (mean ± sd)	5.92 ± 2.75	5.75 ± 3.19	0.914
ESSPRI pain (mean ± sd)	6.62 ± 2.29	5.42 ± 3.06	0.172
ESSPRI fatigue (mean ± sd)	7.15 ± 2.12	5.67 ± 2.84	0.172
ESS (mean ± sd)	5.97 ± 2.54	5.30 ± 3.04	0.305
ESS oral dryness (mean ± sd)	5.92 ± 2.69	5.45 ± 3.42	0.307
ESS ocular dryness (mean ± sd)	6.15 ± 2.79	5.00 ± 2.61	0.104
IgG mg/dl (mean ± sd)	1971.31 ± 611.67	1613.60 ± 651.26	0.041
C3	110.58 ± 20.16	106.30 ± 15.99	0.734
C4	19.92 ± 10.28	21.10 ± 8.37	0.154

pansion, antibody production, Th1 differentiation, and proinflammatory cytokines (TNF, IL-1β) production (2). This evidence led us to start using LEF as an off-label treatment for patients with SS.

Methods: We included patients fulfilling ACR/EULAR 2016 classification criteria for SS who started LEF up to January 2023. We collected demographic and clinical data at the start of LEF and at the last follow-up visit, including disease activity assessed by the EULAR SS Disease Activity Index (ESSDAI), patient symptoms (EULAR SS Patient Reported Index [ESSPRI], EULAR sicca score [ESS]), complement levels and serum IgG. We assessed safety, tolerability, and persistence of LEF. We compared baseline and last follow-up data using Paired T-test. P-value was considered significant at <0.05.

Results: We included 14 female patients with SS who started LEF between September 2020 and January 2023, with a mean age of 49.2±14.7 years and a mean disease duration of 5.6±5.3 years (Table 1). Most of them had xerostomia and xeropthalmia, all were positive for ANA and anti-Ro and around two-thirds had a positive salivary gland biopsy. The starting dose of LEF was 10 and 15mg/day in 92.9 % and 7.1% of patients, respectively. All patients started LEF due to systemic disease activity, with a mean ESSDAI of 6.2±4.4. More than half of the patients (n=8, 57.1%) had moderate-to-severe active disease (ESSDAI ≥5). Glandular and articular activity were the main indication for starting LEF in 35.7% of cases each, followed by skin disease (14.3%), constitutional symptoms (7.1%) and biological activity (7.1%). More than half of the patients were treated with hydroxychloroquine (57.1%) or prednisolone (64.3%), no one was on concomitant methotrexate. Mean follow-up was 12.6±8.3 months. The dose of LEF at the last follow-up visit, was 20, 15 and 10mg/day in 42.9%, 21.4% and 35.7%, respectively. Four patients stopped LEF after a mean of 7±4.24 months: two patients due to adverse events (alopecia, promptly resolved with wash-out procedure; gastrointestinal toxicity), one patient due to persistent disease activity and subsequent enrollment in a clinical trial, and the other one due to pregnancy desire. In terms of efficacy, there was a mean reduction of ESSDAI of 2.1±4.0, with 64.3% of patients achieving an ESSDAI≤3 (Fig. 1). Patient symptoms also improved, with a mean reduction of ESSPRI and ocular dryness visual analog scale of roughly 10% (0.9±2.6 and 1.1±2.0, respectively). Of note, there was a significant decrease in IgG levels, with a mean reduction of 409.5±543.2 mg/dL (p=0.04) and 80% of patients presenting an IgG<2000mg/dL at follow-up.

Conclusions: In our experience, LEF has been an effective, safe, and well-tolerated treatment for patients with SS and active systemic disease. It is particularly effective at improving markers of B cell hyperactivity, such as serum IgG levels, which could be due to a direct suppressive effect on B cell activity, as previously reported (3). These data are encouraging and suggest LEF may become an important drug in the future management of patients with SS.

REFERENCES:

1. van der Heijden, E. H. M. et al. Lancet Rheumatol 2, e260–e269 (2020)
2. Bikker, A. et al. Ann Rheum Dis 71, 1934–1941 (2012)
3. van Woerkom, J. M. et al. Ann Rheum Dis 66, 1026–1032 (2007)

134 - PREDICTORS OF ATYPICAL FRACTURES: A RETROSPECTIVE ANALYSIS OF A FRACTURE LIAISON SERVICE

Filipe Cunha Santos[1], Ana Sofia Pinto[1], Sara Paiva Dinis [1], Duarte Augusto[1], Nathalie Madeira[1], Cláudia Vaz[1, 2], Joana Fonseca Ferreira[1, 2]

[1]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [2]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal

Background: Atypical femoral fractures (AFFs) are an uncommon but potentially serious complication associated with the long-term use of bisphosphonate (BP) medication for osteoporosis. The incidence of AFFs has increased in recent years, prompting investigation into the underlying risk factors and management strategies for this type of fracture.

Objectives: To evaluate predictors of AFF among hip fracture patients.

Material and methods: This is a retrospective case-control study, including patients over 50 years, after operation for osteoporotic hip fracture and followed at our Fracture Liaison Service between September 2019 and January 2023. We classified fracture radiographs and compared demographic, clinical, biochemical features and BP purchase data between patients with AFF and those with typical osteoporotic hip fracture (controls). Normally distributed quantitative variables were compared using the independent samples Student's t-test and qualitative variables were compared using chi-square test or Fisher's Exact test, as appropriate. Logistic regression was performed to assess predictors of atypical fracture. p values were considered statistically significant if p<0.05.

Results: Of 109 patients, 9 (8.3%) had AFF. Patients with AFF were younger than those with trochanteric fractures (71.0±8.8 vs. 78.3±8.6 years, p=0.008), had a higher body mass index (BMI) (28.5±5.2 vs. 24.8±4.5kg/m2, p=0.023) and estimated glomerular

TO 134 – TABLE 1. Baseline characteristics of included patients according to site and type of fracture.

	Typical osteoporotic fracture (*n*=100)	Atypical femoral fractures (*n*=9)	*p* value
Age (years), mean±SD	78.3±8.6	71.0±8.8	0.017
Sex, female *n* (%)	84 (84.0%)	7 (77.8%)	0.457
BMI (kg/m²), mean±SD	24.8±4.5	28.5±5.2	0.023
eGFR (mg/dL), mean±SD	70.9±21.7	88.7±19.3	0.027
Hb (gr/dL), mean±SD	12.7±1.9	12.8±0.9	0.800
Charlson's Comorbidity Index, mean±SD	4.4±1.7	3.6±1.2	0.070
Any treatment with BP, *n* (%)	14 (14.0%)	7 (77.8%)	<0.001
Duration of BP treatment (years), mean±SD	4.3±2.9	6.4±3.3	0.157
PPIs, *n* (%)	54 (54.0%)	7 (77.8%)	0.292
Corticosteroids, *n* (%)	6 (6.0%)	2 (22.2%)	0.154
Anti-depressants, *n* (%)	24 (24.0%)	0 (0.0%)	0.201
Statins, *n* (%)	47 (47.0%)	3 (33.3%)	0.505

BMI Body mass index, *BP* bisphosphonate, *eGFR* estimated glomerular filtration rate, *Hb* Hemoglobin, *PPI* proton pump inhibitors

filtration rate (eGFR) (88.7±19.3 vs. 70.9±21.7mg/dL, p=0.027), and had a higher proportion of any treatment with BP (77.8% vs. 14.0%, p<0.001). These were tested in multivariate logistic regression: X2 (4) 29.2, p<0.001; R2=0.604. Previous treatment with BP and BMI were predictors of atypical fracture (p=0.006 and p=0.017, respectively). Odds ratio (OR) for previous treatment with BP was 58.2 (95%CI 3.1-1086.1) and 1.4 (95%CI 1.1-1.8) for BMI.

Conclusions: In addition to what has already been reported, that previous use of bisphosphonates is related to the appearance of atypical fractures, this work concludes that an increased body mass index appears to also be a predictor of atypical fractures.

REFERENCES

Starr, Jessica et al. "Current Understanding of Epidemiology, Pathophysiology, and Management of Atypical Femur Fractures." Current osteoporosis reports vol. 16,4 (2018): 519-529. doi:10.1007/s11914-018-0464-6

138 - CAN MICROVASCULAR DAMAGE SEVERITY BE ASSOCIATED WITH GLOBAL DISEASE SEVERITY IN PATIENTS WITH SYSTEMIC SCLEROSIS?

Ana Martins[1, 2], Daniela Oliveira[1, 2], Raquel Miriam Ferreira[1, 2], Georgina Terroso[1], Miguel Bernardes[1, 2], Sofia Pimenta[1, 2], Lúcia Costa[1]

[1]Serviço de Reumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal

Background: Systemic sclerosis (SSc) is a heterogeneous disease with variable manifestations and clinical outcomes. Some patients may progress to a severe disease with fatal com¬plications after several months or may remain limited to sclerodactyly and Raynaud's phenomenon. Therefore, considerable efforts are being made to investigate possible biomarkers of severe disease. Nailfold videocapillaroscopy (NVC) is a simple and non-invasive method that allows direct observation of the microcirculation. Some studies support that the degree of microcirculation damage of nailfold capillaries may be associated with the severity of visceral involvement in SSc patients.

Objectives: To analyze if there is an association between the severity of microvascular damage, visceral involvement and global disease activity in SSc patients.

Methods: A monocentric retrospective cohort study of patients with SSc diagnosis (according to 2013 ACR/EULAR criteria) that have a NVC performed within the first 6 months after diagnosis was conducted. Demographic data, visceral involvement in the first 3 years of the disease and NVC findings were collected from the Rheumatic Diseases Portuguese Registry and medical records. NVC was evaluated according to the EULAR Study Group on Microcirculation in Rheumatic Diseases standardized capillaroscopy evaluation chart (Smith et al. 2020). The severity of microvascular damage was

classified into 4 categories, according to the worsening of the NVC patterns (0=non-scleroderma, 1=early, 2=active and 3=late). The severity of organ involvement was accessed by the disease severity scale (DSS) of Medsger for each organ (if the organ category was superior or equal to 2, this organ involvement was classified as severe). As a global measure of disease severity, the sum of the Medsger DSS was used. Pearson correlations and logistic regression analysis were performed.

Results: A total of 86 patients (71 females, 82.6%, mean age of onset of 52.6±15.9 years) with SSc were included. Seventy-four (86.1%) patients had a limited cutaneous disease, 5 (5.8%) diffuse cutaneous disease and 7 (8.1%) SSc sine scleroderma.

A moderate correlation was found between the severity of microvascular damage and the global measure of disease severity (r=0.55, p<0.001). Also, a moderate correlation was found between the severity of microvascular damage and the severity of peripheral vascular involvement (r=0.43, p<0.001) and skin involvement (r=0.339, p=0.001).

Avascular areas in NVC seem to predict the presence of digital ulcers (OR 6.75, 95% CI 1.72-26.45, p=0.006), esophageal involvement (OR 3.43, 95%CI 1.10-11.57, p=0.039), muscular involvement (OR 11.67, 95%CI 1.06-138.94, p=0.04) and calcinosis (OR 22.67, 95%CI 4.76- 107.90, p<0.001). Abnormal capillary shapes seem to predict the presence of digital ulcers (OR 3.48 95%CI 1.12-10.82, p=0.025), muscular involvement (OR 29.82, 95%CI 9.14-97.32 p<0.001), severe skin involvement (OR 5.33, 95%CI 1.17-24.31, p=0.031) and calcinosis (OR 18.85, 95%CI 3.68-77.17, p<0.001).

Conclusion: In our study, the disease severity in SSc is associated with the worsening of NVC pattern. Also, the severity of peripheral vascular involvement and extension of skin involvement was associated with worsening of NVC pattern. Avascular areas and abnormal shapes in NVC seem to predict the presence of digital ulcers, muscular involvement and calcinosis.

This study highlights the importance of NVC as a prognostic tool, as patients with late scleroderma pattern seem to have a higher risk of severe disease.

140 - MUSCULOSKELETAL MANIFESTATIONS OF SARS-COV-2 IN PEDIATRIC POPULATION - A PORTUGUESE MULTICENTRIC STUDY

Augusto Silva[1, 2], Bianca Paulo Correia[1, 2], Helena Sofia Sousa[3], Joana Silva-Dinis[4], Mariana Silva[5], Vanessa Fraga[6], Ana Filipa Mourão[5], Sandra Sousa[6], Filipa Oliveira Ramos [1, 2, 7], Miguel Bernardo[7, 8], Patrícia Costa Reis[7, 8], Raquel Campanilho-Marques[1, 2, 7]

[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Pediatria, Hospital de Vila Franca de Xira, Vila Franca de Xira, Portugal, [4]Unidade de Reumatologia, Hospital de Santo António dos Capuchos - Centro Hospitalar Universitário de Lisboa Central, Lisboa, Portugal, [5]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [6]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [7]Unidade de Reumatologia Pediátrica, Centro Hospitalar Universitário Lisboa Norte, Hospital de Santa Maria, Lisboa, Portugal, [8]Departamento de Pediatria, Hospital de Santa Maria, Centro Hospitalar Universitário Lisboa Norte, Lisboa, Portugal

Introduction: SARS-CoV-2 infections can cause severe inflammation and serve as a trigger for immune-mediated manifestations. Several case reports in adults have described auto-antibodies production (45-57%) and musculoskeletal (MSK) manifestations after the infection (2.7-5.9%), but in children the information is scarce.

Objectives: To describe the MSK manifestations, after SARS-CoV-2 infection, in a multicentric pediatric population without previously known rheumatic disease.

Methods: The clinical records of all new patients, between April 2020 to March 2023, from five centers, with MSK manifestations and/or serologic findings related to rheumatic diseases (RD) after SARS-CoV-2 infection, were reviewed. Based on available data in adults, 60 days were defined as the longest period of time between exposure to the virus and the onset of MSK manifestations or serologic findings. Patients with previous known RD were excluded. Data on demographic variables and clinical features were collected and presented as frequencies and median [interquartile range] for categorical and continuous variables, respectively.

Results: During the study period 1063 new patients were observed, of which 36 (3.4%) had MSK manifestations and serologic findings related to RD after SARS-CoV-2 infection (table 1). Considering these 36 patients (56% female), the median age at infection was 15 [12-16] years and the median time for serological or MSK manifestations after SARS-CoV-2 infection was 23 [15-40] days. All patients had asymptomatic (54%) or mild infection, and none required hospitalization.

Following infection, the main MSK manifestations were arthralgia (23/36, 64%), fatigue (16/36, 44%), myalgia (16/36, 44%) and acrocyanosis (8/36, 22%). The most frequent diagnosis identified were nonspecific musculoskeletal pain (NMKP: 11/36, 31%), perniosis (8/36,

TO 140 – TABLE 1. Main characteristics of musculoskeletal manifestations and immune-mediated diseases after SARS-CoV-2 infection

	Connective tissue diseases N=3		Myositis N=4									
	Systemic lupus erythematosus N=2	Undifferentiated connective tissue disease N=1	Viral myositis N=2	Genetic muscle disorder N=1	Dermatomyositis N=1	Perniosis N=8	Nonspecific musculoskeletal pain N=11	Suspected sarcoidosis N=1	Juvenile idiopathic arthritis N=2	Reactive arthritis N=2	Idiopathic uveitis N=3	Chronic fatigue syndrome N=2
Age at diagnosis - value/range (years)	11-15	16	16-17	4	12	13-17	2-17	12	12-16	16-17	7-16	12-16
Female (%)	100%	100%	100%	0%	0%	37.5%	72.7%	0%	50%	0%	1 (33.3%)	100%
SARS-CoV-2 infection severity:												
- Asymptomatic n (%)	1 (50%)			1 (100%)	1 (100%)	5 (62.5%)	5 (45.5%)	1 (100%)		1 (50%)	1 (33.3%)	2 (100%)
- Mild n (%)	1 (50%)	1 (100%)	2 (100%)			3 (37.5%)	6 (54.5%)		2 (100%)	1 (50%)	2 (66.7%)	
Time between infection and first manifestation: value/range (days)	9-15	49	39-59	12	33	15-46	14-60	30	15-16	49-54	18-60	10-37
Need:												
- Hospitalization n (%)	2 (100%)	0	0	0	1 (100%)	0	0	0	0	0	0	0
- IVIG n (%)	0	0	0	0	0	0	0	0	0	0	0	0
- MP pulses n (%)	1 (50%)	0	0	0	0	0	0	0	0	0	0	0
- Oral PDN n (%)	2 (100%)	0	0	0	1 (100%)	0	0	0	1 (50%)	1 (50%)	2 (66%)	0
- Methotrexate n (%)	0	0	0	0	1 (100%)	0	0	0	1 (50%)	0	1 (33%)	0
- Azathioprine n (%)	1 (50%)	0	0	0	0	0	0	0	0	0	0	0
- Mycophenolate mofetil n (%)	1 (50%)	0	0	0	1 (100%)	0	0	0	0	0	0	0
- Hydroxychloroquine n (%)	2 (100%)	0	0	0	1 (100%)	0	0	0	0	0	0	0
- Topical corticosteroid n (%)	0	0	0	0	1 (100%)	3 (37.5%)	0	0	0	0	1 (33%)	0
- Oral anti-inflammatory n (%)	0	0	1 (50%)	0	0	2 (25%)	5 (45.5%)	0	1 (50%)	2 (100%)	1 (33.3%)	1 (50%)
- Nifedipine n (%)	0	0	0	0	0	1 (12.5%)	0	0	0	0	0	0
- Pentoxifylline n (%)	0	0	0	0	0	2 (25%)	0	0	0	0	0	0
- Muscle relaxant n (%)	0	0	0	0	0	0	6 (54.5%)	0	0	0	0	1 (50%)
Outcome of rheumatic diseases:												
- Controlled disease activity n (%)	1 (50%)	1 (100%)	1 (50%)	1 (100%)	1 (100%)	1 (12.5%)	9 (81.8%)	1 (100%)	1 (50%		1 (33.3%)	2 (100%)
			1 (50%)			7 (87.5%)	2 (18.2%)		1 (50%)	2 (100%)	2 (66.6%)	
- Complete remission n (%)	1 (50%)											
- Maintain disease activity n (%)												

Abbreviations: IVIG - intravenous immunoglobulin; MP - methylprednisolone; PDN - prednisolone; SARS-CoV-2 - severe acute respiratory syndrome coronavirus 2.

22%), myositis (4/36, 11%) and connective tissue diseases (CTD: 3/36, 8%). Two patients (0.2%) had positive serologic findings (one with ANCA-PR3 and one with lupus anticoagulant), but no clinical manifestations related to these markers.
Five patients (14%) were hospitalized due to the severity of the developed RD: two with systemic lupus erythematosus (6%) and three with myositis (8%).
After a median follow-up of 15 [7.5-18] months, nearly all patients presented favorable outcomes. Regarding the patients who fulfilled diagnostic criteria of an inflammatory RD: four (11.1%) patients had complete remission, eight (22.2%) had remained with minimal disease activity and one (2.7%) with active disease. From the non-inflammatory patients, 11 (30.6%) had complete remission.
Conclusion: To the best of our knowledge, this is one of the few studies analyzing the MSK involvement induced by SARS-CoV-2 in a pediatric population. In this cohort, the MSK manifestations were uncommon (3.4%), and the most frequent were NMKP, perniosis, myositis and CTD. The manifestations had a wide spectrum of severity, from mild to potentially fatal, but were early identified, treated and 41.6% reached complete remission.

143 - CANCER IN PATIENTS WITH SYSTEMIC SCLEROSIS – EXPERIENCE FROM A TERTIARY SINGLE CENTRE

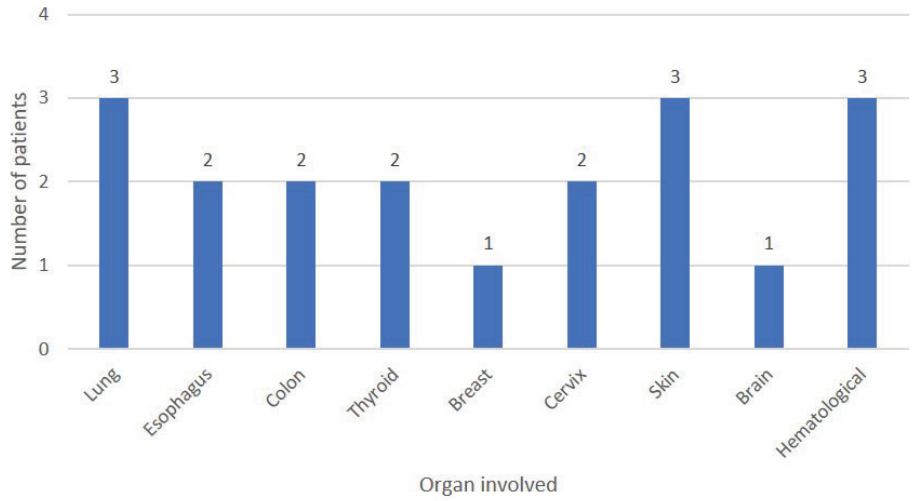
Ana Catarina Duarte[1], Catarina Abreu[2], Maria José Santos[1, 3], Ana Cordeiro[1]
[1]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [2]Reumatology Department, Hospital Garcia de Orta, EPE, Almada, Portugal, [3]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Cancer has been reported in 3-16% of systemic sclerosis (SSc) patients and includes hematological and solid organ malignancies. Several factors have been associated with a higher risk of cancer in SSc patients, such as male sex, exposure to tobacco, chemical products and cytotoxic drugs, diffuse cutaneous subtype and anti-RNA polymerase III antibody positivity. Interstitial lung disease (ILD) also seems to increase the risk of lung cancer.
We aim to characterize SSc patients who developed cancer after the diagnosis of SSc and compare them to the remaining SSc patients.
Methods: Retrospective analysis of SSc patients aged ≥ 18 years at initial diagnosis, fulfilling ACR/EULAR 2013 classification criteria, followed in our centre. Diagnosis of cancer was based on imaging and/or histopathological changes. Basal cell carcinoma was excluded. Only patients with at least 1-year interval between SSc and malignancy diagnosis were included.
Demographic data and previous exposures were collected. Data on SSc included date of diagnosis, clinical subtype, clinical/immunological manifestations and previous/current treatment. Date of cancer diagnosis and organ involved were recorded.
Data are presented as mean ± S.D. for normally distributed and median [IQR] for non-normally distributed continuous variables, and as number (percentage) for categorical variables. Groups were compared using qui-

TO 143 – TABLE 1. Comparison between patients with and without cancer

	With cancer (N=19)	Without cancer (N=128)	Percentage of missing data	p-value
Male	4 (21.1%)	12 (9.4%)	0%	0.131
Age (years)	69.1 ± 11.1	65.4 ± 14.6	6.8%	0.288
SSc duration (years)	13.3 [IQR 4.9-17.4]	12.4 [IQR 7.5-22.4]	6.8%	0.82
Ever smoking	2 (16.7%)	25 (26.6%)	32.9%	0.726
Occupational exposure risk	2 (13.3%)	11 (14.9%)	43.7%	1
Diffuse cutaneous subtype	6 (33.3%)	22 (17.5%)	8.9%	0.081
Anti-topoisomerase I antibody	8 (42.1%)	34 (27.4%)	2.7%	0.278
Anti-RNA polymerase III antibody	3 (15.8%)	2 (1.6%)	2.7%	0.006
Esophageal involvement	12 (63.2%)	71 (55.5%)	0%	0.624
ILD	9 (47.4%)	40 (31.2%)	0%	0.363
Previous treatment with immunosuppressants	9 (52.9%)	76 (65%)	8.8%	0.42
Previous treatment with CYC	2 (11.8%)	2 (1.7%)	8.8%	0.078

Legend: SSc- systemic sclerosis; ILD – interstitial lung disease; CYC – cyclophosphamide; IQR – interquartile range



TO 143 – Figure 1. Distribution of organ involvement of cancer

square test/Fisher's exact test and independent samples t test/Mann-Whitney test, as appropriate.

Results: From 147 SSc patients, 131 (89.1%) were female, with mean age of 65.9±14.2 years and median disease duration of 12.4 [IQR 7.5-20.4] years at last visit. Limited cutaneous subtype was the most frequent (n=102; 69.4%). Antinuclear antibodies were positive in 139 (94.6%) patients, with 75 (51%) presenting anticentromere antibody and 42 (28.6%) anti-topoisomerase I antibody. Near half of the patients (n=85; 53.8%) received immunosuppression.

We identified 19 (12.9%) patients with cancer, which occurred 6 [IQR 1.6-11.1] years after SSc diagnosis. The organs involved are showed in figure 1.

Comparison between patients with and without cancer is represented in table 1.

All patients with lung cancer were non-smokers, had positive anti-topoisomerase I antibodies and SSc-related ILD. Patients with esophagus cancer were former smokers and had gastroesophageal reflux disease; one had anti-RNA polymerase III antibody and the other no specific SSc antibodies.

Eight patients with cancer died, 5 of them related to cancer (3 lung, 2 esophagus).

Conclusion: In our cohort, haematological neoplasms, lung and skin cancer were the most frequently diagnosed. Anti-RNA polymerase III positivity was the only variable significantly associated with cancer, which is in line with published literature. With the development of more effective therapies, it is expected that mortality related to pulmonary arterial hypertension and ILD decreases, making other comorbidities, such as malignancies, increasing causes of death. In the future larger studies are needed to better characterizer SSc patients at higher risk of cancer development and those who will benefit from stricter screening strategies.

145 - RHEUMATOLOGY NURSING OUTPATIENT CLINIC IN PORTUGAL

Lurdes Barbosa[1], Joana Santos[1], Ana Cordeiro[1], Maria José Santos[1, 2], Elsa Pereira[1]

[1]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Background: Systemic inflammatory rheumatic diseases are characterized by being chronic diseases. These diseases affect the person, in their various dimensions and are often associated with pain, loss of function, fatigue, anxiety and depression. The complex needs of these patients and their families require specialized attention that can be provided in a nursing clinic. Awareness of the needs of rheumatic patients, combined with the lack of this type of care in Portugal, led us to develop a nurse-led outpatient rheumatology clinic.

Objectives: Characterize a guided nursing clinic with the aim to support patients with systemic inflammatory rheumatic diseases and their families.

Methods: Summarized description of first nurse guided rheumatology clinic in Portugal.

Results: The nurse-led outpatient clinic was created and developed during the pandemic period of SARS-CoV 2 (starting February 2020). Was developed with the aim to identify the needs of the patient with systemic inflammatory rheumatic diseases (SIRD) and their families, to improve the understanding of the disease and the management of their treatments. For the last three years a total of 793 appointments were performed. The targeted patients had SIRD, mainly rheumatoid arthritis, psoriatic arthritis and spondyloarthritis, treated with oral or subcutaneous (SC) classic / biologic / tar-

get synthetic DMARDs. Appointments are scheduled at the beginning of these therapeutics, 4 weeks later, and also whenever needed, at the request of the patient or the treating physician, in order to ensure a good nurse availability to clarify doubts related to these treatments and improve medication adherence. In the specific case of methotrexate (MTX), a protocol was developed by the Rheumatology multidisciplinary team, defining the parameters eligible for evaluation, and the respective procedures of care. In the last year a total of 139 appointments were performed in patients initiating MTX. During appointments nurses also assess specific disease implications in the person's daily life, both physical, emotional and social domains. Additionally, this is an opportunity to reinforce reliable information on disease management, coping strategies, including information how to manage medication side effects.

Conclusions: The nurse-led rheumatology outpatient clinic provides key additional care and monitorization to patients with SIRD, treated with oral and SC classic / biologic / target synthetic DMARDs. Nurses improve the management of chronic diseases, namely by helping the patients and their families to understand the disease and the treatment. Promoting autonomy of the patient in self-administration of SC therapies at home, counselling and informing about possible adverse effects and respective management strategies, constitute a valid contribution to disease control and patient engagement and education.

147 - A TWO-YEAR COMPARISON OF BACK PAIN AND MORNING STIFFNESS IN AXIAL SPONDYLOARTHRITIS AND NON-AXIAL SPONDYLOARTHRITIS CHRONIC BACK PAIN PATIENTS IN THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

Ana Bento da Silva[1, 2, 3], Miranda van Lunteren[3], Mary Lucy Marques[3, 4], Karen Minde Fagerli[5], Sofia Ramiro[3, 6], Désirée van der Heijde[3], F vanGaalen[3]

[1]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [2]Comprehensive Health Research Centre (CHRC), NOVA Medical School | Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa, Lisboa, Portugal, [3]Department of Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [4]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [5]Department of Rheumatology, Diakonhjemmet Hospital, Oslo, Norway, [6]Rheumatology Department, Zuyderland Medical Center, Heerlen, Netherlands

Background: Treatment of axial spondyloarthritis (axSpA) has been shown to improve symptoms of the disease such as back pain (BP) and morning stiffness (MS). As a result, early referral of patients suspected of having axSpA is strongly recommended. However, little data is available on the disease course of early axSpA in clinical practice, and particularly in comparison to referred patients who have chronic BP (CBP) but not axSpA. We set out to compare spinal symptoms at baseline and after 2 years (2y) in early axSpA and non-axSpA CBP patients in clinical practice.

Methods: The population consisted of adults (≥16 years) with CBP of unknown origin lasting more than 3 months and less than 2y, starting before 45 years, included in the SPondyloArthritis Caught Early (SPACE) cohort. Patients had a diagnosis of axSpA or non-axSpA at 2y with a high level of confidence by the treating rheumatologist (Marques ML, Ann Rheum Dis 2023;82:3-4). Patients reported the severity of BP (total and at night) and MS in the previous week on a numeric rating scale (NRS) ranging from 0 (no symptom) to 10 (unbearable symptom), both at baseline and 2y. For MS duration, a NRS ranging from 0 (0 hours) to 10 (≥2 hours) was used. For the assessment of each outcome, only patients with data at both timepoints were included. Wilcoxon signed-rank tests (for not normally distributed data) were used to compare baseline and 2y results within groups. For the comparison between groups, linear regression models were built, adjusting for the baseline value of the respective outcome, gender, age and use of nonsteroidal anti-inflammatory drugs (NSAIDs).

Results: A total of 434 patients (303 axSpA; 131 non-axSpA) had undergone baseline and 2y visits. Data was available for both timepoints on at least one of the four questions related to BP and MS in 266 (88%) axSpA and 110 (84%) non-axSpA patients. Compared to non-axSpA, axSpA patients were more frequently male (52% vs 25%) and had more SpA features (mean (SD): 5 (2) vs 3 (1)), including HLA-B27 positivity (73% vs 29%). Age (mean (SD): 29 (8) vs 31 (8) years) and symptom duration (mean (SD): 13 (7) vs 13 (7) months) were similar between groups.

Overall, lower levels of BP and MS were observed in axSpA (vs non-axSpA) patients at baseline and 2y (Table 1). After 2y, BP (total and at night) and MS severity and duration significantly improved in both groups (all p≤0.001), even though symptoms persisted in a considerable number of patients (mainly in the non-axSpA group). A mean improvement ranging from 1.4 to 1.6 points for axSpA patients and 1.0 to 1.9 points for non-axSpA patients was reported for each outcome. In adjusted multivariate analysis, axSpA (vs non-axSpA) was an independent predictor of a lower BP at night at 2y (adjusted coefficient = -0.8, 95% CI [-1.5; -0.2]; p=0.012), with no significant differences found for to-

TO 147 – TABLE 1. Baseline and two-years back pain and morning stiffness in early axSpA and non-axSpA chronic back pain patients.

	AxSpA			Non-axSpA		
	Baseline	2 years	p-value (over time)	Baseline	2 years	p-value (over time)
Back pain at any time, N		249			94	
mean (SD)	4.0 (2.4)	2.6 (2.3)	p<0.001*	5.5 (2.2)	3.9 (2.6)	p<0.001*
Change over time, mean (SD)		-1.4 (2.5)			-1.6 (2.4)	
Present (score >2), n (%)	168 (67%)	107 (43%)		85 (90%)	63 (67%)	
Back pain at night, N		247			94	
mean (SD)	3.6 (2.9)	2.2 (2.5)	p<0.001*	4.6 (2.7)	3.6 (2.8)	p=0.001*
Change over time, mean (SD)		-1.4 (2.9)			-1.0 (2.7)	
Present (score >2), n (%)	147 (60%)	82 (33%)		71 (76%)	56 (60%)	
Morning stiffness severity, N		261			106	
mean (SD)	4.7 (2.9)	3.1 (2.6)	p<0.001*	6.0 (2.8)	4.0 (3.0)	p<0.001*
Change over time, mean (SD)		-1.6 (3.2)			-1.9 (2.9)	
Present (score >2), n (%)	186 (71%)	126 (48%)		89 (84%)	66 (62%)	
Morning stiffness duration#, N		260			107	
mean (SD)	3.6 (2.8)	2.3 (2.4)	p<0.001*	4.6 (2.9)	3.1 (2.9)	p<0.001*
Change over time, mean (SD)		-1.2 (3.0)			-1.5 (3.1)	
Present (score >2), n (%)	159 (61%)	97 (37%)		82 (77%)	51 (48%)	

* Statistical significance (p-value <0.05). # Morning stiffness duration was assessed using a numeric rating scale (NRS) ranging from 0 (0 hours) to 10 (≥2 hours); for the other outcomes, a NRS ranging from 0 (no symptom) to 10 (unbearable symptom) was used. axSpA: axial spondyloarthritis.

tal BP (adjusted coefficient = -0.5, 95% CI [-1.1; 0.1]; p=0.088) or MS severity (adjusted coefficient = -0.5, 95% CI [-1.2; 0.1]; p=0.082) and duration (adjusted coefficient = -0.5, 95% CI [-1.1; 0.1]; p=0.114).

Conclusion: Over 2y, BP and MS significantly improve in early axSpA. Although a similar improvement is seen in non-axSpA patients, at 2y most patients have persisting symptoms. AxSpA (vs non-axSpA) is an independent predictor of larger improvement in BP at night but not of the observed improvements in total BP and MS.

149 - CHARACTERIZATION OF SKIN ULCERS IN SCLERODERMA PATIENTS: A RETROSPECTIVE COHORT STUDY

Carolina Vilafanha[1,2], Pedro Miguel Teixeira[1,2], Carolina Mazeda[1,2], C. Pinto Oliveira[1,2], Ana Rita Prata[1,2], Anabela Barcelos[1,2]

[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Introduction: Systemic sclerosis (SSc) is an immune-mediated connective tissue disease. Skin ulcers in SSc are frequent and can lead to decreased functionality and quality of life. Previous cohort studies have studied skin ulcer predictors, although with significant heterogeneity and using different skin ulcer definitions. The aim of this study was to evaluate the prevalence of skin ulcers and their clinical and laboratory-associated factors in a Portuguese SSc cohort.

Material and Methods: Retrospective cohort study including consecutive patients fulfilling the ACR/EULAR 2013 classification criteria for SSc, followed in a single Rheumatology Department with an initial diagnosis between January 1st, 2012, and January 1st, 2022. Demographic and clinical data were collected for each patient. Skin ulcers occurring after diagnosis were retrospectively identified based on patients' registries. Skin ulcers were categorized as ischemical/mechanical/calcinosis-related. Ischemical ulcers were identified using the definition proposed by Suliman et. al. Mann-Whitney U, Kruskal-Wallis and Pearson correlation coefficient tests were performed to assess the clinical and laboratory factors associated with an increased number of ulcers in SSc patients.

Results: We included 30 SSc patients (86.7% females, mean age at diagnosis 52.8 ±11.4 years, median follow-up time of 5.5 ± 5.5 years). Twenty-three patients (76.7%) had Limited Systemic Scleroderma. During follow-up, 30.0% of the patients developed skin ulcers. Among these 44.4% presented with ulcers at the first observation. The median number of skin ulcers during follow-up was 6.0 ± 9.0. Ischemic ulcers were the most frequent (83.8% of all ulcers) followed by calcinosis-related (11.8%) and mechanical ulcers (4.4%).

Regarding location, the hand/digits were the most fre-

quent site of ischemic ulcers (84.2% of all ischemic), and digital lesions were more common on the third and fourth digits (35.1% and 19.3%, respectively). The most frequent sites of calcinosis-related ulcers were the first and second digits (37.5% each). All mechanical ulcers were in the skin overlying the fifth proximal interphalangeal. During follow-up, 8 patients (88.9%) received at least one course of intravenous Alprostadil. In the most recent observation patients were being treated with nifedipine (100%), phosphodiesterase 5 inhibitors (44.4%), topical nitrates (44.4%), and colchicine (22.2%). Five patients (55.6%) had infectious complications, requiring topical and/or oral antibiotics. No critical ischemia leading to gangrene was registered. Baseline modified Rodnan skin score (mRss) showed a positive correlation with the number of ulcers developed during the follow-up ($r = 0.898$; $p = 0.015$).

No statistically significant difference was found regarding the number of ulcers developed during follow-up and SSc subtype, gender, age at diagnosis, smoking status, presence of puffy hands, microstomia, telangiectasias, calcinosis, sclerodactyly or baseline capillaroscopic pattern and immunological profile.

Conclusions: In this single-center based-cohort study, skin ulcers were frequent and the most reported were digital ischemic ulcers of the hands. Infection was a frequent complication. Baseline mRss showed a positive correlation with the number of ulcers developed during the follow-up, as suggested by previous works. Larger sample size studies using uniform validated skin ulcer definitions are needed to better study SSc-associated skin ulcer factors to develop prevention strategies and improve patients' quality of life.

152 - EFFICACY AND SAFETY OF RITUXIMAB IN REFRACTORY SYSTEMIC LUPUS ERYTHEMATOSUS

Filipe Cunha Santos[1], Henry Penn[2], Pedro M Machado[2, 3], Shahir Hamdulay[2]

[1]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [2]Rheumatology, Northwick Park Hospital, London North West University Healthcare NHS Trust, London, United Kingdom, [3]Centre for Rheumatology Research & MRC Centre for Neuromuscular Diseases, University College London, London, United Kingdom

Background: SLE is a complex disease with diverse clinical manifestations, ranging from a mild constitutional syndrome or arthritis, to severe organ and life-threatening disease. B cells play a crucial role in SLE pathogenesis and are a desirable therapeutic target. RTX is a B cell depleting monoclonal antibody, which has shown to be effective in several observational studies, but randomized trials (EXPLORER and LUNAR) did not confirm these findings in moderate-severe SLE or active lupus nephritis, respectively.

Methods: This retrospective study analysed real-world tertiary care data of patients fulfilling EULAR/ACR-2019 classification criteria for SLE and treated with RTX (2g) for disease refractory to conventional immunosuppressant therapy (AZA/MTX/MMF) in combination with HCQ. We also aimed to assess differences between anti-dsDNA positive and anti-dsDNA negative subpopulations.

Data on demographics, disease features, serological markers (anti-dsDNA antibodies, ESR, CRP, C3 and C4 complement, IgG levels), disease activity scores (SLEDAI-2K and SLEDAS) were collected at baseline, after 6 and 12 months of treatment initiation, and prednisolone dosage at baseline and after 12 months. Treatment data on immunosuppressants was collected and safety data (adverse events, serious infections and deaths). The primary analysis investigated pre- and post-RTX differences in disease activity scores and prednisolone dosage (primary outcomes). A secondary analysis investigated subgroup differences based on anti-dsDNA positivity.

Descriptive data was generated. Normally distributed quantitative variables were compared using the independent samples or the paired Student's t-test, and non-normally distributed quantitative variables were compared using the Mann-Whitney or the Wilcoxon sign-rank test, as appropriate. p values were considered statistically significant if $p<0.05$.

Results: A total of 15 patients (10 anti-dsDNA positive and 5 anti-dsDNA negative) treated between 01/2001 and 05/2023 were identified, with a combined mean age of 41±15 years. The majority of patients had skin and joint involvement (Table 1).

There was a statistically significant decrease in disease activity scores at 6 months and 12 months (SLEDAI 2K - T0: 11.3±5.9, T1: 5.3±3.3, T2: 4.3±4.0, $p(T0-T1) = 0.003$, $p(T0-T2) <0.001$; SLEDAS - T0: 16.5 (13.7), T1: 5.2 (5.5), T2: 2.1 (7.3), $p(T0-T1) <0.001$, $p(T0-T2) <0.001$). There was also a significant decrease in mean prednisolone dosage at 12 months after treatment with RTX (T0: 27.2±14.4; T2: 8.9±5.6 mg, $p(T0-T2) <0.001$). ESR, anti-dsDNA levels, total IgG and C4 levels all improved at 12 months after treatment (Table 2). There was no significant difference in other serological markers.

Both anti-dsDNA positive and negative patients had a decrease in their disease activity scores at both timepoints, and no differences were found between both subgroups (Table 3). No serious adverse effects were reported during follow-up.

Conclusions: RTX appears to be in treating patients

TO 152 – TABLE 1.

Variable	RTX group *n*=15
Demographic characteristics	
Age, years	41.1±14.8
Sex (women/men)	14/1
Ethnicity	
South Asian	9 (60.0%)
Afro-Caribbean	5 (33.3%)
Caucasian	1 (6.7%)
Arab	0 (0.0%)
Disease duration, years	10.5±6.8
Clinical manifestations	
Joint involvement	13 (86.7%)
Skin involvement	12 (80.0%)
Serositis	5 (33.3%)
Pleurisy	5 (33.3%)
Nephritis	4 (26.7%)
CNS involvement	6 (40.0%)
SLEDAI-2K baseline	11.3±5.9
SLE-DAS baseline	18.0±9.1
Serological markers	
ANA	15 (100.0%)
Anti-Ro	10 (66.7%)
Anti-La	4 (26.7%)
Anti-dsDNA	10 (66.7%)
Anti-Sm	6 (40.0%)
RF	1 (6.7%)
Anti-RNP	4 (26.7%)
ESR (mm/hour)	46.0 (68.0)
Complement C3 (g/L)	0.9±0.3
Anti-dsDNA (UI/mL)	49.0 (247.0)
IgG (g/L)	14.9 (9.9)
Treatment	
Prednisolone dose at first treatment (mg/day)	20.0 (20.0)
Hydroxychloroquine	14 (93.3%)
Azathioprine	3 (20.0%)
Methotrexate	2 (13.3%)
Mycophenolate	8 (53.3%)
None	2 (13.3%)

Values are mean±standard deviation or median (interquartile range)
ANA: antinuclear antibody; CNS: central nervous system; dsDNA: double stranded DNA; ESR: erythrocyte sedimentation rate; IQR: interquartile range; RNP: ribonucleoprotein; RF: rheumatoid factor; RTX: rituximab; SD: standard deviation; SLE-DAS: Systemic Lupus Erythematosus Disease Activity Score; SLEDAI-2K: Systemic Lupus Erythematosus Disease Activity Index 2000

TO 152 – TABLE 2. Comparison of primary and secondary outcomes at baseline, 6 and 12 months in refractory SLE patients on RTX

Variable	Baseline (T0)	6 months (T1)	12 months (T2)	*p (T0-T1)*	*p (T0-T2)*
Primary outcome					
SLEDAI-2K	11.3±5.9	5.3±3.3	4.3±4.00	**0.003****	**<0.001****
SLEDAS	16.5 (13.7)	5.2 (5.5)	2.1 (7.3)	**<0.001****	**<0.001****
Secondary outcomes					
ESR (mm/h)	46.0 (68.0)	25.0 (41.0)	31.0 (25.0)	**0.004****	**0.027****
CRP (mg/L)	2.5 (24.4)	3.0 (13.2)	2.2 (10.1)	0.972	0.969
C3 (g/L)	0.9±0.3	1.0±0.3	1.1±0.3	0.177	0.078
C4 (g/L)	0.1±0.1	0.2±0.1	0.2±0.1	**0.018****	**0.048****
dsDNA (UI/mL)	49.0 (247.0)	5.8 (180.4)	6.9 (69.2)	0.196	**0.009****
IgG (g/L)	14.9 (9.9)	14.1 (7.7)	14.0 (7.4)	**0.020****	**0.019****

Values are mean±standard deviation or median (interquartile range)
CRP: C-reactive protein; dsDNA: double stranded DNA; ESR: erythrocyte sedimentation rate; RTX: rituximab; SD: standard deviation; SLE-DAS: Systemic Lupus Erythematosus Disease Activity Score; SLEDAI-2K: Systemic Lupus Erythematosus Disease Activity Index 2000
**indicates statistical significance.

TO 152 – TABLE 3. Comparison of diease activity scores between dsDNA negative patients on rituximab.

Variable	dsDNA(+)-RTX group *n=10*	dsDNA(-)-RTX group *n=5*	*p* value
SLEDAI-2K			
Δ 6 months-baseline	-5.6±5.1	-6.6±3.0	0.640
Δ 12 months-baseline	-6.3±6.3	-8.4±3.6	0.425
SLEDAS			
Δ 6 months-baseline	-11.8±9.1	-13.9±10.8	0.709
Δ 12 months-baseline	-11.0±9.1	-14.7±11.4	0.543

Values are mean±standard deviation or median (interquartile range)
dsDNA: double stranded DNA; RTX: rituximab; SD: standard deviation; SLE-DAS: Systemic Lupus Erythematosus Disease Activity Score; SLEDAI-2K: Systemic Lupus Erythematosus Disease Activity Index 2000

with SLE whom have failed a combination of HCQ and other immunosuppressants and aids steroid reduction or withdrawal. These results also suggest benefit in both anti-dsDNA negative as well as anti-dsDNA positive patients.

159 - CLINICAL CHARACTERIZATION OF PATIENTS WITH SJÖGREN'S DISEASE REGISTERED IN REUMA.PT: PORTRESS — THE PORTUGUESE REGISTRY OF SJÖGREN'S SYNDROME

Matilde Bandeira[1, 2], Manuel Silvério-António[1, 2], Roberto Costa[1, 2], Ana Rita Lopes[1, 2], Filipe Cunha Santos[3], Paulo Jorge Pereira[4], Diana Raimundo[5], Anita Cunha[6], C. Pinto Oliveira[7], Ana Catarina Duarte[8], João Madruga Dias[9], Mariana Emília Santos[10], Maria João Gonçalves[10, 11], Ana Catarina Moniz[10], Ana Isabel Maduro[12], Mariana Luis[12, 13], Ana Valido[14], Margarida Oliveira[15], Marília Rodrigues[16], Catarina Tenazinha[17], Nikita Khmelinskii[1, 2], Filipe Barcelos[18, 19], JE Fonseca[1, 2], Vasco C Romão[1, 2], PORTRESS Reuma.pt Task Force[8, 10, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31]

[1]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [4]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [5]Serviço de Reumatologia, Hospital Beatriz Ângelo, Loures, Portugal, [6]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [7]Serviço de Reumatologia, Centro Hospitalar do Baixo Vouga, E.P.E., Aveiro, Portugal, [8]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [9]Serviço de Reumatologia, Centro Hospitalar Médio Tejo, Torres Novas, Portugal, [10]Serviço de Reumatologia, Hospital de Egas Moniz, Centro Hospitalar de Lisboa Ocidental, Lisboa, Portugal, [11]Serviço de Reumatologia, Hospital CUF Santarém, Santarém, Portugal, [12]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [13]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal, [14]Serviço de Reumatologia, Unidade Local de Saúde do Litoral Alentejano, Santiago do Cacém, Portugal, [15]Serviço de Reumatologia, Centro Hospitalar Universitário Cova da Beira, Covilhã, Portugal, [16]Serviço de Reumatologia, Centro Hospitalar de Leiria, Leiria, Portugal, [17]Serviço de Reumatologia, Centro Hospitalar e Universitário do Algarve, Faro, Portugal, [18]Instituto Português de Reumatologia, Lisboa, Portugal, [19]Rheumatology Department, Hospital CUF Descobertas, Lisboa, Portugal, [20]Rheumatology, Centro Hospitalar Entre o Douro e Vouga, Santa Maria da Feira,

Portugal, [21]Unidade de Reumatologia, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [22]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Central, Lisboa, Portugal, [23]Serviço de Reumatologia, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal, [24]Serviço de Reumatologia, Hospital Central do Funchal, Funchal, Portugal, [25]Serviço de Reumatologia, Hospital CUF Cascais, Cascais, Portugal, [26]Serviço de Reumatologia, Centro Hospitalar de São João, Porto, Portugal, [27]Serviço de Reumatologia, Centro Hospitalar Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [28]Rheumatology Department, Hospital Distrital de Santarém, Santarém, Portugal, [29]Serviço de Reumatologia, Hospital do Divino Espírito Santo, Ponta Delgada, Portugal, [30]Rheumatology, Hospital de Santo Espírito, Angra do Heroísmo, Portugal, [31]Unidade de Reumatologia, Quadrantes - Clínica de Cascais, Cascais, Portugal

Background: Recently, a Reuma.pt protocol was specifically designed for Sjögren's Disease (SjD) patients to allow deep patient characterisation and expand our knowledge on this complex condition. This protocol is the basis for PORTRESS, the Portuguese registry of SjD patients.

Objectives: To characterize the largest nationwide cohort of SjD patients.

Methods: Multicentre open cohort study, including patients with a clinical diagnosis of SjD, registered in Reuma.pt up to June 2023. Demographic, clinical, treatment and patient-reported outcomes (PROs) data were collected. Means were compared using paired samples t-student or Wilcoxon signes-rank test according to distribution.

Results: 1077 patients were included (Table 1). Patients fulfilled AECG 2002 or ACR/EULAR 2016 classification criteria in 65.2% and 59.2% of cases, respectively, although a large percentage (60.4%) did not have a complete assessment of all the criteria. Importantly, the vast majority of patients had sicca symptoms (or extraglandular involvement) and a positive anti-Ro or salivary gland (SG) biopsy (n=895/989, 90.5%).

The majority of patients were ANA and anti-Ro positive. Anti-La and rheumatoid factor were positive in almost half the patients and anti-CCP in 7.9%. Hypergammaglobulinemia (n=381/795, 47.9%) and raised immunoglobulin G (n=200/491, 40.7%) were common. A quarter of the patients had raised beta 2-microglobulin (n=111/401, 27.7%) and 16.5% circulating cryoglobulines (n=51/310).

Schirmer's test and unstimulated sialometry were performed in around one third of patients and were positive in 55.4% and 34.1% of cases, respectively. Major SG ultrasound, scintigraphy and minor SG biopsy were positive in 68.7, 55.8 and 52.9% of patients who per-

TO 159 – TABLE 1. Clinical and demographic characteristics of PORTRESS cohort

	PORTRESS cohort n=1077
Age at diagnosis (years)	54.0±20.7 (13.8-91.3)[1]
Age at symptom onset (years)	49.8±22.6 (8.0-85.5)[1]
Diagnosis delay (years)	4.7±6.0 (0.0-45.0)[2]
Disease duration (years)	10.6±10.9 (0.2-58.9)[1]
Female, n (%)	1023 (95)
Ethnicity, n (%)	
Caucasian	697 (93)
African ancestry	40 (5)
Asian	12 (2)
Smoking habits, n (%)	
Has never smoked	507 (75)
Past smoker	115 (17)
Current smoker	54 (8)
Alcohol, n (%)	
Has never consumed	626 (97)
Past consumer	3 (1)
Current consumer	16 (2)
AECG 2002 classification criteria, n (%)	643 (65)
ACR/EULAR 2016 classification criteria, n (%)	355 (59)
ANA, n (%)	825 (90)
Anti-Ro, n (%)	744 (82)
Anti-La, n (%)	409 (47)
RF, n (%)	392 (49)
Anti-CCP, n (%)	39 (8)
ESSDAI (0-123)	3.2±4.8 (0-42)[2]
ESSPRI (0-10)	5.5±2.6 (0-10)[2]
EULAR Sicca Score (0-10)	5.4±2.8 (0-10)[2]
Xerostomia Inventory (11-55)	36.1±10.5 (11-55)[2]
PROFAD-SSI (0-56)	28.5±12.6 (1-56)[2]
CODS (0-9)	3.6±1.7 (0-9)[2]
USF <0.1ml/min, n (%)	114 (34)
Schirmer's ≤5mm/5min, n (%)	200 (55)
van Bijsterveld score ≥4, n (%)	38 (14)
Ocular Staining Score ≥5, n (%)	35 (13)
Minor SG biopsy, Chisholm/Mason grade, n (%)	
Grade 0	41 (10)
Grade 1	86 (21)
Grade 2	65 (16)
Grade 3	83 (20)
Grade 4	133 (33)
Minor SG biopsy, focus score	0.73±1.09 (0-8)[2]
Minor SG biopsy, focus score ≥ 1, n (%)	216 (53)
SG ultrasound OMERACT score, n (%)	
Grade 0	9 (17)
Grade 1	15 (29)
Grade 2	23 (44)
Grade 3	5 (10)

continues on the next page

TO 159 – TABLE 1. Continuation.

	PORTRESS cohort n=1077
SG ultrasound Salaffi score, n (%)	
Grade 0	34 (14)
Grade 1	42 (17)
Grade 2	62 (26)
Grade 3	93 (38)
Grade 4	12 (5)
SG scintigraphy – Schall score, n (%)	
Grade 0	76 (44)
Grade 1	21 (12)
Grade 2	18 (11)
Grade 3	36 (21)
Grade 4	21 (12)
Systemic involvement, n (%)	
Constitutional	184 (21)
Lymphadenopathic	109 (13)
Glandular	301 (35)
Articular	380 (43)
Cutaneous	175 (20)
Pulmonary	74 (9)
Renal	32 (4)
Muscular	15 (2)
PNS	33 (4)
CNS	14 (2)
Hematologic	305 (35)
Biologic*	495 (57)
Gastrointestinal/Hepatobiliary	29 (3)
Other**	138 (17)
Treatment, n (%)	
Hydroxicloroquine	439 (71)
Corticosteroids	269 (43)
Pilocarpine	121 (20)
Methotrexate	99 (16)
Azathioprine	72 (12)
Rituximab	29 (5)
Leflunomide	17 (3)
MMF	15 (2)
IVIG	5 (1)
Anti-TNF	5 (1)
Cyclophosphamide	4 (1)
Tocilizumab	1 (0)
Other therapies, n (%)	
Antidepressants	93 (15)
Benzodiazepines/hypnotics	85 (14)

continues on the next column

TO 159 – TABLE 1. Continuation.

	PORTRESS cohort n=1077
Diuretic drugs	54 (9)
Beta-blockers	41 (7)
NSAIDs	32 (5)
Opioids	23 (4)
Antihistamines	12 (2)

Abbreviations: n – number of patients positive for the variable of interest, RF – rheumatoid factor, USF – unstimulated saliva flow rate, SG – salivary gland, PNS – peripheral nervous system, CNS – central nervous system; MMF – mycophenolate mofetil, IVIG – intravenous Immunoglobulin therapy, NSAIDs – non-steroidal anti-inflammatory drugs; * characterized by laboratorial features of B cell hyperactivity; ** namely Raynaud's phenomenon, pericarditis or pulmonary hypertension among others less frequent; 1 – median±interquartile range (range), 2 – mean±standard deviation (range)

formed it (Table 1).

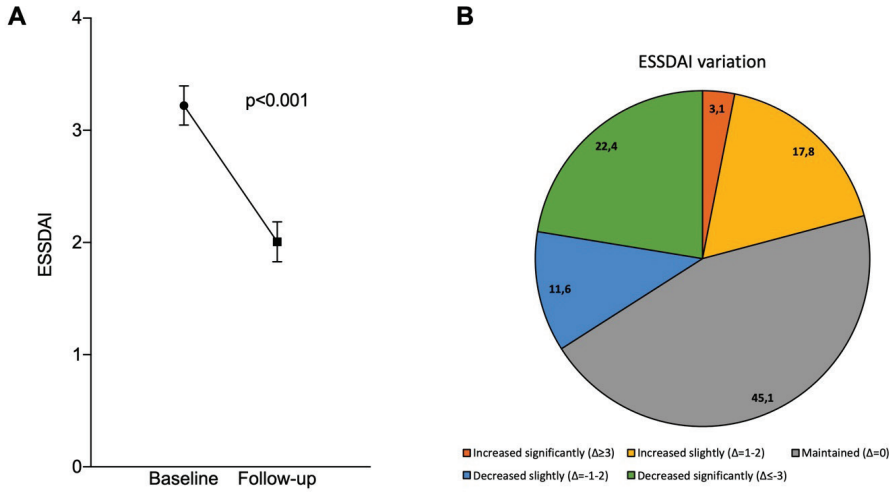
Over the course of the disease, 709/795 patients (89.2%) had some kind of systemic involvement, most commonly in the biological domain, characterized by laboratorial features of B cell hyperactivity (n=495/872, 56.8%). Articular, hematologic and glandular involvements were seen in 35-43% of patients (Table 1).

Hematological neoplasia was found in 19/635 patients (3.0%), most commonly MALT lymphoma. Of note, known risk factors for lymphoma were present in a significant part of the cohort. In addition to cryoglobulines (16.5%), lymphopenia and low C3 were seen in 20.1 and 17.8%, respectively. Furthermore, decreased C4 was present in 8.8%, persistent SG swelling in 7.2%, cutaneous vasculitis in 6.4% and monoclonal gammopathy in 6.2% of patients. When submitted to a salivary gland biopsy, ectopic lymphoid structures were seen in 34/522 patients (6.5%).

The most commonly used drugs were hydroxychloroquine (70.6%) and corticosteroids (43.2%). Pilocarpine was also frequently used (19.5%), whereas up to 15-20% of patients were treated with other immunossupressants (Table 1).

The mean ESSDAI was 3.2±4.8 (range 0-42), corresponding to 75.4% (n=578/767) of patients with low systemic disease activity (ESSDAI<5). At the last follow-up, the mean ESSDAI was 2.0±3.8 (range 0-30), corresponding to a significant decrease from baseline (Fig. 1A). Around 21% of patients experienced disease worsening, whereas 34% improved (Fig. 1B). Finally, dryness, pain and fatigue PROs were high (Table 1).

Conclusions: The Reuma.pt SjD module allows for the registry of the main features of these patients, displaying SjD as the true multisystemic disease it is. Of note, the vast majority of patients had systemic involve-



TO 159 – Figure 1. Systemic disease activity assessed by ESSDAI at baseline and follow-up

ment throughout follow-up and substantial symptom burden.

161 - ANTI-CITRULLINATED PROTEIN ANTIBODIES IN SJÖGREN'S SYNDROME DEFINE A SUBSET OF PATIENTS WITH LOWER B CELL ACTIVATION MARKERS AND HIGHER RISK OF LUNG INVOLVEMENT

Augusto Silva[1,2], Filipa Marques Costa[1,2], Mariana Silva[1,2], G. Fulvio[3], Matilde Bandeira[4,5], Manuel Silvério-António[6,7], Nikita Khmelinskii[1,2], C. Baldini[3], Vasco C Romão[1,2]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas do Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Rheumatology Unit, University of Pisa, Firenze, Italy, [4]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [5]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [6]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [7]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Extraglandular manifestations may occur in up to 40-50% of patients with Sjögren's syndrome (SS), including inflammatory arthralgia and chronic polyarthritis (2,3). Anticyclic-citrullinated protein antibody (ACPA) are prototypical markers of rheumatoid arthritis (RA), but have also been described in 4-10% of patients with SS (3,4). Although ACPA have been associated with articular and lung involvement, their clinical relevance remains to be fully clarified (3,4).
Objective: To evaluate the prevalence of ACPA in SS patients and assess their associations with clinical, laboratorial and radiographic features.
Methods: We screened patients from the Observational Lisbon Sjögren's Syndrome Prospective (OLISSIPO) and University of Pisa (UNIPI) cohorts, included until May 2023, for the presence of ACPA. For each ACPA-positive identified, two ACPA-negative patients matched for age and sex were selected. We collected demographic, clinical and laboratorial variables. t-student or Mann-Whitney tests were used, as appropriate, for continuous variables and chi-square or Fisher tests for categorical variables.
Results: From 438 SS patients tested for ACPA, 57 of them were ACPA-positive (94.7% female), corresponding to a prevalence of 13.0%. After matching for age and sex, 176 patients (95.5% women) were included (Table 1). Age at SS diagnosis and disease duration were similar between groups. There were no differences in systemic disease activity, as assessed by the EULAR Sjögren's Syndrome disease activity index (ESSDAI), nor in dryness, pain and fatigue assessed by the EULAR Patient Reported Index (ESSPRI), EULAR sicca score (ESS), and Profile of Fatigue and Discomfort Sicca Symptoms Inventory (PROFAD-SSI). Similarly, xerostomia, xerophthalmia, salivary gland (SG) swelling and objective measures of reduced glandular function or structural changes (SG ultrasound score) were also

TO 161 – TABLE 1. Clinical features of patients with Sjögren's Syndrome

Parameter (0)	ACPA positive N = 57	ACPA negative N = 119	p-value
Female	54 (94.7%)	114 (95.8%)	0.752
Age at diagnosis (years)	52.7±13.4 (19-78)	50.6±14.6 (17-80)	0.928
Smoking status			
• Never smoker	25 (73.5%)	65 (67.7%)	0.814
• Active Smoker	3 (8.8%)	11 (11.5%)	
• Former smoker	6 (17.7%)	20 (20.8%)	
SS duration (years)	8.9±8.1 (0-39)	10.7±8.5 (1-47)	0.069
Disease assessment at diagnosis			
• ESSDAI	4.0±4.0	3.8±6.1	0.107
• ESSPRI	5.8±2.3	6.0±2.3	0.523
• ESS	5.7±2.6	5.9±2.6	0.891
• PROFAD-SSI	24.7±16.1	27.1±9.2	0.426
Disease assessment at last visit			
• ESSDAI	4.5±7.8	2.0±3.0	0.194
• ESSPRI	4.3±1.8	5.9±1.9	0.373
• ESS	4.3±2.9	5.5±1.9	0.189
Salivary and lacrimal gland assessment			
• Xerostomia	46/54 (85.2%)	40/44 (90.9%)	0.297
• Unstimulated salivary flow (ml/15min)	4.7±5.6	4.1±5.2	0.309
• Clinical oral dryness score	3.7±1.5	3.6±1.8	0.325
• Salivary gland swelling	8/36 (22.2%)	23/107 (21.5%)	0.371
• Xerophthalmia	43/46 (93.5%)	41/45 (91.1%)	0.488
• Schimer's test	3.5±2.2	3.2±2.7	0.487
• van Bijsterveld ocular score	2.4±2.4	1.8±1.8	0.108
• Tear break-up time	7.7±4.6	7.7±4.4	0.776
SG biopsy Chisholm-Mason score, n (%)			
• Grade 0	1 (7.7%)	5 (8.2%)	0.018
• Grade 1	6 (46.2%)	6 (9.8%)	
• Grade 2	3 (23.1%)	14 (23.0%)	
• Grade 3	0	11 (18%)	
• Grade 4	3 (23.1%)	25 (41%)	
SG Biopsy Focus score ≥1, n (%)	3 (23.1%)	36 (59.0%)	0.019
SG Ultrasound score, n (%)			
• Grade 0	1 (5.3%)	4 (6.0%)	0.056
• Grade 1	0 (0%)	5 (7.5%)	
• Grade 2	5 (26.3%)	12 (17.9%)	
• Grade 3	4 (21.1%)	33 (49.3%)	
• Grade 4	9 (47.4%)	13 (19.4%)	
SG Ultrasound score ≥3, n (%)	13 (68.4%)	46 (68.7%)	0.595
Autoantibodies, n (%)			
• ANA	48 (84.2%)	106 (89.1%)	0.147
• Anti-Ro52	30 (52.6%)	84 (70.6%)	<0.001
• Anti-Ro60	29 (50.9%)	75 (63.0%)	0.009
• Anti-Ro52 and Anti-Ro60	26 (46.4%)	72 (69.2%)	0.004
• Anti-La	17 (29.8%)	69 (58.0%)	<0.001
• Rheumatoid Factor	41 (71.9%)	97 (81.5%)	0.107
Biological markers of disease activity			
• ESR elevation	14/50 (28%)	24/77 (31.2%)	0.430
• CRP elevation	15/53 (28.3%)	17/73 (23.3%)	0.332
• C3 consumption	8/57 (14.0%)	10/28 (35.7%)	0.024
• C4 consumption	8/56 (14.3%)	6/28 (21.4%)	0.297
• Hypergammaglobulinemia	17/55 (30.9%)	17/31 (54.8%)	0.026
• Hypogammaglobulinemia	1/57 (1.8%)	0/119 (0%)	0.324
• IgG elevation	11/52 (21.2%)	46/62 (74.2%)	<0.001
• Cryoglobulinemia	2/51 (3.9%)	6/97 (6.2%)	0.414
• MGUS	1/57 (1.8%)	4/119 (3.4%)	0.048

continues on the next pagw

TO 161 – TABLE 1. Continuation

Parameter (0)	ACPA positive N = 57	ACPA negative N = 119	p-value
Joint involvement, n (%)			
• Arthralgia	43/55 (78.2%)	75/105 (71.4%)	0.233
• Arthritis	27/53 (50.9%)	58/105 (55.2%)	0.366
• Fulfil RA criteria ACR/EULAR 2010	22/55 (40%)	4/119 (3.4%)	<0.001
• Radiographic erosions	11/48 (22.9)	2/23 (8.7%)	0.129
• Ultrasound erosions	7/47 (14.9%)	1/17 (5.9%)	0.313
Extra-articular involvement, n (%)			
• Constitutional	21/36 (58.3%)	49/102 (48.0%)	0.087
• Lymphadenopathy	11/56 (19.6%)	25/112 (22.3%)	0.426
• Cutaneous	8/35 (22.9%)	29/108 (26.9%)	0.647
• Pulmonary (1)	8/34 (23.5%)	9/105 (8.6%)	0.028
• Renal	2/35 (2.9%)	4/106 (3.8%)	0.282
• Muscular	1/35 (2.9%)	6/106 (5.7%)	0.284
• Central Nervous System	1/57 (1.8%)	2/119 (1.7%)	0.683
• Peripheral Nervous System	1/35 (2.9%)	7/107 (6.5%)	0.495
• Hemathologic involvement	9/35 (25.7%)	41/107 (38.3%)	0.516
• Vasculopathy (2)	5/56 (8.9%)	10/119 (8.4%)	0.557
• Haematological neoplasms	2/57 (3.5%)	0/119 (0%)	0.104
• Solid neoplasms	1/57 (1.8%)	4/119 (3.4%)	0.478

Abbreviations: ACPA - Anticyclic-citrullinated protein antibody; CRP - C-reactive Protein; ESR - Erythrocyte sedimentation rate; ESSPRI - Eular Sjögren's Syndrome Patient Reported Index; ESSDAI - EULAR Sjögren's syndrome disease activity index; ESS - EULAR sicca score; MGUS - monoclonal gammopathy of undetermined significance; RA - Rheumatoid arthritis; PROFAD-SSI - Profile of Fatigue and Discomfort Sicca Symptoms Inventory; SS - Sjögren's syndrome; SD - Standard Deviation; (0) Continuous variables presented as mean ± sd (range); categorical variables presented as n (%) or n/N (%). (1) Pulmonary - Nonspecific interstitial pneumonia, ground-glass pattern, pulmonary amyloid tumour, pulmonary micronodules and bronchiectasis. (2) Vasculopathy - Raynaud's Phenomenon.

comparable between ACPA-positive and ACPA-negative patients. However, ACPA-positivity was associated with a significantly lower SG lymphocytic infiltration (focus score≥1: 23.1% vs 59.0%, p=0.019) and a reduced frequency of anti-Ro52 (52.6% vs 70.6%, p<0.001), anti-Ro60 (50.9% vs 63.0%, p=0.009), and anti-SSB (29.8% vs 58.0%, p<0.001) antibodies, but no differences in rheumatoid factor (RF) positivity (71.9% vs 81.5%, p=0.107). In accordance, markers of B cell hyperactivation such as C3 consumption (14.0% vs 35.7%, p=0.024), hypergammaglobulinemia (30.9% vs 54.8%, p=0.026) and IgG elevation (21.2% vs 74.2%, p<0.001) were also less commonly associated with the presence of ACPA. Of note, articular involvement or erosive disease were not more frequent in ACPA-positive patients. Nonetheless, a higher number of ACPA-positive patients met the ACR/EULAR 2010 classification criteria for RA (40.0% vs 3.4%, p<0.001). Finally, lung disease was significantly more common in the ACPA-positive group (23.5% vs 8.6%, p=0.028), whereas involvement of additional organs was not.

Conclusion: In patients with SS, ACPA is not associated with joint involvement, but, surprisingly, seems to define a subgroup of patients with lower markers of B cell activation (such as SG lymphocytic infiltration, circulating anti-SSA/B, complement consumption and raised immunoglobulins). Furthermore, ACPA was associated with lung involvement, despite a similar distribution of known markers of pulmonary disease in SS such as RF. These findings deserve further confirmation in larger cohorts.

162 - OUTCOMES OF A FRACTURE LIAISON SERVICE - A SINGLE CENTRE EXPERIENCE

Augusto Silva[1, 2], Ana Margarida Duarte-Monteiro[1, 2], Ana Rita Lopes[1, 2], Ângela Pinto[3], Manuel Silvério-António[1, 2], Margarida Magalhães[4], Ana Rita Cruz-Machado[1, 2], Ana Margarida Silva[3], António Tirado[3], José Carlos Romeu[1, 2]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Ortopedia e Traumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa,, Lisboa, Portugal, [4]Unidade de Saúde Familiar do Parque, Agrupamento de Centros de Saúde Lisboa Norte, Lisboa, Portugal

Introduction: Fracture Liaison Services (FLS) are coordinated care programs that identify and manage patients with fragility fractures (FF), to prevent further fractures and minimize the osteoporosis' burden (1,2). FLS can lower the probability of subsequent fractures

TO 162 – TABLE 1. Demographic and clinical data of patients referred to the Fracture Liaison Service

Parameter	Intervention group N = 393	Control group (3) N = 114	p value
Age at the fracture - mean ± SD (years)	83.1 ± 8.8	81.9 ± 10.8	0.152
Female gender, n (%)	313 (79.6%)	87 (76.3%)	0.222
Walking autonomy prior to fracture, n (%)			
• Unassisted walking	205 (52.7%)	44 (38.6%)	0.018
• Assisted walking	169 (43.4%)	63 (55.3%)	
• Bedridden	15 (3.9%)	4 (3.5%)	
• Unknown	0	3 (2.6%)	
Follow-up time of all patients - median [IQR], min-max (months)	17 [11 - 24], 0 - 40	15 [3.75 - 25], 0 - 39	0.06
Follow-up of the living patients - median [IQR], min-max (months)	19 [13 - 25], 9 - 40	16 [10.75 - 25], 9 - 39	0.09
Number of comorbid conditions associated with higher fracture risk (1), n (%)			
• 0 - 2	153 (38.9%)	45 (39.5%)	0.457
• 3 - 5	190 (48.3%)	53 (46.5%)	
• > 5	50 (12.7%)	16 (14%)	
Number of drugs being taken regularly associated with increased risk of fracture (2), n (%)			
• 0 - 2	153 (38.9%)	55 (48.2%)	0.369
• 3 - 5	190 48.3%)	44 (38.6%)	
• > 5	50 (12.7%)	15 (13.2%)	
Previous fragility fractures, n (%)			
• 0	225 (63.6%)	83 (72.8%)	0.002
• 1	118 (30%)	20 (17.5%)	
• > 1	25 (6.4%)	3 (2.6%)	
• Unknown	0	8 (7%)	
Location of the previous fracture, n (%)			
• Femur	44 (30.8%)	10 (43.5%)	0.334
• Spine	68 (47.6%)	4 (17.4%)	
• Other location	25 (17.5)	8 (34.8%)	
• Unknown	6 (4.2%)	1 (4.3%)	
Current type of fracture, n (%)			
• Fragility	389 (99%)	114 (100%)	0.153
• Atypical	2 (0.5%)	0 (0%)	
• Pathological	2 (0.5%)	0 (0%)	
Current fragility fracture location, n (%)			
• Femur	391 (99.4%)	113 (99.1%)	0.242
• Spine	1 (0.3%)	0 (0%)	
• Other location	1 (0.3%)	1 (0.9%)	
Location of the current femoral fracture, n (%)			
• Subcapital	167 (42.7%)	42 (37.2%)	0.097
• Pertrochanteric	193 (49.4%)	58 (51.3%)	
• Subtrochanteric	25 (6.4%)	11 (9.7%)	
• Persubtrochanteric	6 (1.5%)	2 (1.8%)	
Time between the current fracture and the surgery, mean ± SD, min.-max. (days)	4.6 ± 3.3, 0 - 27	4.1 ± 3.6, 0 - 28	0.085
Complications during surgery or hospitalisation, n (%)			
• No	304 (77.4%)	93 (81.6%)	0.139
• Yes	87 (23.2%)	20 (17.5%)	
• Unknown	2 (0.5%)	1 (0.9%)	
Length of hospitalisation - median [IQR], min-max (days)	13 [9 - 20], 0 - 99	10 [6 - 17], 2 - 61	0.001
Patient's destiny after the discharge, n (%)			
• Home	225 (57.3%)	65 (57%)	0.425
• Nursing home / Long-term care unit	154 (39.2%)	38 (33.3%)	
• Death during hospitalisation	10 (2.5%)	6 (5.3%)	
• Unknown	4 (1%)	5 (4.4%)	

continues on the next pagw

TO 162 – TABLE 1. Continuation

Parameter	Intervention group N = 393	Control group (3) N = 114	p value
FLS recommendation to start an antiresorptive treatment, n (%)			
• No	48 (12.2%)	-	-
• Yes:	345 (87.8%)	-	
• Alendronate	• 126 (36.5%)		
• Zoledronate	• 173 (50.1%)		
• Denosumab	• 42 (12.2%)		
• Unknown*	• 4 (1.2%)		
Beginning the antiresorptive treatment proposed in the FLS (IG group) or any antiresorptive treatment (CG group), n/N (%)			
• No	134/341 (39.3%)	89/114 (78.1%)	< 0.001
• Yes	161/341 (47.2%)	10/114 (8.8%)	
• Unknown	46/341 (13.5%)	15/114 (13.2%)	
Patients who started the antiresorptive drug proposed, n/N (%)			
• Alendronate	69/126 (54.8%)	-	-
• Zoledronate	68/173 (39.3%)		
• Denosumab	24/42 (57.1%)		
Patients who have discontinued the antiresorptive therapy, n/N (%)		-	-
• Alendronate	26/69 (37.7%)		
• Zoledronate	34/68 (50%)		
• Denosumab	9/24 (37.5%)		
Period of intake of antiresorptive drug in those who stopped it, n (%)		-	-
- Alendronate			
• < 6 months	14 (53.8%)		
• 6-12 months	12 (46.2%)		
- Zoledronate			
• No administrations	7 (20.6%)		
• 1 administration only	27 (79.4%)		
- Denosumab			
• 1 administration	4 (44.4%)		
• 2 administrations	5 (55.6%)		
Period of the antiresorptive drug in those who keep taking, n/N (%)		-	-
- Alendronate			
• 6-12 months	7/43 (16.3%)		
• >12 months	36/43 (83.7%)		
- Zoledronate			
• 1 administration	7/34 (20.6%)		
• > 1 administration	27/34 (79.4%)		
- Denosumab			
• 2 administrations	1/15 (6.7%)		
• > 2 administrations	14/15 (93.3%)		
Healthcare provider referral after FLS, n (%)			
• General practitioner	291 (76%)	-	
• Metabolic bone disease outpatient clinic	40 (10.4%)	-	
• Nursing home / Long-term care unit physician	35 (9.1%)	-	-
• Rheumatology private physician	12 (3.1%)	-	
• Unknown	5 (1.3%)	-	
The patient has a general practitioner, n (%)			
• No	34 (8.7%)	12 (10.5%)	0.185
• Yes	301 (76.6%)	77 (67.5%)	
• Unknown	58 (14.8%)	25 (21.9%)	
Primary healthcare codification of osteoporosis / fragility fracture in patients with/without general practitioner, n (%)			
• No	93 (23.7%)	40 (35.1%)	0.106
• Yes	183 (46.6%)	37 (32.5%)	
• Unknown	59 (14.8%)	12 (10.5%)	
• Not applicable	58 (15%)	25 (21.9%)	

continues on the next pagw

TO 162 – TABLE 1. Continuation

Parameter	Intervention group N = 393	Control group (3) N = 114	p value
Patients with new fragility fractures after discharge, n (%)			
• 0	290 (73.8%)	74 (64.9%)	0,078
• 1	33 (8.4%)	12 (10.5%)	
• > 1	2 (0.5%)	0 (0%)	
• Unknown	58 (14.8%)	22 (19.3%)	
• Not applicable	10 (2.5%)	6 (5.3%)	
Hospital readmissions after discharge, n (%)			
• No	230 (58.5%)	64 (56.1%)	0.185
• Yes	110 (28%)	32 (28.1%)	
• Unknown	43 (10.9%)	12 (10.5%)	
• Not applicable	10 (2.5%)	6 (5.3%)	
Death, n (%)			
• No	298 (75.8%)	78 (68.4%)	0.061
• Yes	94 (23.9%)	36 31.6%)	
• Unknown	1 (0.3%)	0 (0%)	

Abbreviations: FLS - Fracture Liaison Service; IQR - interquartile range; SD - standard deviation;
* Patients who were referred to the outpatient clinic but did not attend. (1) Comorbid conditions associated with higher fracture risk: Diabetes mellitus, hyperparathyroidism, hyperthyroidism, hypogonadism, alcoholism, hypocalcaemia, vitamin D deficiency, chronic liver disease, malabsorption syndromes (celiac disease, Crohn's disease and ulcerative colitis), impaired balance and reflexes, stroke, dementia, decreased visual acuity, chronic obstructive pulmonary disease, chronic kidney disease, neurological diseases (depression, Parkinson's disease, Epilepsy, Peripheral neuropathy), cardiovascular diseases (congestive heart failure, arrhythmia, previous acute myocardial infarction, angina pectoris), inflammatory rheumatic diseases, HIV infection, prolonged immobilisation, haematological neoplasms (leukaemia, lymphoma, myeloma) and thalassaemia. (2) Drugs associated with higher fracture risk: anti-arrhythmics, anticoagulants, anticonvulsants, antidepressants, benzodiazepines, hypnotics and sedatives, diuretics, oral or inhaled glucocorticoids, H2 receptor inhibitors, proton pump inhibitors, aromatase inhibitors, anti-Parkinson's medication, vasodilators (nitrates) and thiazolidinedione. (3) Control group: the control group was made up of patients referred to the FLS program but who were not submitted to this evaluation due to nonfulfilment of requirements (e.g. missing analytical study) or due to their discharge prior to the FLS team being able to carry out this evaluation.

and mortality (3), but their success depends on its organization and coordination with other healthcare providers (2).

Objective: Describe the results of a single centre type A FLS care after its implementation.

Methods: We reviewed the clinical records of patients admitted to the orthopaedic department with a FF referenced to the FLS program between January 2020 to June 2022. Demographic and clinical data were collected from April to May 2023. We considered the intervention group (IG) patients effectively evaluated, and the control group (CG) those who were not. Demographic and clinical data were collected and presented as frequencies and mean (±SD) or median [interquartile range], as appropriate, for categorical and continuous variables, respectively.

Results: 507 patients with a FF were referred to the FLS program and most of them - 393 (77.5%) - were formally evaluated (IG). 79.6% were female, the mean age at fracture diagnosis was 83.1±8.8 years and the median follow-up time was 17 [11-24] months (table 1). Most patients in the IG had proximal femur FF - 391 (99.4%). In this group, antiresorptive therapy was recommended to 345 patients (87.8%): zoledronate in 173 (50.1%) of cases, alendronate in 126 (36.5%), and denosumab in 42 (12.2%); 4 patients didn't receive treatment recommendation as they missed the appointment for treatment decision. Of the 341 patients with therapeutic recommendation, 161 (47.2%) started the treatment: alendronate in 69 patients (54.8%), zoledronate in 68 (39.3%) and denosumab in 24 (57.1%). Currently, most patients are on treatment, but 1/3 of patients on alendronate and denosumab and half of patients on zoledronate have discontinued the treatment, besides those who died, most of the time for unknown reasons (table 1).

The IG and the CG were similar at baseline, but a difference was noted at the level of autonomy prior to the fracture (p=0.018); number of previous FF (p=0.002) and length of hospitalization (p=0.001), all lower in the CG. As expected, the frequency of antiresorptive therapy initiation was higher in the IG (p<0.001). No differences were found in the number of readmissions (p=0.185), new FF (p=0.078) or deaths (p=0.061) so far, although there was a favorable trend for the IG in the last two outcomes.

Discussion and **Conclusion:** Evaluating FLS outcomes, 30 months after its implementation, is important for improving its efficiency. One of the strengths of this report, unlike most studies, is that the CG is made up of patients with FF admitted at the same time frame as the IG.

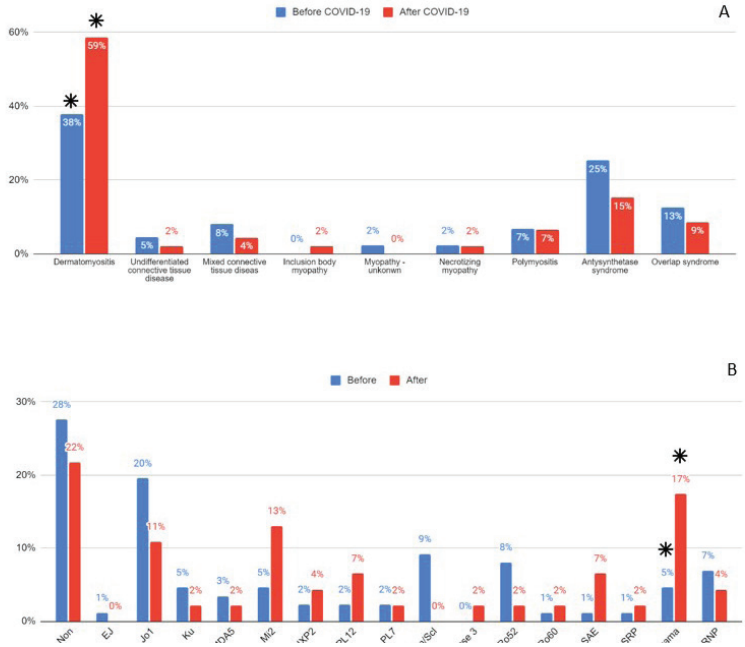
The results regarding initiation and maintenance of

therapy will reflect, at least partially, the circumstances observed at the date to which they refer, coinciding with the beginning and most critical period of the COVID-19 pandemic, with a reduced accessibility to health care (4,5).
FLS care was not associated with a significant reduction in subsequent fractures in studies with a follow-up ≤ 2 years, as in our case, and reduction in mortality was only observed in studies comparing before and after the introduction of FLS (3).
Although our results look promising, there is a need to optimize the coordination and organization of the FLS, namely in collaboration with the healthcare providers, and to ensure a regular monitoring of its outcomes.

167 - CANCER-ASSOCIATED MYOSITIS BEFORE AND AFTER THE COVID-19 PANDEMIC ONSET – A CHANGING TREND

Filipa Marques Costa[1,2], Bianca Paulo Correia[1,2], Matilde Bandeira[1,2], Eduardo Dourado[1,2], Ana Teresa Melo[1,2], Sofia C Barreira[1,2], Raquel Campanilho-Marques[1,2]
[1]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Cancer associated myositis (CAM) can occur in up to 25% of cases. The COVID-19 pandemic has been linked to a significant delay in cancer diagnosis, potentially impacting the incidence of paraneoplastic conditions. Furthermore, an increased incidence of CAM after the COVID-19 pandemic has been reported in Israel.
Objectives: To determine the incidence of CAM before and after the onset of the COVID-19 pandemic in a single center.
Methods: We included Inflammatory idiopathic myopathies (IIM) patients followed at a tertiary Rheumatology Department between June 2016 and June 2023. Patients were divided into two groups: those diagnosed with IIM before the COVID-19 pandemic (June 2016 to December 2019) and those diagnosed after the onset of the pandemic (January 2020 to June 2023). CAM was defined as the occurrence of neoplasia within three years (before or after) of the IIM diagnosis. Differences between groups were assessed using chi-square, Fisher's exact, or Mann-Whitney tests. Binomial logistic regression was used to find independent predictors of CAM. We considered likely associations when $p<0.10$, and definite associations when $p<0.05$.
Results: 133 patients were included, with a mean age of 50.4±19.7 years at the time of the IIM diagnosis, 64% (n=87) diagnosed prior and 36% (n=46) after the start of the pandemic. Demographic data were not statistically different between groups (p=0.680 for age, p=0.500 for sex).
For patients diagnosed before the COVID-19 pandemic, the most common IIM subtypes were dermatomyositis



TO 167 – Figure 1. Prevalence of IIM subtype (a) and autoantibodies (B) before and adter COVID-19. (*) Marks the variables with a statistically significant difference.

TO 167 – TABLE 1. Neoplasia and IIM characterization before and after the onset of the COVID- 19 pandemic

IIM date of diagnosis	Gender	Age	IIM	Neoplasia date of diagnosis	Neoplasia	Antibody
June 2016 – December 2019	M	88	Necrotizing myopathy	2021	Colon	-
January 2020 – June 2023	F	52	AASD	2022	Lung	Jo1
	F	54	Overlap syndrome	2020	Skin	PL12
	F	52	DM	2021	Breast	Mi2b
	F	50	DM	2022	Breast	TIF1γ
	F	87	DM	2019	Colon	TIF1γ
	F	67	DM	2022	Lung	TIF1γ
	M	60	DM	2023	Gastrointesti nal – non specified	TIF1γ
	F	50	DM	2023	Breast	TIF1γ

Abbreviations: AASD – antysynthetase syndrome; DM – dermatomyositis; IIM – inflammatory idiopathic myopathy; Jo-1 - anti-histidyl tRNA synthetase; PL12 - Anti-alanyl tRNA synthetase; TIF1γ - anti-transcription intermediary factor 1-γ

(DM, 27%, n=24) and anti-synthetase syndrome (ASSD, 25%, n=22), and the most frequent auto-antibody was anti-histidyl tRNA synthetase (anti-Jo1, 19%, n=17). After the COVID-19 pandemic onset, DM remained the most frequent IIM subtype (39%, n=19/46), but with significantly higher relative prevalence (p=0.030), and anti-TIF1γ was the most common auto-antibody, also with a significantly higher relative prevalence (17%, n=8/46 vs 4%, n=4/87, p=0.030, Figure 1).

The incidence of CAM was significantly higher after the COVID-19 pandemic (8 vs 1 new case in analogous period, p<0.001). Accordingly, there was a significantly higher proportion of CAM after the COVID-19 pandemic onset (17%, n=8/46 vs 1%, n=1/87, p<0.001).

Among the eight patients diagnosed with CAM after the pandemic onset, the most common IIM subtype was DM (75%, n=6), mainly in anti-TIF1γ-positive patients (62%, n=5).

Regarding all patients with CAM, they had more frequently anti-TIF1γ-positivity (p<0.001) and an IIM diagnosis occurring after the pandemic (p=0.001) than non-CAM-IIM patients.

Anti-TIF1γ-positivity (OR 9.998, 95% CI 1.904-52.500, p=0.007) and diagnosis after the COVID-19 pandemic onset (OR 13.24, 95% CI 1.483-118.261, p=0.021) were independent predictors of CAM among IIM patients.

Conclusion: In our cohort there was a significant increase in the incidence of CAM since the COVID-19 pandemic started. Accordingly, DM and anti-TIF1γ positivity became significantly more prevalent. IIM diagnosis occurring after the COVID-19 pandemic onset is associated with CAM, irrespective of age and sex. This adds to the previously reported increasing CAM incidence observed elsewhere, a worrying trend that may be related to worse management of non-COVID-19-related healthcare conditions, including cancer screening and early diagnosis since the pandemic started.

169 - DYSREGULATION OF CIRCULATING T FOLLICULAR CELLS, BUT NOT CLINICAL FEATURES, IS ASSOCIATED WITH ANTIPHOSPHOLIPID ANTIBODIES POSITIVITY IN PATIENTS WITH THROMBOTIC PRIMARY ANTIPHOSPHOLIPID SYNDROME

Pedro Gaspar[1, 2], Ana Rita Cruz-Machado[3, 4], Filipa Ribeiro[2], Vasco C Romão[3, 4], L Graca[2]

[1]Serviço de Medicina 2, Hospital de Santa Maria, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Lymphocyte Regulation Lab, Instituto de Medicina Molecular João Lobo Antunes, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia e Doenças Ósseas

Metabólicas, Hospital de Santa Maria, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [4]Rheumatology Research Unit, Instituto de Medicina Molecular João Lobo Antunes, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

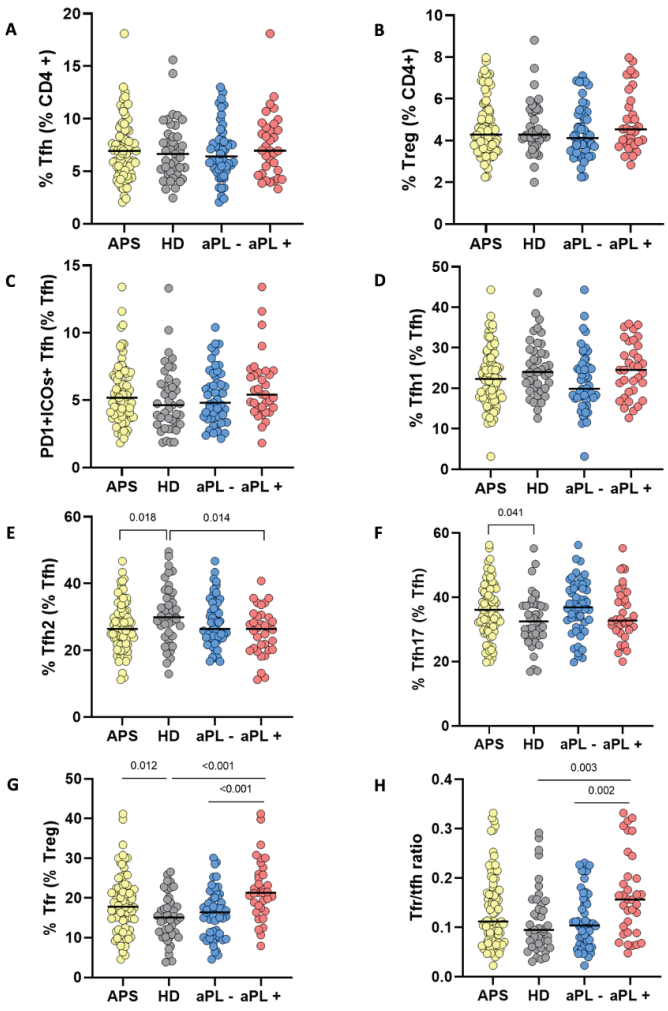
Background: Primary antiphospholipid syndrome (PAPS) is a systemic autoimmune disease caused by autoantibodies targeting protein-binding phospholipids (aPL). Follicular helper (Tfh) and regulatory T cells (Tfr) are critical for B cell maturation and antibody production in germinal centres, but their role in PAPS is scarcely studied. Whether aPL profile, including non-criteria aPL, dictates clinical, laboratory and immunological differences is unclear.

Objectives: To characterize PAPS patients clinically and immunologically based on current aPL status.

Methods: We prospectively recruited 85 adult thrombotic PAPS patients (fulfilling Sidney criteria) with and 41 age- and sex-matched healthy donors (HD). Regardless of lupus anticoagulant (LA) status, patients were categorized in two groups according to current aPL profile (anticardiolipin [ACL] IgM and/or IgG; ß2-glycoprotein [ß2GPI] IgM and/or IgG; phosphatidylserine/prothrombin complex [PS/PT] IgM and/or IgG; and Domain-1 of ß2GPI [D1- ß2GPI] IgG): aPL+, positive for at least one aPL (n=34); and aPL-, negative for all aPL, with previous and/or current positive for LA only (n=51). We considered aPL <30 UQ as negative (ELISA immunoassays). Tfh (CD4+FoxP3-CD25-CD45RO+CXCR5+), activated Tfh (PD1+ICOS+ Tfh), Tfh1-like (CXCR3+CCR6- Tfh), Tfh2-like (CXCR3-CCR6- Tfh), Tfh17-like (CXCR3+CCR6+ Tfh), Treg (CD4+CD25+FoxP3+), and Tfr (CD4+CD25+FoxP3+CXCR5+) cells were analysed by flow cytometry.

Results: Most patients were women (n=48, 56.5%), with a mean age at disease onset of 42 (49 – 32) years, with a mean disease duration of 4 (10 – 2) years (Table 1). Venous and arterial thrombosis occurred similarly as first manifestation (49.4%). Previous and current criteria-aPL positivity were as follows, respectively: LA (63.5%; 28.2%); ACL IgM and/or IgG (40.0%; 23.5%); ß2GPI IgM and/or IgG (34.1%; 27.1%). As for non-criteria-aPL positivity, PS/PT IgM and/or IgG were currently present in 25.9% of patients and D1-ß2GPI IgG in 18.8%. Sex distribution, age, disease duration, type of first thrombotic event, international normalized value, d-dimers, erythrocyte sedimentation rate and c-reactive protein were comparable between aPL+ and aPL- PAPS patients. Only activated partial thromboplastin time and gamma-globulin values were significantly increased in aPL+ compared to aPL- patients (Table 1). Circulating Tfh and Treg cells were comparable between HD and PAPS patients (Fig. 1A and 1B). Within the Tfh subset, activated Tfh cells were similarly distributed between aPL+ and aPL- patients (Fig. 1C). Among PAPS patients, the frequencies of Tfh1-like and Tfh17-like cells were comparable between aPL+, aPL- patients and HD, but Tfh2-like cells were decreased in aPL+ patients compared to HD patients (p=0.014) (Fig. 1D to F). In the regulatory compartment, circulating Tfr and Tfr/Tfh ratio were significantly increased in aPL+ patients compared to aPL- patients (p<0.001; p=0.0012) and HD (p<0.001; p=0.003) (Fig. 1G and 1H).

Conclusion: Despite clinical and laboratory similarities among thrombotic PAPS patients, imbalance of circulating Tfh and Tfr subsets changes according to aPL profile. Increased Tfr cells and Tfr/Tfh ratio might indicate ongoing humoral response in aPL+ patients. aPL- patients display a similar immunological profile compared to healthy individuals.



TO 169 – Figure 1. T Folicular cells subsets distribution

TO 169 – Table 1. Clinical and laboratory characterization of PAPS patients according to current antiphospholipid antibody profile.

	Total n=85	aPL positive n=34	aPL neg n=51	p-value
Demographics				
Female sex	48 (56.5)	23 (67.7)	25 (49.0)	0.090
Current age (y)	49 (57 – 43)	49 (57 – 43)	50 (57; 42)	0.904
Age onset (y)	42 (49 – 32)	42 (49 – 32)	42 (51 – 31)	0.774
Disease duration (y)	4 (10 – 2)	5 (12 – 1)	4 (6 – 2)	0.561
Clinical characterization				
Type of first thrombotic event				
Arterial	42 (49.4)	21 (61.8)	21 (41.2)	0.063
Venous	42 (49.4)	13 (38.2)	29 (56.9)	0.092
Microvascular	2 (2.4)	0	2 (3.9)	-
Previous aPL profile				
Lupus anticoagulant	54 (63.5)	18 (52.9)	36 (70.6)	0.112
Anticardiolipin (IgM/IgG)	34 (40.0)	27 (79.4)	7 (13.7)	<0.001
ß2-glycoprotein (IgM/IgG)	29 (34.1)	22 (64.7)	7 (13.7)	<0.001
Triple positive	13 (15.3)	12 (35.3)	1 (1.9)	<0.001
Current aPL profile				
Lupus anticoagulant	24 (28.2)	16 (47.1)	8 (15.7)	0.003
Anticardiolipin (IgM/IgG)	20 (23.5)	20 (58.8)	0	-
ß2-glycoprotein (IgM/IgG)	23 (27.1)	27 (67.7)	0	-
Triple positive	14 (16.5)	14 (41.2)	0	-
Phosphatidylserine/prothrombin complex (IgM/IgG)	22 (25.9)	22 (64.7)	0	-
Domain-1 of ß2GPI (IgG)	16 (18.8)	18 (47.1)	0	-
Laboratory				
Hb (g/dL)	14.5 (15.4 – 13.1)	14.0 (15.1 – 13.0)	14.5 (15.4 – 13.2)	0.498
MCV (fL)	88.6 (90.8 – 85.7)	89 (93.6 – 86)	88.3 (90.7 – 85.6)	0.644
RDW (CV%)	13.6 (15.5 – 13.3)	13.8 (14.5 – 13.2)	13.6 (15.5 – 13.3)	0.760
ESR (mm/1sth)	15 (26 – 8)	19 (31 – 12)	13.5 (23 – 7.5)	0.120
Leucocytes (x10^{6}/L)	6300 (7800 – 5000)	5750 (7400 - 4900	6500 (7900 – 5200)	0.286
Neutrophil (x10^{6}/L)	3700 (4840 – 2760)	3485 (5170 – 2760)	3910 (4840 – 2750)	0.673
Lymphocyte (x10^{6}/L)	1840 (2430 – 1480)	1790 (2170 – 1340)	1930 (2490 – 1580)	0.082
Neutrophil/lymphocyte ratio	2 (2.6 – 1.5)	2.02 (2.8 – 1.6)	1.91 (2.5 – 1.4)	0.866
D-dimer (ug/mL)	0.3 (0.4 – 0.2)	0.3 (0.4 – 0.2)	0.2 (0.4 – 0.2)	0.465
Aptt (s)	38.4 (43.7 – 34.2)	40.9 (48.3 – 35.9)	36.9 (39.9 – 32.4)	0.032
Fibrinogen (mg/dL)	300 (346 – 262)	318.5 (346 – 265)	292 (351 – 253)	0.615
INR	2.2 (2.9 – 1.3)	2.2 (2.7 – 1.2)	2.3 (2.9 – 1.5)	0.641
CRP (mg/dL)	0.2 (0.4 – 0.1)	0.2 (0.3 – 0.1)	0.2 (0.4 – 0.1)	0.705
Gamma-globulin (g/dL)	1.0 (1.2 – 0.9)	1.2 (1.3 – 1.0)	1.0 (1.2 – 0.8)	0.031

Data are shown as number (%) and median (IRQ). Continuous variables were compared using Mann-Whitney U test, and categorical variables using the Chi-square test or the Fisher's exact test when appropriate. aPL, antiphospholipid antibody; ESR, erythrocyte sedimentation rate; Hb, haemoglobin; INR, international normalized ratio; MCV, mean corpuscular volume; RDW, red cell distribution
with; S, seconds; Y, years.

170 - COGNITIVE DYSFUNCTION IN FIBROMYALGIA: PREVALENCE, IMPACT, AND CORRELATION WITH DISEASE ACTIVITY, SEVERITY, AND OTHER CLINICAL MANIFESTATIONS - A CASE-CONTROL STUDY

Sofia Ferreira Azevedo[1, 2], Inês Genrinho[1, 2, 3], Joana saldanha[2, 4], Inês Cunha[1]

[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal, [3]Unidade de Reumatologia, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [4]Medicina Física e de Reabilitação, Centro Hospitalar Entre Douro e Vouga, Aveiro, Portugal

Introduction: Fibromyalgia (FM) is one of the primary causes of chronic widespread pain. While pain is the distinguishing feature of the disease, there is a complex variety of symptoms that characterize this syndrome. Cognitive dysfunction is associated with FM at an uncertain level and represents some of the most severe manifestations of the disease.

The aim of our study is to assess the prevalence of cognitive impairment and its impact on fibromyalgia patients, as well as the relationship between cognitive impairment and pain, depression, and anxiety.

Methods and Materials: A case-control, single-centre study conducted on Portuguese patients diagnosed with FM according to the 2016 ACR criteria, and a matched healthy control group. Socio-demographic, chronic medication, and clinical data were collected. The Montreal Cognitive Assessment (MoCA) test, visual analogic scales (VAS) for pain and fatigue, the EQ5D-5L questionnaire, the Hospital Anxiety and Depression Scale (HADS), the Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT-F), and the Pittsburgh Sleep Quality Index were applied to both patients and controls. A FM Impact Questionnaire validated for the Portuguese population (FIQ-P) was also applicated the FM group. Cognitive impairment was defined as MoCA score < 26 points.

Statistical analysis was performed using SPSS software, and a significance threshold of $p \leq 0.05$ was set.

Results: A total of 66 participants were included in the study (47 FM patients and 19 controls), all of whom were women. The mean age was 47.56 ±10.68 years.

There were no significant differences between groups regarding education (primary and secondary school vs. high school and university). We found a higher frequency of cognitive impairment in FM patients [34 (72.44%) vs. 1 (5.26%); $p < 0.001$]. Other statistically significant differences were also observed for VAS pain and fatigue scores, HADS anxiety and depression, FACIT-F, EQ-5D-5L, and SARC-F questionnaires scores.

When evaluating the correlation between cognition and other clinical characteristics, we found an inverse weak correlation between MoCA scores and VAS pain ($r = -0.334$; $p = 0.02$), and between MoCA scores and HADS anxiety and depression scores ($r = -0.334$; $p = 0.02$ and $r = 0.477$; $p = 0.01$, respectively). We also found an inverse moderate correlation between FIQ-P scores and MoCA ($r = -0.431$; $p = 0.003$), and weak correlation between MoCA scores and SARC-F ($r = -0.373$; $p = 0.01$). Furthermore, we found a positive correlation between MoCA scores and FACIT-F ($r = 0.366$; $p = 0.01$), and moderate positive correlation between total EQ-5D-5L scores and MoCA ($\rho = 0.555$; $p < 0.001$). When comparing patients with mild cognitive impairment with patients without cognitive impairment, we found higher VAS pain scores ($p = 0.03$), lower total EQ5D-5L scores ($p < 0.001$), and higher FIQ-P scores ($p < 0.001$) in patients with cognitive impairment.

On multivariate analysis Cognitive disfunction was independently associated with higher FIQ-P scores [OR1.11 (CI1.1.04-1.20); p=0.03], and there were an independent inverse relation between depression and MoCA scores [OR-2.47 CI (-3.99- -0.95); p=0.002], and between MoCA and VAS pain [OR -0.41 CI(-0.65- -0.23); p<0.001].

Conclusion: Cognitive dysfunction is common in FM and appears to be related to disease activity, with higher levels of pain, depression, and major impact in patient's quality of life. These findings underscore the importance of screening for cognitive impairment and adopting a multidisciplinary approach in the management of these patients.

171 - DOES DENOSUMAB CONTRIBUTE TO FUNCTIONAL RECOVERY IN HIP FRACTURE PATIENTS?

Filipe Cunha Santos[1], Duarte Augusto[1], Ana Sofia Pinto[1], Sara Paiva Dinis [1], Nathalie Madeira[1], Cláudia Vaz[1, 2], Joana Fonseca Ferreira[1, 2]

[1]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [2]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal

Background: Hip fractures in osteoporotic patients often result in functional impairment and reduced quality of life. Treatment options such as bisphosphonates (BP) and denosumab (DSM) aim to improve bone health and prevent subsequent fractures. However, the impact of these medications on functional recovery following hip fracture remains unclear.

Objectives: To determine if DSM contributes to better functional recovery after osteoporotic hip fracture.

Material and Methods: This is a retrospective nested case-control study, including patients over 50 years, af-

TO 171 – TABLE 1.

	Treated with bisphosphonates (*n*=10)	Treated with denosumab (*n*=41)	*p* value
Age (years)	70.4±10.9	78.4±6.5	**0.004****
Sex, female *n* (%)	9 (90.0%)	36 (87.8%)	0.666
BMI (kg/m²)	25.9±3.2	24.2±3.5	0.174
Hand grip strength (kg)	20.6±7.4	19.2±4.2	0.421
Sarcopenia at baseline, *n* (%)	3 (30.0%)	12 (29.3%)	0.621
FRAX major (%)	19.0 (4.6; 37.0)	22.0 (5.4; 50.0)	0.585
FRAX minor (%)	7.4 (0.2; 29.0)	10.2 (1.7; 43.0)	0.233
T-score lumbar spine	-1.0±1.8	-1.8±1.2	0.083
T-score femoral neck	-1.7±0.7	-2.3±0.7	0.028
TUG			
Δ	-123.5 (-144.0; -11.0)	-104.0 (-141.0; -21.0)	0.887
52-weeks	13.7 (6.0; 76.0)	16.8 (9.0; 67.0)	0.342
Baseline	150.0 (19.0; 150.0)	150.0 (31.0-150.0)	0.817
5MWT			
Δ	-82.5±54.9	-74.7±32.4	0.561
52-weeks	16.2±15.1	16.2±10.7	0.984
Baseline	98.7±54.9	90.9±35.1	0.575
30STS			
Δ	9.8±2.8	9.0±3.5	0.509
52-weeks	12.0±4.1	9.3±3.6	0.047
Baseline	2.2±3.7	0.3±1.5	0.013

Values are mean±standard deviation or median (minimum-maximum)

30STS 30-seconds sit to stand, *5MWT* 5-meter walk test, *BMI* Body mass index, *SD* Standard deviation, *TUG* timed up and go

**indicates statistical significance.

ter operation for osteoporotic hip fracture and followed at our Fracture Liaison Service, which includes rehabilitation nurses and a nutritional appointment, between September 2019 and January 2023. We compared demographic and clinical features, as well as timed up and go (TUG), 5-meter walk test (5MWT) and 30-seconds sit to stand (30STS) at discharge and at 52-weeks between patients treated with DSM and those treated with BP (controls).

Results: Of 114 patients, 51 had one-year follow-up and were treated either with BP (n=10, 19.6%) or DSM (n=41, 80.4%). Patients treated with DSM were older than those treated with BP (78.4±6.5 vs. 70.4±10.9 years, p=0.004) and had a worse femoral neck T-score (-2.3±0.7 vs. -1.7±0.7, p=0.028). No statistically significant difference was found in the delta of TUG, 5MWT or 30STS at 52-weeks and baseline (p=0.887, p=0.561 and p=0.509, respectively). Mean 30STS at 52-weeks and baseline was statistically significant difference (p=0.047 and p=0.013, respectively).

Conclusions: No difference was found in functional recovery outcomes between patients treated with BP or DSM. Despite this, and even though the DSM group was older, they achieved a similar functional recovery. We can't ignore the limitations of this work, namely low number of included patients and inexistence of randomization.

REFERENCES:

Miedany, Yasser El et al. "Is there a potential dual effect of denosumab for treatment of osteoporosis and sarcopenia?." Clinical rheumatology vol. 40,10 (2021): 4225-4232. doi:10.1007/s10067-021-05757-w

173 - INTERSTITIAL LUNG DISEASE IN A COHORT OF 200 PATIENTS WITH IDIOPATHIC INFLAMMATORY MYOPATHIES

Bianca Paulo Correia[1,2], Filipa Marques Costa[1,2], Matilde Bandeira[1,2], Eduardo Dourado[1,2], Ana Teresa Melo[1,2], Nikita Khmelinskii[1,2], Sofia C Barreira[1,2], Raquel Campanilho-Marques[1,2]

[1]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Interstitial lung disease (ILD) is a common extramuscular manifestation of Idiopathic inflammatory myopathies (IIM). The presence of myositis-specific or myositis-associated autoantibodies (MSA or MAA) is a key diagnostic finding for IIM, and different autoantibodies have been associated with high rates of ILD.

Objectives: This work aims to describe ILD's prevalence and its subtypes and find predictors of lung involvement in a cohort of patients with IIM.

Methods: We retrospectively included all patients followed at our Rheumatology Department, from June 2016 to June 2023, with a confirmed IIM diagnosis. Associations between the different variables were tested using Chi-Square, Fischer's Exact, Student's t or Mann-Whitney tests, as appropriate. Multivariate analysis to identify predictors of ILD was performed using a binary logistic regression model.

Results: 200 patients were included, of whom 148 (74.0%) were females, with a median age at diagnosis of 47.5 (interquartile range, IQR 20.0) years and a median disease duration of 5.0 (IQR 7.0) years. Sixty-four patients (32.0%) had ILD based on high-resolution computed tomography scans (HRCT) performed anytime during the follow-up. The most common diagnosis among patients with ILD was antisynthetase syndrome [34/64 (53.1%)], followed by dermatomyositis [13/64 (20.3%)]. Of these, forty-six patients (71.9%) exhibited a radiological pattern compatible with non-specific interstitial pneumonia (NSIP), of whom seven (15.2%) had fibrotic NSIP. Eight patients [8/64 (12.5%)] had usual interstitial pneumonia (UIP), while the remaining had organizing pneumonia (OP) [5/64 (7.8%)], OP/NSIP overlap [2/64 (3.1%)], acute interstitial pneumonia [1/64 (1.6%)], desquamative interstitial pneumonia [1/64 (1.6%)], or lymphocytic interstitial pneumonia [1/64 (1.6%)]. The prevalence of different subtypes of ILD according to the IIM diagnosis is shown in Table 1. Compared with IIM patients without ILD, those with ILD had a higher prevalence of mechanic's hands [19/63 (30.2%) vs 15/135 (11.1%), p<0.001], arthritis [39/62 (62.9%) vs 37/135 (27.4%), p<0.001], Raynaud's phenomenon [29/63 (46.0%) vs 40/135 (29.6%), p=0.024], and gastroesophageal reflux disease [11/63

TO 173 – TABLE 1. Clinical and serological features of patients with and without interstitial lung disease

	Patients with ILD (n = 64)	Patients without ILD (n = 136)	Univariate analysis (p-value)
Age at diagnosis, median (IQR)	50.5 (25.8)	46.5 (33.5)	p = 0.165
Disease duration (in years), median (IQR)	4.0 (3.0)	5.0 (8.0)	p = 0.072
Female, n (%)	42 (65.6)	106 (77.9)	p = 0.064
Mortality, n (%)	1 (1.6)	7 (5.1)	p = 0.440
Diagnosis	ILD subtypes		
Antisynthetase syndrome (N = 34)	NSIP, n (%): 26 (76.5) UIP, n (%): 5 (14.7) OP, n (%): 1 (2.9) OP/NSIP overlap, n (%): 1 (2.9) LIP, n (%): 1 (2.9)	NA	NA
Dermatomyositis (N = 13)	NSIP, n (%): 8 (61.5) OP, n (%): 3 (23.1) OP/NSIP overlap, n (%): 1 (7.7) AIP, n (%): 1 (7.7)	NA	NA
Polymyositis (N = 2)	NSIP, n (%): 1 (50.0) DIP, n (%): 1 (50.0)	NA	NA
Overlap syndrome (N = 10)	NSIP, n (%): 6 (60.0) UIP, n (%): 3 (30.0) OP, n (%): 1 (10.0)	NA	NA
Undifferentiated connective tissue disease (N = 3)	NSIP, n (%): 3 (100.0)	NA	NA
Mixed connective tissue disease (N = 2)	NSIP, n (%): 2 (100.0)	NA	NA
Musculoskeletal involvement			
MMT8 last value, median (IQR)	150.0 (8.0)	150.0 (16.0)	p = 0.301
Maximum	150.0	150.0	
Minimum	100.0	86.0	
Arthritis, n/N (%)	39/62 (62.9)	37/135 (27.4)	p < 0.001
Skin involvement			
Highest modified DAS skin, median (IQR)	0.0 (2.0)	0.0 (2.0)	p = 0.855
Calcinosis, n/N (%)	3/63 (4.8)	12/135 (8.9)	p = 0.396
Mechanic's hands, n/N (%)	19/63 (30.2)	15/135 (11.1)	p < 0.001
Raynaud's phenomenon, n/N (%)	29/63 (46.0)	40/135 (29.6)	p = 0.024
Internal organ involvement			
Heart involvement	2/64 (3.1)	4/136 (2.9)	p = 1.000
Esophageal involvement, n/N (%)	11/63 (17.5)	6/135 (4.4)	p = 0.002
Neoplasia			
Diagnosis of neoplasia, n/N (%)	5/64 (7.8)	12/136 (8.8)	p = 0.811
Antibodies	N = 60	N = 98	
Anti-Ro52, n (%)	4 (6.7)	9 (9.2)	p = 0.768
Anti-Ro60, n (%)	1 (1.7)	1 (1.0)	p = 1.000
Anti-Jo1, n (%)	25 (41.7)	4 (4.1)	p < 0.001
Anti-PL7, n (%)	4 (6.7)	3 (3.1)	p = 0.428
Anti-PL12, n (%)	6 (10.0)	1 (1.0)	p = 0.012
Anti-EJ, n (%)	1 (1.7)	0 (0.0)	p = 0.380
Anti-PM/Scl100, n (%)	1 (1.7)	3 (3.1)	p = 1.000
Anti-PM/Scl75, n (%)	5 (8.3)	4 (4.1)	p = 0.302
Anti-Ku, n (%)	1 (1.7)	7 (7.1)	p = 0.157
Anti-U1-RNP, n (%)	1 (1.7)	14 (14.3)	p = 0.009
Anti-Mi2a, n (%)	0 (0.0)	7 (7.1)	p = 0.045

continues on the next pagw

TO 173 – TABLE 1. Continuation

	Patients with ILD (n = 64)	Patients without ILD (n = 136)	Univariate analysis (p-value)
Anti-Mi2b, n (%)	0 (0.0)	13 (13.3)	p = 0.002
Anti-TIF1γ, n (%)	2 (3.3)	10 (10.2)	p = 0.134
Anti-MDA5, n (%)	7 (11.7)	3 (3.1)	p = 0.043
Anti-NXP2, n (%)	0 (0.0)	4 (4.1)	p = 0.298
Anti-SAE1, n (%)	1 (1.7)	5 (5.1)	p = 0.409
Anti-SRP, n (%)	0 (0.0)	6 (6.1)	p = 0.083
ANA, n/N (%)	47/60 (78.3)	66/115 (57.4)	p = 0.006

ILD: interstitial lung disease; n: number of patients positive for the variable of interest; N: number of patients without missing information regarding the variable of interest; NA: not applicable; NSIP: non-specific interstitial pneumonia; OP: organizing pneumonia; UIP: usual interstitial pneumonia; AIP: acute interstitial pneumonia; LIP: lymphocytic interstitial pneumonia; DIP: desquamative interstitial pneumonia. MMT8, manual muscle testing; DAS, disease activity score; Jo1, histidyl tRNA synthetase; PL7, threonyl tRNA synthetase; PL12, anti-alanyl tRNA synthetase; EJ, glycyl tRNA synthetase; Pm/Scl, polymyositis/scleroderma; RNP, ribonucleoprotein; TIF1g, transcription intermediary factor 1-gamma; MDA5, melanoma differentiation-associated gene 5; NXP2, nuclear matrix protein 2; SAE, small ubiquitin-like modifier activating enzyRo52/me; SRP, signal recognition particle; ANA, anti- nuclear antibodies.
All statistically significant differences between groups are shown in bold.

(17.5%) vs 6/135 (4.4%), p=0.002].
Anti-Jo1 [25/60 (41.7%) vs 4/98 (4.1%), p<0.001], anti-PL12 [6/60 (10.0%) vs 1/98 (1.0%), p=0.012], anti-MDA-5 [7/60 (11.7%) vs 3/98 (3.1%), p=0.043], and anti-nuclear (ANA) antibodies [47/60 (78.3%) vs 66/115 (57.4%), p=0.006] were more commonly identified in patients with ILD. In contrast, anti-Mi2 and anti-U1-RNP antibodies were less frequent in the ILD group (Table 1).
In the multivariate analysis, a total of 141 patients were included. Anti-Jo1 positivity (OR 8.78, 95%CI: 2.36-32.62, p=0.001), mechanic's hands (OR 4.89, 95%CI: 1.20-19.89, p=0.027), and gastroesophageal reflux (OR 8.31, 95%CI: 1.54-44.89, p=0.014) were identified as independent predictors of ILD regardless of sex, age at diagnosis, arthritis and Raynaud's phenomenon. Anti-U1-RNP antibody was identified as a protective factor for ILD (OR 0.04, 95%CI: 0.003-0.427, p=0.008).
Conclusions: In a large cohort of 200 patients with IIM, 32.0% had ILD, with NSIP being the most frequent pattern identified on HRCT scans. Anti-Jo1 antibody positivity, mechanic's hands, and gastroesophageal reflux disease were identified as independent predictors of ILD in our cohort. Anti-U1-RNP antibody positivity was identified as a protective factor for ILD.

175 - POLYMYALGIA RHEUMATICA AFTER COVID-19 VACCINATION: A GLOBAL PHARMACOVIGILANCE STUDY USING EUDRAVIGILANCE DATABASE

C. Pinto Oliveira[1, 2], Sofia Ferreira Azevedo[1, 2], Carolina Vilafanha[1, 2], Ana Rita Prata[1, 2], Anabela Barcelos[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar do Baixo Vouga, E.P.E., Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Introduction: Polymyalgia rheumatica (PMR) is an inflammatory disease characterized by pain and stiffness in the shoulder and pelvic girdle of older individuals. Although its cause remains unknown, environmental triggers, such as vaccination, might play a role (1). It has been postulated that molecular mimicry and certain vaccine adjuvants can induce autoimmune syndromes after vaccination (2). Recently, the novel coronavirus (COVID-19) pandemic led to rapid development of new vaccines. Potential side effects of vaccines have been notified on global pharmacovigilance databases, such as EudraVigilance and VigiBase. These databases are useful for detecting and analyzing potential risks of medicines after their placement on the market (3), ultimately contributing for better public health. In fact, a study by Montastruc et al, using reports in VigiBase until 30 June, 2021, found 290 potential PMR cases following COVID-19 vaccination (4).
Objective: To analyze reported cases of PMR following COVID-19 vaccination, using the EudraVigilance database.
Methods: Accessing the EudraVigilance database, we retrieved Individual Case Safety Reports (ICSRs) with a reported suspected reaction of PMR from 1 January, 2022 to 1 May, 2023, attributed to COVID-19 vaccines approved by the European Medicines Agency. A detailed analysis of each ICSR was carried out to eliminate potential duplicates or cases of aggravated preexisting PMR. We then performed a descriptive analysis of the available data, including sociodemographic variables, respective outcome and severity of suspected adverse reactions.
Results: Of the 708 876 reports in EudraVigilance concerning adverse reactions associated with COVID-19 vaccines, between 1 January, 2022 and 1 May, 2023,

TO 175 – TABLE 1. Continuation

Characteristic	N (%)
Reporting region	
European Economic Area	548 (72.8)
Non European Economic Area	205 (27.2)
Reporter qualification	
Healthcare professional	357 (47.4)
Non healthcare professional	396 (52.6)
COVID-19 vaccine type	
mRNA vaccine	628 (83.4)
Viral vector vaccine	125 (16.6)
Sex	
Female	398 (52.9)
Male	347 (46.1)
Not specified	8 (1.1)
Age range	
18 to 64 years	266 (35.3)
65 to 85 years	417 (55.4)
Older than 85 years	30 (4.0)
Not specified	40 (5.3)
Seriousness	653 (86.7)
Seriousness criteria	
Resulting in other medically important condition	505 (67.1)
Requiring or prolonging hospitalization	113 (15)
Resulting in disability/incapacity	110 (14.6)
Life threatening	5 (0.7)
Resulting in death	1 (0.1)
Reaction outcome at time of report	
Not recovered	380 (50.5)
Recovering	188 (25)
Recovered	43 (5.7)
Recovered with sequelae	28 (3.7)
Fatal	1 (0.1)
Unknown	113 (15)
Suspected drug	
Only COVID-19 vaccine	712 (94.6)
Other suspected drug	41 (5.4)

948 (0.13%) included suspected PMR. After exclusion of duplicates / cases of aggravated PMR, we found 753 reports (table 1). Most were performed by non-healthcare professionals (n=396; 52.6%), within the European Economic Area (n=548; 72.8%). The majority of cases concerned women (n=398; 52.9) and individuals within an age range of 65 to 85 years (n=417; 55.4%). Concerning types of vaccines, mRNA vaccines were more frequently involved (n=628; 83.4%) than viral vector vaccines. At least one criterion of seriousness was reported in 653 cases (86.7%).

Conclusion: Using EudraVigilance database, we found a relatively small number of potential PMR cases following COVID-19 vaccination, in comparison with the magnitude of other adverse reactions. More data is paramount to better understand potential interactions between COVID-19 vaccines and the development of PMR.

REFERENCES:

1. Falsetti, Paolo, et al. "Polymyalgia rheumatica following infective triggersor vaccinations: a different subset of disease? " Reumatologia/Rheumatology 58.2 (2020): 76-80.
2. Chen, Yue, et al. "New-onset autoimmune phenomena post-COVID-vaccination." Immunology 165.4 (2022): 386-401.
3. Postigo, Rodrigo, et al. "EudraVigilance medicines safety database: publicly accessible data for research and public health protection." Drug safety 41 (2018): 665-675.
4. Mettler, Camille, et al. "Risk of giant cell arteritis and polymyalgia rheumatica following COVID-19 vaccination: a global pharmacovigilance study." Rheumatology 61.2 (2022): 865-867.

176 - CARDIOVASCULAR DISEASE IN PSORIATIC ARTHRITIS: IS HYPERURICEMIA AN INDEPENDENT RISK FACTOR?

Pedro Miguel Teixeira[1, 2], Carolina Vilafanha[1, 2], Susana Silva[1, 2], Sofia Ferreira Azevedo[1, 2], C. Pinto Oliveira[1, 2], Carolina Mazeda[1, 2], Gisela Eugénio[1, 2], Anabela Barcelos[1, 2]

[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Background: Cardiovascular disease significantly contributes to the heightened morbidity and mortality observed among patients with psoriatic arthritis (PsA). Hyperuricemia (HUC) is among the metabolic abnormalities found in these patients, potentially linked to increased cell turnover and chronic inflammation, but its role as a major cardiovascular risk remains unclear.1

Objectives: To characterize the prevalence of HUC in PsA patients, associated risk factors and its relationship with cardiovascular disease.

Methods: A unicentric, cross-sectional study including adult patients with PsA (who fulfilled the Classification for Psoriatic Arthritis (CASPAR) criteria) from a Rheumatology outpatient clinic was performed. Data on demographic, clinical, comorbidities, pharmacologic treatments and laboratory variables were collected. HUC was defined as serum uric acid levels exceeding 6.8 mg/dL, and patients were categorized into two groups accordingly. Univariate analysis and subsequent multivariate logistic regression were employed to assess potential factors associated with HUC, and a p-value ≤0.05 was considered statistically significant.

Results: A total of 166 patients were included: 66 (39.8%) were women and the mean age was 56,0±11,9 years. One hundred and sixty-three (98%) had personal history of psoriasis, 77 (46%) were on a csDMARD alone, 44 (27%) on a bDMARD alone and 22 (13%) on combined csDMARD and bDMARD. HUC was present in 46 (27.7%) of PsA patients and was associated with higher rates of hypertension (p=0.005), diabetes mellitus (p=0.016), heart failure (p<0.001), ischemic heart disease (p<0.001), atrial fibrillation (p<0.001), peripheric arterial disease (p<0.001), and stroke (p<0.001).

TO 176 – TABLE 1. Demographics, clinical and commodities associated with hyperuricemia in PsA patients

	With HUC (n=46)	Without HUC (n=120)	Significance p
Mean age (SD) (years)	60.5 (11.1)	54.32 (11.8)	0.002
Women, n (%)	7 (15.2%)	59 (49.2%)	<0.001
Body mass index (SD) (kg/m^2)	28.7 (4.1)	28.3 (4.6)	0.612
PsA disease duration (SD) (years)	12.1 (1.4)	11.3 (0.7)	0.573
DAPSA, mean (SD)	10,2 (1,9)	8,00 (0.8)	0.296
Serum uric acid (mg/dL) (SD)	7.6 (0.55)	5.1 (0.61)	<0.001
Ongoing treatment			
No treatment, n (%)	4 (8.7%)	9 (7.5%)	0.797
NSAIDs, n (%)	4 (8.7%)	23 (19.2%)	0.102
PDN daily dose ≥ 5mg, n (%)	10 (21.7%)	18 (15.0%)	0.299
csDMARD alone, n (%)	20 (43.5%)	57 (47.5%)	0.644
bDMARD alone, n (%)	13 (28.3%)	31 (25.8%)	0.751
csDMARD+bDMARD, n (%)	7 (15.2%)	15 (12.5%)	0.644
Hypertension, n (%)	26 (56.5%)	39 (32.5%)	0.005
Dyslipidaemia, n (%)	28 (60.9%)	55 (45.8%)	0.083
Obesity, n (%)	15 (32.6%)	38 (35.2%)	0.758
Diabetes mellitus, n (%)	14 (30.4%)	17 (14.2%)	0.016
Heart failure, n (%)	10 (21.7%)	4 (3.3%)	<0.001
Ischemic heart disease, n (%)	12 (26.1%)	2 (1.7%)	<0.001
Atrial fibrillation, n (%)	15 (32.6%)	6 (5.0%)	<0.001
Arterial peripheral disease, n (%)	9 (19.6%)	2 (1.7%)	<0.001
Stroke, n (%)	6 (13.0%)	1 (0.8%)	<0.001
Nephrolithiasis, n (%)	5 (10.9%)	4 (3.3%)	0.055
Current or previous smoking, n (%)	11 (23.9%)	24 (20.0%)	0.580
Alcohol intake >30g/day, n (%)	11 (23.9%)	9 (7.8%)	0.005

PsA: Psoriatic Arthritis; HUC: hyperuricemia; SD: standard deviation; DAPSA: Disease Activity in Psoriatic Arthritis; NSAIDs: non-steroidal anti-inflammatory drugs; PDN: Prednisolone; csDMARD: conventional synthetic disease-modifying anti-rheumatic drugs; bDMARD: biologic disease-modifying anti-rheumatic drugs

Furthermore, there was also a positive association with older age (p=0.002), male sex (p<0.001) and alcohol intake (p=0.005). In the multivariate-adjustment analysis, HUC was independently associated with heart failure (OR 3.23 (1.39-11.99); p=0.021) and atrial fibrillation (OR 3.98 (1.61-13.38) p<0.001).

Conclusion: In our cohort of patients with PsA, HUC was shown to be an independent risk factor for cardiovascular disease, including heart failure and atrial fibrillation. However, it is yet uncertain whether implementing strategies aimed at normalizing uric acid levels in patients with PsA will lead to better cardiovascular outcomes. Prospective studies are necessary to determine the significance of HUC as a biomarker for cardiovascular diseases.

REFERENCES

1. Tripolino, Cesare, et al. "Hyperuricemia in psoriatic arthritis: epidemiology, pathophysiology, and clinical implications." Frontiers in Medicine 8 (2021): 737573.

177 - ABSENCE OF SCLERODERMA PATTERN IN NAILFOLD CAPILLAROSCOPIC: A DISTINCT SYSTEMIC SCLEROSIS SUBSET?

Ana Isabel Maduro[1], André Saraiva[1], Marcelo Neto[1], Fernando Albuquerque[1], Maria João Salvador[1, 2], Tânia Santiago[1, 2]

[1]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal

Background: Peripheral microangiopathy is a hallmark of systemic sclerosis (SSc) and can be detected by nailfold videocapillaroscopy (NVC). Scleroderma-type patterns have been reported in up to 90% of SSc patients with clinically significant disease and are associated with more severe disease. However, data about patients with normal or non-specific abnormalities in NVC is scarce.

Objective: To describe the demographic, clinical and immunological features in SSc patients with normal or non-specific abnormalities in NVC and to compare them to those with scleroderma NVC patterns.

Materials and Methods: Cross-sectional single-center study including SSc patients fulfilling the ACR'1980, ACR/EULAR'2013 or LeRoy's classification criteria, followed in our University Hospital. Demographic features; SSc subtype [Very Early Diagnosis of Systemic Sclerosis (VEDOSS), Limited, Diffuse]; cumulative clinical manifestations [Raynaud phenomenon, telangiectasias, skin thickening assessed by modified Rodnan Skin Score (mRSS), digital ulcers or pitting scars, articular involvement, myositis, gastrointestinal and renal involvement, interstitial lung disease (ILD) and pulmonary arterial hypertension]; immunological

TO 177 – TABLE 1. Frequency of SSc-associated clinical manifestations and immunological features in SSc patients with normal/nonspecific vs specific videocapillaroscopy pattern

	Whole cohort n=158 (100%)	Normal/Nonspecific NVC n=74 (46.8%)	Specific NVC n = 84 (53.2%)	p-value
Age at diagnosis, years (median IQR)	52.0 (15.3)	55.0 (19.3)	51.5 (15.8)	0.090
Disease duration, years (median IQR)	11.1 (11.0)	9.2 (7.5)	11.6 (12.1)	0.249
Sex (n [%])				
- Female	136 (86.1)	63 (85.1)	73 (86.9)	0.749
- Male	22 (13.9)	11 (14.9)	11 (13.1)	
Subtype (n [%])				
- VEDOSS	43 (27.2)	31 (41.9)	12 (14.3)	<0.001
- Limited cutaneous	85 (53.8)	38 (51.4)	47 (56.0)	0.563
- Diffuse cutaneous	30 (19.0)	5 (6.8)	25 (29.8)	<0.001
Clinical features (n [%])				
- Raynaud´s Phenomenon	149 (94.3)	67 (90.5)	82 (97.6)	0.084
- Skin thickening (mRSS)	4.0 (9.0)	3.12 (4.0)	4.0 (12.0)	<0.001
- Telangiectasis	48 (30.4)	17 (23.0)	31 (36.9)	0.057
- Digital ulcers or pitting scars	45 (28.5)	11 (14.9)	34 (40.5)	<0.001
- Articular involvement	38 (24.1)	20 (27.0)	18 (21.4)	0.411
- Myositis	4 (2.5)	1 (1.4)	3 (3.6)	0.623
- Gastrointestinal involvement	44 (27.8)	12 (16.2)	32 (38.1)	0.002
- Renal involvement	1 (0.6)	0 (0)	1 (1.2)	1.000
- Interstitial lung disease	39 (24.7)	11 (14.9)	28 (33.3)	0.007
- Pulmonar arterial hypertension	22 (13.9)	7 (9.5)	15 (17.9)	0.128
Laboratory features (n [%])				
- Antinuclear antibodies	155 (98.1)	73 (98.6)	82 (97.6)	1.000
- Anti-centromere	93 (58.9)	46 (62.2)	47 (56.0)	0.429
- Anti-topoisomerase I	35 (22.2)	10 (13.5)	25 (29.8)	0.014
- Anti-RNA polymerase III	4 (2.5)	2 (2.7)	2 (2.4)	1.000
- C Reactive Protein (median IQR)	0.6 (0.6)	0.2 (0.4)	0.3 (0.7)	0.350
- Erythrocyte sedimentation rate (median IQR)	14 (16.0)	12 (16.0)	15 (14.8)	0.117
Respiratory Function Tests (median IQR)				
- DLCO (mmol/min/kpa)	89 (28.2)	93.2 (25.7)	85.4 (30)	0.015
- FVC (%)	100 (27.4)	106.4 (23.1)	98.8 (24.3)	0.010

IQR: Interquartile range; VEDOSS: Very Early Diagnosis of Systemic Sclerosis (VEDOSS DLCO: Diffusing lung capacity for carbon monoxide; FVC: Forced vital capacity; Interstitial lung disease as assessed by high-resolution computed tomography; pulmonary arterial hypertension as based on a pulmonary artery systolic pressure ³ 35mmHg, estimated by echocardiography or evaluated by right heart catheterisation.

characteristics [positivity to antinuclear antibodies, anti-centromere (ACA), anti-topoisomerase I (anti-Scl70) and anti-ribonucleic acid polymerase III (anti-RNA-pol III)]; and respiratory function tests [diffusing lung capacity for carbon monoxide (DLCO) and forced vital capacity (FVC)] were collected. The chi-squared test/Fisher test and Mann-Whitney U test were used to compare the groups. P-value <0.05 was considered statistically significant.

Results: 158 patients were included. A normal or non-specific NVC pattern was found in 13 (8.2%) and 61 (38.6%) patients, respectively, in a total of 74 (46.8%) patients. Of these, 85.1% (n=63) were female, and the median age was 55.0 (IQR 19.3) years. VEDOSS was the SSc subtype more prevalent in the normal/nonspecific NVC pattern group (41.9% vs 14.3%, p<0.001), and the diffuse subtype was the least frequent (6.8 vs 29.8%, p<0.001). Regarding clinical manifestations, it was found a statistically significant lower proportion of digital ulcers or pitting scars (14.9% vs 40.5%, p<0.001), gastrointestinal involvement (16.2% vs 38.1%, p=0.002), ILD (14.9% vs 33.3%, p=0.007), and a lower median in mRSS [3.12 (IQR 4.0) vs 4.0 (IQR 12.0)] in this group. Concerning specific antibodies, a positive titer of anti-Scl70 was less common in SSc patients with normal/nonspecific NVC pattern (13.5 vs 29.8%, p=0.014). About respiratory function tests, there was a statistically significant increase in the median of DLCO [93.2 (IQR 25.7) vs 85.4 (IQR 30) mmol/min/kPa, p=0.015] and FVC [106.4 (IQR 23.1) vs 98.8 (24.3), p=0.01]. No differences in demographic features, disease duration, Raynaud's phenomenon, telangiectasis, articular and renal involvement, myositis, anti-centromere and anti-RNA polymerase III antibodies were found between the groups.

Conclusion: In this cohort, we found a normal/non-specific NVC pattern in 74 (46.8%) patients. These patients represent a subset of SSc with a significantly lower frequency of organ involvement than those with a SSc-specific NVC pattern. VEDOSS was the SSc subtype most associated with normal/non-specific abnormalities. Patients with normal/non-specific NVC pattern may have a lower risk of organ involvement and less severe disease, but prospective studies are needed.

179 - WHAT IS THE CLINICAL RELEVANCE OF RHEUMATOID FACTOR IN PRIMARY SJÖGREN'S SYNDROME?

Ana Isabel Maduro[1], Ana Rita Prata[2], Marlene Sousa[1], Liliana Saraiva[1], Helena Assunção[3], Mariana Luis[1, 4], Tânia Santiago[1, 4], Ines L[1, 5], JAP da Silva[1, 4]

[1]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar do Baixo Vouga, E.P.E., Aveiro, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal, [4]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal, [5]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal

Background: Primary Sjögren's syndrome (pSS) is a complex and heterogeneous autoimmune disorder characterised by a wide spectrum of glandular and extraglandular features. Rheumatoid factor (RF) is particularly common in patients with pSS, and its prevalence varies from 36 to 74%, depending on the study and population being evaluated.

Objective: To evaluate the RF prevalence and clinical and serological correlations in a cohort of pSS patients.

Methods: Cross-sectional single-center study including pSS patients fulfilling the ACR/EULAR 2016 and/or AECG 2002 classification criteria followed in our University Hospital. Demographic features and cumulative clinical manifestations, comorbidities and immunological characteristics were collected and compared between pSS patients with and without RF. Sjögren's syndrome disease activity index (ESSDAI) score of the last visit was considered to assess disease activity. The continuous variables were described as mean or median, according to distribution. Categorical variables were expressed in percentages. The chi-squared test or Fisher test and the independent sample t-test or Mann–Whitney U-test were used for comparisons of categorical variables and continuous variables, as appropriate. A p-value <0.05 was considered statistically significant.

Results: 140 pSS patients were included. Of these, 94.3% (n=132) were female, the mean age was 60.0 (± 13.2) years, and the mean age at diagnosis was 53.2 (±15.6) years. Median ESSDAI score was 0.9 (IQR 2.9). RF was found in 53% (n=74) of the patients. More frequent clinical features in the RF group were: Parotitis (21.6% vs 6.1%, p=0.009); parotid gland enlargement (16.2% vs 4.5%, p=0.026); arthritis (21.6% vs 6.1%, p=0.009). Patients with RF showed a higher frequency of hypergammaglobulinemia (79.7% vs 40.0%, p<0.001), hypocomplementemia (31.1% vs 13.8%, p=0.016), anti-Ro/SSA60 (98.6% vs 84.8%, p=0.002), anti-La/SSB (68.9% vs 24.2%, p=<0.001) and high titer of antinuclear antibodies (titer >1:1280). No significant difference was found in demographic features, sicca complaints, comorbidities and ESSDAI score.

Conclusion: A positive FR was associated with a pSS phenotype with more frequent systemic clinical features and immunological markers. This subgroup may require more frequent monitoring and treatment for systemic disease.

TO 179 – TABLE 1. Frequency of pSS-associated clinical manifestations, comorbidities, and serological characteristics in patients with and without rheumatoid factor

	Whole Cohort n=140 (100%)	Positive RF n=74 (53%)	Negative RF n=66 (47%)	p-value
Age at time of study mean (SD)	60.0 (13.2)	58.4 (14.0)	61.8 (12.2)	0.125
Age at diagnosis mean (SD)	53.2 (15.6)	50.7 (16.3)	56.0 (14.4)	0.057
Sex (n [%])				
- Female	132 (94.3)	71 (95.9)	61 (92.4)	0.475
- Male	8 (5.7)	3 (4.1)	5 (7.6)	
pSS clinical features [n (%)]				
- Xerophthalmia	138 (98.6)	73 (98.6)	65 (98.5)	1.000
- Xerostomia	131 (93.6)	72 (97.3)	59 (89.4)	0.084
- Parotitis	20 (14.3)	16 (21.6)	4 (6.1)	0.009
- Parotid gland enlargement	15 (10.7)	12 (16.2)	3 (4.5)	0.026
- Lymphadenopathy	9 (6.4)	6 (8.1)	3 (4.5)	0.500
- Raynaud's phenomenon	28 (20.0)	15 (20.3)	13 (19.7)	0.933
- Arthritis	20 (14.3)	16 (21.6)	4 (6.1)	0.009
- Pulmonary involvement	16 (11.4)	10 (13.5)	6 (9.1)	0.412
- Neurological involvement	5 (3.6)	1 (1.4)	4 (6.1)	0.188
- Renal involvement	5 (3.6)	3 (4.1)	2 (3.0)	0.824
- Cutaneous involvement	24 (17.1)	13 (17.6)	11 (16.7)	0.888
Comorbidities [(%)]				
- Lymphoma	8 (5.7)	5 (6.8)	3 (4.5)	0.574
- Fibromyalgia	41 (23.9)	20 (27.0)	21 (31.8)	0.580
- Depression	52 (37.1)	23 (31.1)	29 (43.9)	0.116
- Osteoarthritis	38 (27.1)	19 (28.8)	19 (28.8)	0.679
- Osteoporosis	21 (15.0)	11 (14.9)	10 (15.2)	0.962
Laboratory features [n (%)]				
- ANAs	139 (99.3)	74 (100.0)	65 (98.5)	0.471
*ANAs, titer >1:1280	34 (28.3)	25 (40.3)	9 (15.5)	0.003
- Leukopenia (<3.5x10^{9}L)	54 (38.6)	29 (39.2)	25 (37.9)	0.874
- Lymphopenia (<1.5x10^{9}L)	48 (34.3)	31 (41.9)	17 (25.8)	0.045
- Thrombocytopenia (<150x10^{9}L)	17 (12.1)	6 (8.1)	11 (16.7)	0.122
- Hypergammaglobulinemia	85 (61.2)	59 (79.7)	26 (40.0)	<0.001
- Hypocomplementemia	32 (23.0)	23 (31.1)	9 (13.8)	0.016
- Cryoglobulinemia	8 (16.3)	4 (16.0)	4 (16.7)	1.000
- Anti-Ro/SSA60	129 (92.1)	73 (98.6)	56 (84.8)	0.002
- Anti-SS52	23 (16.4)	14 (18.9)	9 (13.6)	0.400
- Anti-La/SSB	67 (47.9)	51 (68.9)	16 (24.2)	<0.001
ESSDAI median (IQR)	0.9 (2.9)	1.1 (2.9)	0.7 (2.9)	0.084

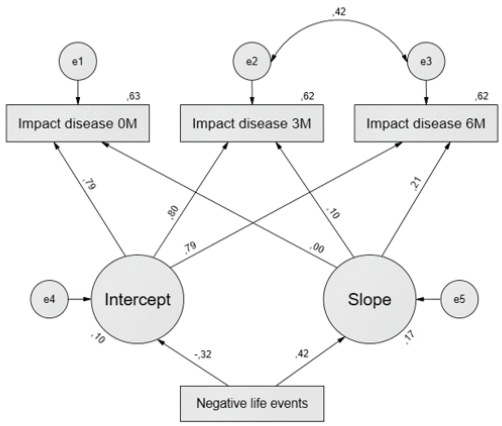
SD: standard deviation; IQR: Interquartile range; ANAs: antinuclear antibodies; ESSDAI: Sjögren's syndrome disease activity index

181 - NEGATIVE LIFE EVENTS INCREASE PERCEIVED IMPACT OF DISEASE IN PATIENTS WITH RHEUMATOID ARTHRITIS: A CONDITIONAL LATENT GROWTH MODEL APPROACH

Maria João Cadório[1], Eduardo Santos[1, 2, 3], João Alexandre Oliveira[1], Sofia C Barreira[4, 5], Pedro Ávila Ribeiro[4, 5], Flávio Campos Costa[6], João Freitas[7], Filipe Araújo[8], Raquel Freitas[9], José Marona[10], Santiago Andres Rodrigues Manica[11, 12], Joana Fonseca Ferreira[13, 14], Couto M[15], Miguel Guerra[10], Ricardo J. O. Ferreira[1, 2, 16], JAP da Silva[1, 17], Cátia Duarte[1, 17]
[1]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Health Sciences Research Unit: Nursing (UICISA: E), Nursing School of Coimbra, Coimbra, Portugal, [3]Instituto de Ciências Biomédicas Abel Salazar, Porto, Portugal, [4]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [5]Rheumatology Research Unit, Instituto de Medicina Molecular, Faculdade de Medicina da Universidade de Lisboa, Lisboa, Portugal, [6]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/ Espinho, Vila Nova de Gaia, Portugal, [7]Hospital de Santo André, Centro Hospitalar de Leiria, E.P.E., Leiria, Portugal, [8]Rheumatology and Osteopororis Unit, Hospital de Sant Ana, Lisboa, Portugal, [9]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [10]Rheumatology Department, Centro Hospitalar e Universitário Cova da Beira, Covilhã, Portugal, [11]Serviço de Reumatologia, Centro Hospitalar Lisboa Ocidental | Hospital Egas Moniz, Lisboa, Portugal, [12]Centro de Estudos de Doenças Crónicas (CEDOC), NOVA Medical School, Faculdade de Ciências Médicas, Lisboa, Portugal, [13]Rheumatology

Department, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [14]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal, [15]Rheumatology Department, Centro Hospitalar Tondela-Viseu, Viseu, Portugal, [16]Centro de Investigação, Inovação e Desenvolvimento em Enfermagem de Lisboa (CIDNUR), Escola Superior de Enfermagem de Lisboa (ESEL), Lisboa, Portugal, [17]Coimbra Institute for Clinical and Biomedical Research (iCBR) - Faculty of Medicine, University of Coimbra, Coimbra, Portugal

Background: Negative life events and stressful situations are known to cause psychological distress with consequences overtime, such as exacerbation of previous conditions or worsening of self-perceived impact of disease.
Objective: This study aimed to evaluate the effect of negative life events on perceived impact of disease in Rheumatoid Arthritis (RA) patients.
Methods: This is an ancillary study of RAID.PT, a prospective, and multicentre study performed in 10 Portuguese rheumatology departments. Patients were followed and treated according to standard guidelines and assessed at baseline, 3 and 6 months.
Disease impact was measured through Rheumatoid Arthritis Impact of Disease (RAID) score and disease activity was measured using the Disease Activity Score 28 joints (DAS28), in its three variables (3v) and C-reactive protein (CRP) variant – DAS28CRP3v. Anxiety and depressive symptoms were assessed using the Hospital Anxiety and Depression Scale (HADS) and Happiness through the Subjective Happiness Scale (SHS). Personality was evaluated by the Ten Item Personality Inventory (TIPI). The presence of negative life events was assessed through the application of a structured questionnaire that focused on the death of relatives and/or close people, divorce, unemployment, and other negative events considered relevant by the participant that had occurred since the last consultation.
Descriptive and comparative analyses were performed using SPSS®, v. 29. Structural equation modelling, more specifically a latent growth model, was used to evaluate the influence of negative life events in the progression of impact of disease overtime, after accounting for individual differences in the disease status at baseline. Statistically significant effects were assumed for $p<0.05$.
Results: In total, 250 RA patients were included (80% female, mean age 57±11.5 years old, mean disease duration 8.8±8.3 years), mostly in remission (DAS-28CRP3v 2.4±1) and with moderate impact of disease (RAID 3.9±2.3). Most patients remained stable over the 6 months of follow-up. Negative life-events were reported by 11.9% of patients during the 6 months of follow-up.
The conditional model showed that the occurrence of negative life events is associated with the change of impact of disease between baseline and 6 months of follow-up ($\beta=0.42$; $p=0.010$), indicating that patients with more negative life events reported a higher rate of impact of disease change across time (Figure 1).
Conclusion: The occurrence of negative life events seems to have an important role in self-perceived impact of disease in RA patients. This evidence reinforces the need to use a holistic approach to people living with RA, that goes beyond a treat-to-target strategy.



Circles represent latent factors. Squares represent measured variables (the scale scores). Arrows connecting circles and rectangles in one direction show a hypothesised direct relationship between the two variables. Circles with the letter "e" written in it represent the associated error. The model is broken down in two latent constructs. The intercept factor represents the mean starting point of impact of disease across the three time periods (base-average values). The slope factor represents the line of trajectory over time of the impact of the disease (mean growth rate).

TO 181 – Figure 1. The influence of negative life events on impact of disease. M, Months.

186 - EXTRACTABLE NUCLEAR ANTIBODIES IN A COHORT OF UNDIFFERENTIATED CONNECTIVE TISSUE DISEASE PATIENTS: RELEVANCE AND CLINICAL IMPLICATIONS

Ana Isabel Maduro[1], Beatriz Mendes[1], Liliana Saraiva[1], Tânia Santiago[1, 2], Ines L[1, 3], JAP da Silva[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal, [3]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal

Background: Undifferentiated Connective Tissue Diseases (UCTD) are systemic autoimmune syndromes within the spectrum of connective tissue diseases

(CTDs) not fulfilling the classification criteria for any of the differentiated CTDs. Autoantibodies against Extractable Nuclear Antigens (ENAs) are frequently found in UCTD patients. However, there are healthy carriers of ENAs autoantibodies that do not progress to present any CTD clinical features.

Objective: To examine the frequency of autoantibodies against ENAs in a cohort of UCTD patients and explore their associations with different clinical and analytical manifestations.

Methods: Cross-sectional single-center study including UCTD patients fulfilling the preliminary LeRoy 1980/Mosca 2014 criteria followed in our University Hospital. Demographic features and cumulative clinical and serological manifestations were collected and compared between UCTD patients with and without

TO 186 – Table 1. Frequency of clinical and serological manifestations in UCTD patients with and without positive ENAs

	Whole Cohort n=184 (100%)	Positive ENAs n=72 (39.1%)	Negative ENAs n=112 (60.9%)	p-value
Age at time of study mean (SD)	52.8 (15.5)	54.1 (16.6)	51.8 (14.6)	0.491
Age at diagnosis mean (SD)	40.7 (15.0)	43.8 (15.1)	37.2 (14.2)	0.051
Female Sex [n (%)]	166 (90.2)	65 (90.3)	101 (90.2)	0.982
Clinical features [n (%)]				
- Sicca syndrome	31 (18.1)	20 (29.0)	11 (10.8)	0.002
- Alopecia	12 (7.1)	9 (13.2)	3 (2.9)	0.014
- Photosensitivity	9 (5.3)	4 (5.9)	5 (4.9)	1.000
- Skin Rash	14 (7.6)	10 (13.9)	4 (3.6)	0.01
- Raynaud's phenomenon	57 (33.3)	15 (22.1)	42 (40.8)	0.011
- Inflammatory arthralgias	53 (31.0)	14 (20.6)	39 (37.9)	0.017
- Arthritis	28 (16.4)	8 (11.8)	20 (19.4)	0.186
- Pulmonar involvement	2 (1.2)	2 (2.9)	0 (0)	-
- Pulmonar arterial hypertension	1 (0.6)	1 (1.5)	0 (0)	-
Antinuclear antibodies [n (%)]				
- Titer [n (%)]				
1:160	171 (95.0)	74 (97.4)	97 (93.3)	0.306
1:320	117 (63.6)	54 (75.0)	63 (56.3)	0.051
1:640	69 (37.5)	38 (52.8)	31 (27.7)	<0.001
1:1280	42 (22.8)	23 (31.9)	19 (17.0)	0.018
1:1280	21 (11.4)	14 (19.4)	7 (6.3)	0.006
- Pattern [n (%)]				
AC-1	16 (8.7)	6 (8.3%)	10 (8.9%)	0.889
AC-2	83 (45.1)	12 (16.7)	71 (63.4)	<0.001
AC-4	44 (23.9)	35 (48.6)	9 (8.0)	<0.001
AC-5	10 (5.4)	6 (8.3)	4 (6.1)	0.193
AC-6	4 (2.2)	2 (2.8)	2 (1.8)	0.645
AC-8	3 (1.6)	2 (2.8)	1 (0.9)	0.562
Other serological features [n (%)]				
- Leukopenia (<4.0x10^{9}L)	39 (21.4)	18 (25.0)	21 (19.1)	0.342
- Lymphopenia (<1.5x10^{9}L)	79 (43.4)	35 (48.6)	44 (40.0)	0.252
- Autoimmune anemia	2 (1.1)	2 (2.8)	0 (0)	0.155
- Thrombocytopenia (<150x10^{9}L)	21 (11.5)	8 (11.1)	13 (11.8)	0.884
- Hypocomplementemia	29 (16.2)	9 (12.9)	20 (18.3)	0.408
- Anti-dsDNA	25 (13.6)	7 (9.7)	18 (16.1)	0.220
- Rheumatoid Factor	10 (6.4)	7 (11.7)	3 (3.1)	0.044

ENAs: Extractable Nuclear Antigen Antibodies. SD: standard deviation; AC-01: Nuclear homogeneous; AC-02: Nuclear dense fine speckled; AC-04: Nuclear fine speckled; AC-05: Nuclear large/coarse speckled; AC-06: Multiple nuclear dots; AC-08: Homogeneous nucleolar; AC-10: Punctate nucleolar

positive ENAs. ENA screening was performed by ELISA (ENA-7 profile). A positive result was further tested by immunoblot assays to determine specificity for SSA52, SSA60, SSB, RNP, Jo-1, Sm and Scl-70. Continuous variables were described as mean or median, according to distribution. Categorical variables were expressed in percentages. The chi-squared test or Fisher test and the independent sample t-test or Mann-Whitney U test were used for comparisons of categorical variables and continuous variables, as appropriate, with p-value <0.05 considered as statistically significant.

Results: 184 UCTD patients were included (90.2% female, mean age 52.8±15.5). ENAs were found in 39.1% of these patients, and the most common ANAs pattern detected in the ENAs positive subgroup was the nuclear fine-speckled pattern (AC-04) (48.6%) (table 1). The most frequently detected anti-ENAs antibodies were antiSSA60 (25.5%), followed by antiSSA52 (11.4%) (table 1). UCTD patients with positive ENAs presented a higher frequency of sicca syndrome (29.0% vs 10.8%; p=0.002), skin rash (13.9% vs 3.6%, p=0.01), alopecia (13.2% vs 2.9%; p=0.014), rheumatoid factor (11.7% vs 3.1%; p=0.044) and high ANAs titer ≥1:640 (52.8% vs 27.7%, p <0.001) (table 1).

Conclusion: UCTD patients with positive ENAs presented a distinctive clinical phenotype. Prospective studies are needed to understand the role of ENAs in the progression of UCTD.

189 - MANIFESTAÇÕES NEUROLÓGICAS NA ARTRITE REUMATÓIDE

Beatriz Samões[1], MD[2], Ana Paula Sousa[3], Raquel Samões[3], Ernestina Santos[3]

[1]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [2]Neuropsicologia, Centro Hospitalar Universitário de Santo António, Porto, Portugal, [3]Neurology, Centro Hospitalar Universitário de Santo António, Porto, Portugal

Introdução: O envolvimento neurológico é uma manifestação extra-articular possível de artrite reumatóide, cuja prevalência tem diminuído nos últimos anos graças ao seu diagnóstico e tratamento mais atempados e à introdução de terapêuticas modificadoras de atividade da doença cada vez mais eficazes.

Objetivo: Caracterizar retrospetivamente as manifestações neurológicas de artrite reumatóide de doentes referenciados à consulta de Neuro-imunologia de um hospital terciário.

Métodos: Estudo observacional retrospetivo de uma coorte de doentes referenciada à consulta especializada de Neuro-imunologia do Centro Hospitalar Universitário de Santo António. Foram selecionados os doentes codificados com o diagnóstico de artrite reumatóide da base de dados da consulta e analisados os seus processos clínicos, extraindo-se dados demográficos, referentes ao envolvimento neurológico e à doença reumática.

Resultados: De um total de 1979 doentes observados na consulta de Neuro-imunologia desde 1976, 36 tinham o diagnóstico de artrite reumatóide, a maioria do sexo feminino (n=32, 89%) e com idade média de 54,4 ± 13,7 anos. Destes, 10 (28%) tinham manifestações neurológicas atribuíveis à doença reumática (tabela 1), 5 (14%) tinham comorbilidades autoimunes neurológicas e 21 (58%) padeciam de outra doença ou alteração neurológica. Entre os doentes com manifestações neurológicas de artrite reumatóide, a maioria tinha doença seropositiva (n=8, 80%), prolongada (duração média de 13,5 ± 9,3 anos) e com baixa atividade ou em remissão (n=5/6, 83,3%) à data do quadro neurológico. Excluindo os casos de iatrogenia, o sistema nervoso periférico foi o mais frequentemente acometido (n=5, 50%), sendo a manifestação mais prevalente a de neuropatia sensitiva distal. Registaram-se três casos de envolvimento do sistema nervoso central, dois dos quais de meningite reumatóide que implicaram uma otimização da imunossupressão e um de trombose dos seios venosos numa doente com outros fatores de risco pró-coagulantes nomeadamente o início de contracetivo estrogénico e o diagnóstico recente de neoplasia mamária. Assinalaram-se dois casos de eventos neurológicos atribuíveis à toma de anti-TNF que implicaram a suspensão do fármaco com melhoria do quadro. Em relação às comorbilidades autoimunes neurológicas, quatro doentes eram seguidos por miastenia gravis e um por esclerose múltipla. Nos restantes doentes, os sintomas enquadravam-se nos seguintes grupos de doenças: perturbação cognitiva (n=8), cefaleia (n= 7), síndrome vertiginoso (n=1), epilepsia (n=1), encefalopatia hipertensiva (n=1), diplopia (n=1), perturbação da marcha (n=1) e síndrome depressivo (n=1).

Conclusão: As manifestações neurológicas de artrite reumatóide, embora raras, continuam presentes na prática clínica e podem ser graves ao ponto de implicar um reforço da imunossupressão ou a suspensão de terapêuticas, caso haja uma suspeita de iatrogenia. Não obstante, a maioria dos sintomas neurológicos exibidos por doentes com artrite reumatóide não são atribuíveis à doença reumática. É fundamental uma cooperação multidisciplinar entre a Reumatologia e a Neurologia para que haja um correto reconhecimento e valorização das queixas neurológicas dos doentes com artrite reumatóide.

TO 189 – Table 1.

ID	Sexo	Artrite reumatóide					Manifestação neurológica					
		Idade*	FR/ ACCP	Erosiva	Atividade*	Duração (anos)*	Grupo	Tipo	Sintomas	MCDTs	Tratamento	Resultado
1	F	50	S	S	NR	20	SNC	Meningite reumatóide	Cefaleia, disfasia e alteração do estado de consciência	PL: pleocitose (15 leucócitos) e ↑índice IgG; RMN: ↑ captação de contraste leptomeníngea; biópsia: inflitrado linfo-histiocitário das leptomeninges	Corticóide em alta dose + Ciclofosfamida	Melhoria
2	F	52	S	N	Baixa	13	SNC	Meningite reumatóide	Cefaleia	PL: normal; RMN: espessamento e ↑ captação de contraste da dura-máter	Corticóide em alta dose	Melhoria
3	F	47	NR	NR	NR	23	SNC	Trombose dos seios venosos	Cefaleia súbita intensa	Anti-SAF negativos; RMN: trombose dos seios venosos	Hipocoagulação	Melhoria
4	F	68	S	S	NR	8	SNP	Vasculite reumatóide	Disestesias e parestesias dos pés	EMG: mononeuropatia múltipla	Corticóide em alta dose + Azatioprina	Melhoria
5	F	74	S	N	NR	2	SNP	Neuropatia sensitiva distal STC	Disestesias e parestesias das mãos e pés	EMG: neuropatia sensitiva distal e STC bilateral	Gabapentina	Estabilidade
6	F	48	N	N	Baixa	1	SNP	Neuropatia sensitiva distal STC	Hipostesia e parestesias das mãos e pés	EMG: neuropatia sensitiva distal e STC bilateral	Pregabalina	Estabilidade
7	F	58	S	N	Baixa	11	SNP	Neuropatia sensitiva distal	Hipostesia do pé direito	EMG: PNP sensitiva axonal distal; Biópsia de nervo: PNP axonal	Gabapentina	Melhoria
8	F	60	S	N	Moderada	22	SNP	STC	Parestesias noturnas dos dedos à direita	EMG: STC direito ligeiro	-	Estabilidade
9	F	54	S	N	Baixa	7	Iatrogenia	Défice cognitivo associado a anti-TNF	Défice cognitivo ligeiro não mnésico	PL: normal; RMN: lesões inflamatórias de substância branca de predomínio peri-ventricular	Stop etanercept + Rituximab	Melhoria
10	F	57	S	S	Remissão	28	Iatrogenia	Défice neurológico transitório associado a anti-TNF	Episódio de parestesias e hipostesia do hemicorpo esquerdo	Anti-MOG e anti-AQP4 negativos; PL: normal; RMN CE: dois focos subcorticais de hipersinal em T2 e FLAIR inespecíficos; RMN medular: normal	Stop etanercept	Melhoria

Legenda: *aquando do diagnóstico da manifestação neurológica; ACCP – Anticorpos anti-peptídeos citrulinados; AQP4 – Aquaporina-4; F – Feminino; FR – Fator reumatóide; EMG – Eletromiografia; ID – Identificação; MCDT – Exames complementares de diagnóstico; MOG - glicoproteína de oligodendrócito de mielina; N – Não; NR – Não registado; PL – Punção lombar; PNP – Polineuropatia; RMN – Ressonância magnética; S – Sim; SAF – Síndrome anti-fosfolipídico; SNC – Sistema nervoso central; SNP – Sistema nervoso periférico; STC – Síndrome do túnel cárpico; TNF – Fator de necrose tumoral.

191 - BASELINE CHARACTERISTICS OF PATIENTS WITH DIFFICULT-TO-TREAT RHEUMATOID ARTHRITIS IN A SECONDARY CENTRE

Sara Paiva Dinis [1, 2], Hugo Parente [1], Catarina Soares[1], Maria Pontes Ferreira[1], Anita Cunha[1], Daniela Santos Faria [1], Carmo Afonso[1], Daniela Peixoto[1], Filipa Teixeira[1], Francisca Guimarães[1], Soraia Azevedo[1], José Tavares-Costa[1]
[1]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [2]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal

Background: Rheumatoid arthritis' (RA) treatment has substantially improved over the past decades, with the widespread of treat-to-target (T2T) strategy and remarkable progress in therapeutic agents enabling patients to achieve remission or low disease activity. However, there is still a subgroup of patients in which disease activity is difficult to control despite treatment according to the current management recommendations, with up to twenty percent of RA patients suffering from difficult-to-treat disease. In 2021, the European League Against Rheumatism (EULAR) task force proposed the definition of difficult-to-treat (D2T) RA, providing greater consistency in clinical and research settings.[1-2]

Objectives: To evaluate baseline characteristics of RA patients at initiation of biological treatment in our rheumatology department and identify and compare D2T RA with non-D2T RA patients.

Methods: A retrospective single-centre observational study was performed including all patients under biological or targeted synthetic disease-modifying anti-rheumatic drugs (b/tsDMARDs) in our centre. Baseline clinical characteristics, lifestyle habits and treatment history of RA patients who were receiving biological treatment with more than 6 months follow-up until May 2023 were obtained from electronical med-

TO 191 – TABLE 1. Comparison of the baseline clinical characteristics of patients with difficult-to-treat RA (D2T RA) and non-D2T RA at baseline.

	All n=77	D2T RA n=14	Non-D2T RA n=63	p-value
Sex, female, n/N (%)	54/77 (70.1)	10/14 (71.4)	44/63 (69.8)	0.592
Age at diagnosis, years, mean ± SD	46.3 ± 14.2	46.7 ± 14.2	44.4 ± 15.6	0.597
Symptoms duration until first b/tsDMARD, years, mean ± SD	11.2 ± 8.0	11.4 ± 9.7	11.2 ± 7.7	0.923
Time until first b/tsDMARD, years, mean ± SD	8.4 ± 6.8	7.5 ± 6.9	8.6 ± 6.8	0.583
Smoking history, n/N (%)	18/58 (31)	6/12 (50)	12/46 (26.1)	0.161
Erosive disease, n/N (%)	54/77 (70.1)	13/14 (92.9)	41/63 (65.1)	0.049
BMI	26.9 ± 5.3	23.4 ± 3.2	28.1 ± 5.4	0.02
Serology				
RF-positive, n/N (%)	58/77 (75.3)	11/14 (78.6)	47/63 (74.6)	1.00
ACPA-positive, n/N (%)	61/75 (81.3)	12/14 (85.7)	49/61 (80.3)	1.00
History of treatment				
Number of used csDMARds, mean ± SD	1.9 ± 0.6	1.7± 0.7	1.9 ± 0.6	0.243
PDN user, n/N (%)	65/77 (84.4)	12/14 (85.7)	53/63 (84.1)	1.00
Disease activity				
DAS-28-ESR, mean ± SD	5.1 ± 1.3	5.5 ± 1.8	5.0 ± 1.2	0.367
CDAI, mean ± SD	24.9 ± 13.3	28.2 ± 17.3	24.2 ± 12.3	0.416
SDAI, mean ± SD	26.8 ± 14.1	30.6 ± 17.3	25.8 ± 13.2	0.345
TJC (0-28), mean ± SD	8.6 ± 7.1	12.4 ± 9.4	7.7 ± 6.3	0.097
SJC (0-28), mean ± SD	7.0 ± 4.7	9.3 ± 6.0	6.5 ± 4.2	0.117
VAS (0-100), mean ± SD	52.7 ± 27.9	62.1 ± 29.3	50.6 ± 27.4	0.163
PGA (0-100), mean ± SD	52.2 ± 25.2	59.6 ± 26.5	50.5 ± 24.8	0.225
HAQ, mean ± SD	1.7 ± 38.0	1.4 ± 0.9	1.71 ± 4.8	0.822
CRP, mean ± SD	2.5 ± 3.6	4.4 ± 6.8	2.1 ± 2.3	0.234
ESR, mean ± SD	30.7 ± 21.2	32.9 ± 23.5	30.2 ± 20.8	0.669

ical records and Rheumatic Disease Portuguese Registry (Reuma.pt). For this analysis, D2T RA was defined as RA with at least moderate disease activity at the last visit according to the disease activity scores, namely disease activity score of 28 erythrocyte sedimentation rate (DAS28-ESR)>3.2 and/or clinical disease activity index (CDAI) >10 and/or simplified disease activity index (SDAI) >11, despite the use of at least two b/tsDMARDs. SPSS V.25 was used for statistical analysis. DT2 and non-DT2 RA patients were compared using Chi-squared test and student t-test/Mann-Whitney tests, as appropriate. Significant level was 2-sided<0.05.

Results: 77 RA patients were enrolled in this study, 70.1% were female, with a mean age at disease onset of 46.3(±14.2) years and a mean disease duration at the first b/tsDMARD of 11.2 (±8.0) years. Patients with D2T RA accounted for 18.2% of overall patients. Erosive disease (92.9 vs 65.1%, p=0.049) and lower body mass index (BMI) (23.4 ± 3.2 vs 28.1 ± 5.4, p=0.02) had significant association with D2T RA. Although without reaching statistical significance, D2T RA patients had a higher baseline visual analogic scale of pain and SDAI score (62.1 vs 50.6 and 30.6 vs 25.8, respectively). Additionally, the mean baseline counts of tender and swollen joints were higher in D2T RA (12.4 ± 9.4 vs 7.7 ± 6.3 and 9.3 ± 6.0 vs 6.5 ± 4.2, respectively), same being with C-reactive protein (4.4 ± 6.8 vs 2.1 ± 2.3). A higher proportion of D2T RA patients presented smoking history (50.0 vs 26.1%).

Conclusions: These results indicate that in routine clinical practice, a significant proportion of RA patients do not achieve clinically relevant improvement even after 2 b/tsDMARDs and may require specific management. Although patients and physician opinion were not possible to obtain, an even higher number of patients might meet D2T EULAR 2021 criteria.

REFERENCES:

1. Roodenrijs NM, et al. Difficult-to-treat rheumatoid arthritis: contributing factors and burden of disease. Available from: https://academic.oup.com/rheumatology/advance-article/doi/10.1093/rheumatology/keaa860/6040764
2. Nagy G, et al. EULAR definition of difficult-To-Treat rheumatoid arthritis. Ann Rheum Dis. 2021 Jan 1;80(1):31–5

194 - SEGURANÇA, EFICÁCIA E PERSISTÊNCIA DA TERAPÊUTICA COM RITUXIMAB EM DOENTES COM SÍNDROME DE SJÖGREN: ESTUDO LONGITUDINAL MULTICÊNTRICO

Carla Campinho Ferreira[1], Matilde Bandeira[2, 3], Susana Matias[4], Inês Almeida[5], Vasco C Romão[2, 3], Joana Leite Silva[1]
[1]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [4]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [5]Unidade de Reumatologia, Centro Hospitalar Tondela-Viseu, Viseu, Portugal

Introdução: Cerca de metade dos doentes com síndrome de Sjögren (SS) podem ter envolvimento extraglandular. A terapêutica destes doentes assenta no tratamento sintomático e no recurso a imunossupressores nas manifestações sistémicas. Os ensaios clínicos randomizados com rituximab (RTX) na SS não atingiram os endpoints primários. No entanto, estudos observacionais reportaram melhoria clínica com a sua utilização, estando atualmente recomendada para manifestações graves.

Objetivos: Caraterizar uma coorte de doentes com SS tratados com RTX.

Métodos: Estudo retrospetivo com doentes com SS cumprindo os critérios de classificação ACR/EULAR 2016 sob terapêutica atual ou prévia com RTX, seguidos em 4 centros terciários de Reumatologia. Os dados demográficos e as variáveis clínicas e serológicas prévias ao início do tratamento e aos 6 e 12 meses após o tratamento com RTX foram analisados. Foi avaliada a segurança, eficácia e persistência do tratamento. Foi considerada resposta clínica uma variação do EULAR SS Disease Activity Index (ESSDAI) ≥ 3.

Resultados: Foram incluídos 22 doentes, 95,5% do sexo feminino, com uma idade média de 53,7 ± 13,2 anos e uma média de duração de doença de 8,3 ± 7,9 anos. As características clínicas e analíticas à baseline encontram-se detalhadas na tabela 1. Os motivos para o início de RTX foram: envolvimento articular (n=7), pulmonar (n=6), cutâneo (n=4), hematológico (n=2), neuropático (n=1), renal (n=1) e linfoma MALT (n=1). O ESSDAI médio antes do início do fármaco era de 15,95 ± 10,66, sendo que 15 doentes (68%) se encontravam sob 2 ou mais fármacos imunossupressores e a dose média de prednisolona era de 18 ± 22,2 mg/dia. Após o primeiro ciclo, 10 doentes (45,5%) mantiveram tratamento regular de 6/6 meses, 4 mantiveram tratamento consoante necessidade clínica e 1 doente fez tratamento segundo esquema de hemato-oncologia (1g mensal durante 10 meses). Em 2 doentes o tratamento foi suspenso após o primeiro ciclo por estabilidade clínica, 2 suspenderam por efeitos adversos (anafilaxia e infeção respiratória com necessidade de internamento) e 1 doente suspendeu por iniciativa própria. Aos 12 meses após início do tratamento, a média de ciclos efetuados foi de 1,6 ± 0,8 (n=19). Dois terços dos doentes (n=12/18) apresentaram uma boa resposta com di-

TO 194 – TABELA 1. Características clínicas à baseline nos doentes com SS tratados com RTX

Características	
Sexo, % sexo feminino	95,5
Idade, média ± DP anos	53,7 ± 13,2
Tempo de evolução de doença, média ± DP anos	8,3 ± 7,9
Comorbilidades	
HTA, n/N (%)	4/22 (18,2)
Dislipidemia, n/N (%)	3/22 (13,6)
Diabetes mellitus tipo 2, n/N (%)	5/22 (22,7)
Anticorpos antinucleares positivos, n/N (%)	22/22 (100)
SSA positivo, n/N (%)	12/22 (54,5)
SSA e SSB positivo, n/N (%)	8/22 (36,4)
SSA e SSB negativos, n/N (%)	2/22 (9,1)
Fator reumatoide positivo, n/N (%)	13/22 (59,1)
Consumo de C3, n/N (%)	4/16 (25)
Consumo de C4, n/N (%)	3/16 (18,8)
Hipergamaglobulinemia, n/N (%)	7/20 (35)
Proteína C reativa, média ± DP mg/L (n=20)	16,98 ± 30,54
Velocidade de sedimentação, média ± DP mm/h (n=21)	52,3 ± 31,8
Terapêutica imunossupressora	
Um fármaco, n/N (%)	5/20 (25)
Prednisolona (PDN), n	3
Hidroxicloroquina (HCQ), n	2
Dois fármacos, n/N (%)	10/20 (50)
PDN + HCQ, n	3
PDN + Metotrexato (MTX), n	3
PDN + Leflunomida (LEF), n	2
PDN + Azatioprina (AZA), n	1
PDN + Micofenolato de Mofetil, n	1
Três fármacos, n/N (%)	5/20 (25)
PDN + HCQ + AZA, n	2
PDN + HCQ + LEF, n	1
PDN + HCQ + MTX, n	1
PDN + HCQ + Imunoglobulinas, n	1
Dose de PDN, média ± mg/d (n=20)	18,13 ± 22,18

minuição do ESSDAI ≥ 3. Estratificando para os domínios, independentemente do motivo de início, todos os doentes com envolvimento renal (n=2) e linfadenopático (n=4) e 80% dos doentes com envolvimento glandular (n=4) e articular (n=8) apresentaram melhoria clínica. Dos 18 doentes sob corticoterapia prévia, 11 (61%) conseguiram diminuir a dose aos 6 meses de follow-up. Foram documentados os seguintes efeitos adversos em 5 doentes: anafilaxia (n=1), toxicidade gastrointestinal (n=1) 5 dias após perfusão, infeção respiratória (n=1) 2 semanas após o tratamento, cefaleia, dispneia e fadiga (n=1) 2 semanas após o tratamento e lesões cutâneas púpura-like (n=1) no dia seguinte à perfusão.

Conclusão: Na amostra de doentes estudada, o RTX foi eficaz no tratamento do envolvimento sistémico na SS, com mais de metade dos doentes a apresentarem uma resposta ESSDAI aos 6 meses. A segurança foi de acordo com o esperado para este fármaco. No entanto, são necessários mais estudos a longo prazo para avaliar a eficácia e a segurança da terapêutica com RTX nos doentes com SS.

195 - RHEUMATOLOGY AND DERMATOLOGY CLINIC FOR PSORIATIC ARTHRITIS: TEN YEARS OF EXPERIENCE IN A TERTIARY CENTER

Carolina Ochôa Matos[1, 2], Filipe Cunha Santos[3], Augusto Silva[1, 2], Miguel Alpalhao[4, 5], P Filipe[4, 5], JE Fonseca[1, 2], Carlos Miranda Rosa[1], Antunes J[4, 5], Elsa Vieira-Sousa[1, 2]
[1]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Hospital de Santa Maria, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [4]Serviço de Dermatologia, Centro Hospitalar e Universitário Lisboa Norte, Hospital de Santa Maria, Lisboa, Portugal, [5]Clínica Universitária de Dermatologia de Lisboa, Hospital de Santa Maria, Centro Hospitalar Universitário de Lisboa Norte, Lisboa, Portugal

Introduction: Psoriatic arthritis (PsA) is a chronic inflammatory disease characterized by highly heterogeneous phenotypes combining psoriasis (PsO), with peripheral and axial arthritis/enthesitis. Its clinical diversity makes early and definite diagnosis, as well as remission targets, difficult to establish.

Despite data reporting bidisciplinary care units still being scarce, they show that a multidisciplinary approach, encompassing both dermatology and rheumatology specialities, has better results in both joint and skin improvement, as well as high patient and physician satisfaction levels. The objective of this study was to describe the main outcomes of the Rheumatology/Dermatology clinic established at Hospital de Santa Maria over a period of approximately 10 years.

Methodology: Single center, cross sectional study of all patients observed at the Rheumatology/Dermatology Clinic of Hospital de Santa Maria, between November 2010 and February 2021. Patients are referred due to diagnostic or therapeutic indications, such as suspected

PsA or PsO, or due to inadequate control of PsA and/or PsO disease activity. The total number of visits and patients; the indication for referral; definite skin and musculoskeletal diagnosis, including confirmation and exclusion of PsO and PsA; and treatment modifications, were registered. Demographics for the whole population, PsA and PsO disease subtypes, disease duration, previous treatments and disease activity indices for both PsO and PsA were captured using Reuma.pt.

Results: A total of 569 visits were performed, corresponding to a total of 509 patients, 54.9% male, with a mean age of 50.4 years.

The main indication for referral was uncertainty of either a skin or musculoskeletal (MSK) diagnosis (55.2%). A "de novo" diagnosis of PsO and PsA was established in 28.7% and 42.5% of the cases, respectively. For those in whom a PsA diagnosis was not established the main alternative diagnosis was osteoarthritis: peripheral (n=70) or axial (n=29); fibromyalgia (n=22); tendinitis/enthesitis (n=20); carpal tunnel syndrome (n=19) and axial spondylarthritis without PsO (n=19). The main alternative dermatological diagnosis were seborrheic dermatitis (n=15), nail dystrophy (n=15), onychomycosis (n=14) and eczema (n=9).

In those with a confirmed PsA diagnosis (n=329), the most prevalent PsA subtype was symmetric polyarthritis (50.9%), followed by olygoarthritis (25.3%), predominant spondylitis (15.3%) and the distal interphalangeal predominant subtype (7.5%). There were three cases of arthritis mutilans. Considering extra-articular manifestations, 81.4% of the PsA patients had a history of (or current) cutaneous psoriasis, 36.6% of nail involvement, 27.4% dactylitis, 3.7% anterior uveitis, and 0.6% ulcerative colitis.

Prior to the first appointment, 54.3% had already been treated with disease-modifying antirheumatic drugs (DMARD), mainly methotrexate, and 15.5% had received treatment with at least one biological DMARD. Treatment was modified due to uncontrolled skin activity (61.5%), or MSK activity (28.8%), or both (9.6%).

Conclusion: This study shows the impact, through diagnostic precision and treatment modifications, from over 10 years of the implementation of a national multidisciplinary Rheumatology and Dermatology PsA Clinic. Furthermore, it reinforces the usefulness of shared Rheumatology and Dermatology assessment and decisions for the care of a large population of PsA patients.

Acknowledgments: Statistical analysis provided by X2-Science Solutions supported by Novartis.

196 - INFECTION RISK WITH RITUXIMAB – THE EXPERIENCE OF A SECONDARY RHEUMATOLOGY CENTRE

Maria Pontes Ferreira[1], Hugo Parente[1], Catarina Soares[1], Sara Paiva Dinis [2], Anita Cunha[1], Soraia Azevedo[1], Daniela Peixoto[1], Filipa Teixeira[1], Daniela Santos-Faria[1], Carmo Afonso[1], Francisca Guimarães[1], José Tavares-Costa[1]
[1]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [2]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal

Background: Rituximab (RTX) is an anti-CD20 monoclonal antibody used for treatment of a variety of rheumatic diseases, such as rheumatoid arthritis (RA), anti-neutrophil cytoplasmic antibodies-associated vasculitis (AAV), systemic sclerosis (SSc), Sjögren's syndrome (SS) and systemic lupus erythematosus (SLE)1. Despite previous studies revealing a reasonable risk/benefit ratio, it has an increased infection risk, correlated with hypogammaglobulinemia and cytopenia2.

Objective: To assess the incidence of infections associated with RTX treatment in rheumatic diseases and compare patients with and without infections regarding sociodemographic and clinical characteristics.

Methods: A retrospective single-centre observational study was performed including all patients who received RTX from January/2000-December/2022. Clinical records were reviewed for sociodemographic data, comorbidities, clinical diagnosis, disease characteristics and activity, concomitant disease-modifying antirheumatic drugs (DMARDs), prednisolone (PDN) and RTX dose, and hypogammaglobulinemia/cytopenia at the time of infection. Infections and severity after beginning RTX were recorded. Major infections were defined as those requiring hospital admission. SPSS V. 25 was used for statistical analysis. Patients with and without infections were compared using Chi-square test and student t-test/Mann-Whitney tests, as appropriate. Significant level was 2-sided $p<.05$.

Results: 41 patients were included, 30 were woman, with a mean age of 52.2 years at RTX's first infusion. RA and SLE were the commonest diagnosis (43.9% and 22.0%). 73.2% of patients had concomitant comorbidities, mainly overweight or obesity (58.3%), smoking habits (40.0%) and hypertension (26.8%). 8 patients (19.5%) already had a severe infection before RTX. RTX's major indication was joint involvement (43.9%) and RTX was the first biological treatment in 56.1% of the patients. 27 patients were concomitantly treated with conventional DMARDs, mainly MTX (36.3%).

A total of 88 infections were recorded, on 35 patients (85.5%); 21 were considered major infections (8 patients). Most infections affected the respiratory and genitourinary systems.

RTX's discontinuation rate was 31.4%, significantly higher in SAE group (75.0% vs. 18.5%, p=0.003); death rate was 8.6%.

TO 196 – TABLE 1. Patient and disease characteristics at baseline of RTX and at infection

	All patients N=41	No infection N=6	Infection, N=35		p-value
			Minor infection N=31	Major infection N=8	
Sociodemographic characteristics					
Age at diagnosis (years) – mean±SD	42.0±16.2	47.27±20.2	41.1±15.6		NS
			40.4±15.6	43.7±16.6	NS
Gender (female) – n/N (%)	30/41 (73.2)	5/6 (83.3)	25/35 (71.4)		NS
			20/27 (74.1)	5/8 (62.5)	NS
Comorbidities – n/N (%)	30/41 (73.2)	3/6 (50)	21/35 (60)		NS
			19/27 (70.4)	7/8 (87.5)	NS
Number of comorbidities – median (IQR)	1.0 (1.0)	1.0 (3.0)	1.0 (1.0)		NS
			1.0 (1.0)	1.0 (2.0)	NS
Smoking status (Smoker/ Ex-smoker) – n/N (%)	12/30 (40.0)	2/6 (33.3)	10/24 (41.7)		NS
			8/19 (42.1)	2/5 (40.0)	NS
Moderate/heavy drinker alcohol consumption – n/N (%)	4 (18.2)	1/4 (25)	3/18 (16.7)		NS
			3/14 (21.4)	0/4 (0.0)	NS
Hypertension – n/N (%)	11/41 (26.8)	1/6 (16.7)	10/35 (28.6)		NS
			8/27 (29.6)	2/8 (25.0)	NS
Diabetes mellitus – n/N (%)	4/41 (9.8)	1/6 (16.7)	3/35 (8.6)		NS
			1/27 (3.7)	2/8 (25.0)	NS
ILD – n/N (%)	9/41 (22.0)	2/6 (33.3)	7/35 (20.0)		NS
			5/27 (18.5)	2/8 (25.0)	NS
Overweight/obesity – n/N (%)	7/12 (58.3)	1/2 (20)	6/10 (60)		NS
			4/7 (57.1)	2/3 (66.7)	NS
CKD – n/N (%)	2/41 (4.9)	0	2/35 (5.7)		NS
			0	2/8 (25.0)	0.047
Previous neoplasia – n/N (%)	2/41 (4.9)	1/6 (16.7)	1/35 (2.9)		NS
			0	1/8 (12.5)	NS
Previous severe infection – n/N (%)	8/41 (19.5)	1/6 (16.7)	7/35 (20)		NS
			5/27 (18.5)	2/8 (25.0)	NS
At baseline of RTX					
Age (years) – mean±SD	52.2±13.9	61.2±16.3	50.3±13.2		NS
			50.0±13.0	52.9±13.95	NS
Years of disease – mean±SD	10.5±7.3	13.9±7.4	9.9±7.3		NS
			10.10±7.7	9.14±5.3	NS
Diagnosis					
RA – n/N (%)	18/41 (43.9)	5/6 (83.3)	13/35 (37.1)		
			10/27 (37)	3/8 (37.5)	
Connective tissue disease – n/N (%)	19/41 (46.3)	1/6 (16.7)	18/35 (51.4)		NS
			14/27 (51.9)	4/8 (50.0)	
AAV – n/N (%)	4/41 (9.8)	0	4/35 (11.4)		
			3/27 (11.1)	1/8 (12.5)	
Prednisolone doses (or equivalent) – median (IQR)	5.0±10.0	5.0±4.4	14.7±15.0		NS
			5.0±10.0	10.0±30.0	NS

continues on the next pagw

TO 196 – TABLE 1. Continuation

	All patients N=41	No infection N=6	Infection, N=35		p-value
			Minor infection N=31	Major infection N=8	
Inflammatory parameters					
ESR (mm/h) – mean±SD	37±28	27±17	39±30		NS
			35±28	52±33	NS
Hypogammaglobulinemia – n/N (%)	2/13 (15.4)	0	2/12 (16.7)		NS
			0	2/3 (66.7)	0.045
Leuko/neutropenia – n/N (%)	8/41 (19.5)	0	8/35 (22.9)		NS
			8/27 (29.6)	0	NS
Pneumocystis jirovecii prophylaxis – n/N (%)	5/41 (12.2)	0	5/35 (14.3)		NS
			5/27 (18.5)	0	NS
cDMARDs					
MTX – n/N (%)	15/41 (36.3)	4/6 (66.7)	11/35 (31.4)		NS
			6/27 (22.2)	5/8 (62.5)	0.031
LEF – n/N (%)	5/41 (12.2)	1/6 (16.7)	4/35 (11.4)		NS
			3/27 (11.1)	1/8 (12.5)	NS
MMF – n/N (%)	5/41 (12.2)	0	5/35 (14.3)		NS
			3/27 (11.1)	2/8 (25.0)	NS
AZA – n/N (%)	2/41 (4.9)	0	2/35 (5.7)		NS
			2/27 (7.4)	0	NS
Number of previous b/tsDMARD – median (IQR)	0.0±1.0	0.0±1.0	0.5±1.0		NS
			0.0±1.0	0.5±1.0	NS
Reason for RTX treatment					
Articular – n/N (%)	18/41 (43.9)	4/6 (66.7)	14/35 (40.0)		
			12/27 (44.4)	2/8 (25.0)	
Pulmonary/serosae – n/N (%)	7/41 (17.1)	2/6 (33.3)	5/35 (14.3)		-
			3/27 (11.1)	2/8 (25.0)	
Other – n/N (%)	16/41 (39.0)	0	16/35 (45.7)		
			12/27 (44.4)	4/8 (50.0)	
Moderate/high disease activity – n/N (%)	31/37 (90.2)	4/5 (80.0)	27/32 (84.4)		NS
			21/25 (84.0)	6/7 (85.7)	NS
At infection (first or most severe)					
Infections by system					
Respiratory – median (IQR)	-	-	1.0±1.0		-
			1.0±1.0	2.5±3.0	NS
Mucocutaneous – median (IQR)	-	-	1.0±1.0		
			1.0±1.0	1.5±0.0	NS
Genitourinary – median (IQR)	-	-	2.0±4.0		
			2.0±2.0	-	NS
Others – median (IQR)	-	-	1.0±1.0		
			1.0±0.0	1.0+0±1.0	NS
Age (years) – mean±SD	-	-	52.2±13.1		-
			51.5±13.0	54.7±14.1	NS
Months after RTX – median (IQR)	-	-	13.1±13.8		-
			13.1±13.8	15.0±38.1	NS

continues on the next pagw

TO 196 – TABLE 1. Continuation

	All patients N=41	No infection N=6	Infection, N=35		p-value
			Minor infection N=31	Major infection N=8	
Prednisolone doses (or equivalent) – median (IQR)	-	-	5.0±10.0		-
			5.0±7.3	7.50±13.8	NS
cDMARDs					
MTX – n/N (%)	-	-	10/35 (28.6)		-
			5/27 (18.5)	5/8 (62.5)	0.027
LEF – n/N (%)	-		3/35 (8.6)		-
			2/27 (7.4)	1/8 (12.5)	NS
MMF – n/N (%)	-	-	4/35 (11.4)		-
			3/27 (11.1)	1/8 (12.5)	NS
AZA – n/N (%)	-	-	2/35 (5.7)		-
			2/27 (7.4)	0	NS
Cumulative doses of RTX (mg) – median (IQR)	-	-	4000±4000		-
			4000±4000	4500±6625	NS
Inflammatory parameters					
ESR (mm/h) – mean±SD	-	-	34.3±24.5		-
			27±9	62±9	0.001
CRP (mg/day) – median (IQR)	-	-	2.9±5.5		-
			1.0±3.9	8.7±12.0	0.002
Leuko/neutropenia – n/N (%)	-	-	4/34 (11.8)		-
			4/26 (15.4)	-	-
Moderate/high disease activity – n/N (%)	-	-	15/32 (46.9)		-
			10/26 (38.5)	5/6 (83.3)	0.047
Suspension of RTX – n/N (%)	-	-	11/35 (31.4)		-
			5/27 (18.5)	6/8 (75)	0.003
Months until suspension – mean±SD	-	-	15.1±16.3		-
			16.8±13.3	13.7±19.6	-
Deaths related to infections – n/N (%)	-	-	3/35 (8.6)		-
			0	3/8 (37.5)	-

Legend: AAV – anti-neutrophil cytoplasmic antibodies-associated vasculitis, AZA – azathioprine, b/tsDMARDs – biological/targeted synthetic disease-modifying antirheumatic drugs (bDMARDs), cDMARDs – conventional disease-modifying antirheumatic drugs, CKD – chronic kidney disease, CRP – C-reactive protein, ESR – erythrocyte sedimentation rate, ILD – interstitial lung disease, LEF – leflunomide, MMF – mycophenolate mofetil, MTX – methotrexate, RA – rheumatoid arthritis, RTX – rituximab, NS – not significant, n – number of patients, N – number of patients with available data.
Categorical variables are presented as number/total population (percentage); continuous variables are presented as mean ± standard deviation when data followed a normal distribution and median (interquartile range) when data did not follow normal distribution.

MTX's concomitant use, moderate/high disease activity at the time of infection and hypogammaglobulinemia were associated with serious infections (18.5% vs 62.5%, p=0.027; 38.5 vs. 83.3%, p=0.047, 0 vs 66.7%, p=0.045, respectively). Although not statistically significant, the average dose of PDN tended to be higher on the severe infection group (5.1 vs 11.3 mg/day), the same being with cumulative mean dose of RTX (4364mg vs. 6063mg). Diabetes Mellitus was more frequent in the group of patients with severe infections (3.7 vs. 25.0%). Although RA represented the majority of patients in this cohort, most of patients with infections had a connective tissue disease, such as SLE. **Conclusion:** In this cohort infection rate and seriousness seem to be more common in patients with higher disease burden. Although several confounding factors such as RTX indication, disease activity and concomitant medications can play a role, this study warns for the infection risk of RTX and the need for adequate risk mitigation strategies (monitoring, prophylaxis and vaccination).

REFERENCES:
1. Schioppo T et al. Current perspective on rituximab in rheumatic diseases. Drug Des Devel Ther. 2017
2. Varley CD et al. Long-Term Safety of Rituximab (Risks of Viral and Opportunistic Infections). Curr Rheumatol Rep. 2021

202 - TREATMENT OF INFLAMMATORY RHEUMATIC DISEASE-ASSOCIATED INTERSTITIAL LUNG DISEASES WITH NINTEDANIB – A MULTICENTER NATIONWIDE STUDY

Augusto Silva[1,2], Ana Martins[3,4], Beatriz Samões[5], Carla Campinho Ferreira[6], C. Pinto Oliveira[7], João Aguiar[1,2], Laura Gago[8], Carlos Costa[9], Ana Catarina Duarte[10], Filipe Araújo[11], Francisca Guimarães[12], Maria João Salvador[13], Vítor Teixeira[14], Natália Melo[15], Joana Leite Silva[6], JE Fonseca[1,2], Maria João Gonçalves[8], Nikita Khmelinskii[1,2], P Pinto[5], Ana Rita Prata[7], Vasco C Romão[1,2], Miguel Bernardes[3,4], Cristina Ponte[1,2], Sofia C Barreira[1,2], Catarina Resende[1,2]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas do Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar de São João, Porto, Portugal, [4]Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [5]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [6]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [7]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [8]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [9]Serviço de Reumatologia, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal, [10]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [11]Serviço de Reumatologia, Hospital CUF Cascais, Cascais, Portugal, [12]Rheumatology Department, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [13]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [14]Serviço de Reumatologia, Centro Hospitalar e Universitário do Algarve, Faro, Portugal, [15]Serviço de Pneumologia, Centro Hospitalar Universitário de São João, Porto, Portugal

Background: Interstitial lung disease (ILD) is a severe manifestation of inflammatory rheumatic diseases (IRD). Conventional therapies have demonstrated limited effectiveness in controlling ILD with short-lived benefits. Recently, Nintedanib (NTD) has received approval for ILD associated with systemic sclerosis (SSc) and progressive fibrosing ILD(1). However, the knowledge of NTD in ILD associated with other IRDs remains limited.

Objectives: To describe the clinical features and outcomes of patients with IRD-ILD treated with NTD across twelve Portuguese rheumatology departments.

Methods: Retrospective cohort study with chart review of medical records to identify IRD-ILD patients who were treated with NTD. The clinical characteristics of patients are described as frequencies for categorical variables and mean ± standard deviation for continuous variables. Differences between groups were evaluated through chi-square or t-test, as appropriate. A linear regression analysis was performed to verify the independent association of relevant covariables.

Results: 70 patients (71.4% women) were included (Table 1) with a mean age at IRD and ILD diagnosis of 56.2±12.1 and 59.4±11.4 years, respectively. The mean time from ILD diagnosis to NTD initiation was 4.9±3.7 years. The most common underlying IRDs were SSc (38, 54.3%) and rheumatoid arthritis (19, 27.1%). Usual interstitial pneumonia (UIP) was identified in 39 (55.7%) cases, nonspecific interstitial pneumonia (NSIP) in 30 (42.9%) and organizing pneumonia in one (1.4%). A pulmonary artery systolic pressure over 20 mmHg was found in 38 (54.3%) patients, with pulmonary hypertension (PH) confirmed by right heart catheterization in 19 of them. All patients had previously received immunosuppressive therapy, most commonly glucocorticoids (50, 71.4%) and mycophenolate mofetil (29, 41.4%). Mean NTD treatment duration was 23.9±16.2 months, with 26 patients receiving treatment for more than 24 months. Baseline single-breath diffusing capacity of the lungs for CO (DLCO) was 39.6±16.3%, increasing significantly after 6 months to 42.8±18.0% (p=0.046), but not in the other time points (Figure 1). Baseline forced vital capacity (FVC) was 66.5±20.6%, with a trend to stabilisation, but with no significant differences during follow-up. Concomitant treatment with methotrexate (MTX) was the only variable associated with DLCO variation at 6 months, and a linear regression adjusted for the diagnosis of PH confirmed a positive association between MTX intake and DLCO improvement at 6 months (HR 18.9 95%CI 1.66-36.14, p=0.036). In patients with less than one year of ILD duration (n=13), FVC variation had a significant improvement at 12 months (10.6±8.0 vs -0.7±10.6, p=0.029) which was not observed for the DLCO nor in any other time points. Regarding tolerance: 35 (50%) patients were able to maintain a daily dose of 150 mg bid, 31 (44.3%) reduced to 100 mg bid due to gastrointestinal symptoms (GIS) and 4 (5.7%) discontinued NTD due to hepatotoxicity and GIS. During follow-up 12 (17.1%) patients died after a mean of 24.5±15.9 months.

Conclusion: IRD-ILD patients treated with NTD showed DLCO improvement at 6 months, particularly

TO 202 – Table 1. Clinical features of patients with Inflammatory Rheumatic Disease-associated Interstitial Lung Disease treated with Nintedanib

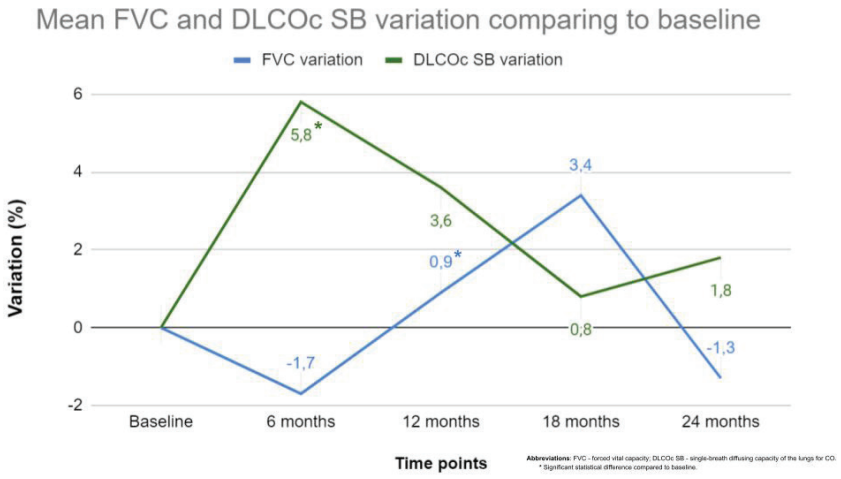
	N = 70
Female, n (%)	50 (71.4%)
Smoke status, n (%)	
• Never smoked	41 (58.6%)
• Smoker	11 (15.7%)
• Ex-smoker	18 (25.7%)
Age at diagnosis of the IRD, mean±SD, range (years)	56.2±12.1, 32-81
Inflammatory Rheumatic Disease (IRD), n (%)	
• Systemic sclerosis	38 (54.3%)
• Rheumatoid arthritis	19 (27.1%)
• Sjögren's syndrome	6 (8.6%)
• Mixed connective tissue disease	2 (2.9%)
• Others (1)	5 (7.1%)
Age at diagnosis of the ILD, mean±SD, range (years)	59.4±11.4, 33-81
Interstitial lung disease (ILD) on computed tomography, n (%)	
• Usual interstitial pneumonia	39 (55.7%)
• Nonspecific interstitial pneumonia	30 (42.9%)
• Organizing pneumonia	1 (1.4%)
Time from IRD diagnosis to ILD diagnosis, mean±SD (years)	3.2±5.9
Autoantibodies, n (%)	
• Antinuclear antibody	56 (80%)
• Anti Scl70	31 (44.3%)
• Rheumatoid factor	23 (32.9%)
• Anticyclic-citrullinated protein antibody	20 (28.6%)
• Anti Ro52	10 (14.3%)
• Anti-Ro60	9 (12.9%)
• Anti-RNP	5 (7.2%)
• Anticentromere	4 (5.8%)
• Anti-dsDNA	3 (4.2%)
• Antisynthetase antibodies	3 (4.2%)
• Anti-PM/Scl	3 (4.2%)
• Anti-RNA polymerase III	2 (2.9%)
• Anti-nucleosome	1 (1.4%)
• Anti-myeloperoxidase	1 (1.4%)
Time for NTD initiation after ILD diagnosis, mean±SD, range (years)	4.9±3.7, 0-19
NTD treatment duration, mean±SD, range (months)	23.9±16.2, 2-67
Adverse effects (associated with NTD), n (%)	
• Nausea	13 (18.6%)
• Vomits	10 (14.3%)
• Diarrhoea	35 (50%)
• Hepatotoxicity	7 (10%)
• Anorexia	5 (7.1%)
• Weight loss	3 (4.3%)
• Others (2)	5 (7.1%)
Nintedanib tolerated dose, n (%)	
• 150 mg twice a day	35 (50%)
• 100 mg twice a day	31 (44.3%)
• Temporary suspension needed	14 (20%)
• Definitive suspension needed	4 (5.7%)
Concomitant glucocorticoids, n (%)	50 (71.4%)
Concomitant csDMARDs, n (%)	
• Mycophenolate mofetil/Mycophenolic acid	35 (50%)
• Methotrexate	11 (15.7%)
• Hydroxychloroquine	10 (14.3%)
• Cyclophosphamide	6 (8.6%)
• Azathioprine	4 (5.8%)
• Sulfasalazine	2 (2.9%)
• Leflunomide	1 (1.4%)

continues on the next pagw

TO 202 – Table 1. Continuation

	N = 70
Concomitant bDMARDs, n (%)	
• Rituximab	28 (40%)
• Abatacept	1 (1.4%)
FVC, mean±SD % (n/N)	
• baseline	66,5±20,6 (69/70)
• 6 months	66,5±16,9 (33/70)
• 12 months	69,1±16,9 (35/70)
• 18 months	66,2±20,8 (12/70)
• 24 months	62,5±19,7 (19/70)
DLCOc SB, mean±SD % (n/N)	
• baseline	39,6±16,3 (53/70)
• 6 months	42,8±18,0 (28/70)
• 12 months	41,9±16,6 (29/70)
• 18 months	39,6±11,0 (8/70)
• 24 months	37,9±13,5 (13/70)
Pulmonary Hypertension, n/N (%)	
• PSAP > 20 mmHg	38/39 (97.4%)
• Confirmation by right heart catheterization	19/37 (51.4%)
Radiographic progression (3) at 12 months, n/N (%)	
• Worsening	22/62 (35.5%)
• Stabilisation	40/62 (64.%)
• Improvement	0/62 (0%)
Deaths, n (%)	12 (17.1%)

Abbreviations: bDMARD - biological disease modifying anti-rheumatic drugs; csDMARD - conventional disease-modifying antirheumatic drugs; FVC - forced vital capacity; DLCOc SB - single-breath diffusing capacity of the lungs for CO; ILD - Interstitial Lung Disease; IRD - Inflammatory Rheumatic Disease; NTD - Nintedanib; SD - Standard Deviation.
(1) Others - dermatomyositis, microscopic polyangiitis, polymyositis, rheumatoid arthritis and systemic sclerosis overlap syndrome, systemic lupus erythematosus.
(2) Others - abdominal cramps, colitis, constipation, epigastralgia, lower limbs oedema.
(3) Improvement or worsening defined as a change of at least 10% in pulmonary fibrosis by an experienced radiologist.



TO 202 – Figure 1. Mean forced vital capacity and single-breath diffusing capacity of the lungs for CO variations up to 24 months of follow-up

in cases of concomitant MTX intake. An improvement in FVC was also observed at 12 months for patients with less than one year of disease, pointing towards a benefit in early initiation of treatment. Although, only half of the patients were assessed at 6 months and even less at the other time points, which restricts the conclusions and could potentially explain the inconsistency of the outcomes.

208 - THE LINK BETWEEN HYPERURICEMIA AND PSORIATIC ARTHRITIS: A DISTINCT CLINICAL PROFILE?

Pedro Miguel Teixeira[1, 2], Carolina Vilafanha[1, 2], Susana Silva[1, 2], Sofia Ferreira Azevedo[1, 2], Carolina Mazeda[1, 2], C. Pinto Oliveira[1, 2], Gisela Eugénio[1, 2], Anabela Barcelos[1, 2]
[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Background: Hyperuricemia is commonly associated with Psoriatic arthritis (PsA), although it is uncertain whether it is directly correlated with the severity of psoriatic skin lesions, chronic inflammation or as a result of coexisting comorbidities. It is also unclear whether hyperuricemia is linked with a particular clinical phenotype of PsA or a more destructive disease.
Objective: To investigate the relationship between hyperuricemia and Psoriatic Arthritis' clinical characteristics and outcomes
Methods: A single-centre, cross-sectional study was conducted, involving adult patients with PsA who met the Classification for Psoriatic Arthritis (CASPAR) cri-

TO 208 – Table 1. Demographics and clinical characteristics associated with hyperuricemia in PsA patients.

	With hyperuricemia (n=46)	Without hyperuricemia (n=120)	Significance p
Mean age (SD) (years)	60.5 (11.1)	54.32 (11.8)	0.002
Women, n (%)	7 (15.2%)	59 (49.2%)	<0.001
Body mass index (SD) (kg/m^2)	28.7 (4.1)	28.3 (4.6)	0.612
Serum uric acid (mg/dL) (SD)	7.6 (0.55)	5.1 (0.61)	<0.001
Psoriasis, n (%)	46 (100%)	118 (98.3%)	0.905
Psoriasis disease duration (SD) (years)	21.6 (2.4)	21.2 (1.5)	0.896
PsA disease duration (SD) (years)	12.1 (1.4)	11.3 (0.7)	0.573
HLA-27, n (%)	3 (6.5%)	10 (8.3%)	0.663
ESR (mm/h) (Q1-Q3)	17 (13-24)	14 (11-20)	0.605
CRP (mg/dL) (Q1-Q3)	0.73 (0.45-1.06)	0.48 (0.33-0.62)	0.115
DAPSA, mean (SD)	10.2 (1,9)	8.00 (0.8)	0.296
Type of involvement			
Peripheric, n (%)	37 (80.4%)	105 (87.5%)	0.247
Oligoarticular, n (%)	19 (41.3%)	38 (31.7%)	0.242
Polyarticular, n (%)	17 (37%)	63 (52.5%)	0.073
Axial, n (%)	16 (34.8%)	24 (20.0%)	0.046
Peripheric and axial, n (%)	8 (17.4%)	17 (14.2%)	0.522
Enthesitis, n (%)	8 (17.4%)	19 (15.8%)	0.849
Dactylitis, n (%)	11 (23.9%)	27 (22.5%)	0.897
Radiographic damage, n (%)	23 (50.0%)	38 (31.7%)	0.028
Ongoing treatment			
No treatment, n (%)	4 (8.7%)	9 (7.5%)	0.797
NSAIDs, n (%)	4 (8.7%)	23 (19.2%)	0.102
PDN daily dose ≥ 5mg, n (%)	10 (21.7%)	18 (15.0%)	0.299
csDMARD alone, n (%)	20 (43.5%)	57 (47.5%)	0.644
bDMARD alone, n (%)	13 (28.3%)	31 (25.8%)	0.751
csDMARD+bDMARD, n (%)	7 (15.2%)	15 (12.5%)	0.644

bDMARD: biologic disease-modifying anti-rheumatic drugs; csDMARD: conventional synthetic disease-modifying anti-rheumatic drugs; CRP: C-reactive protein; DAPSA: Disease Activity in Psoriatic Arthritis; ESR: erythrocyte sedimentation rate; NSAIDs: non-steroidal anti-inflammatory drugs; PDN: Prednisolone; PsA: Psoriatic Arthritis; Q1-Q3: quartile 1 to 3; SD: standard deviation

teria. Demographic, clinical, laboratory, and treatment outcome data were collected. Hyperuricemia was characterized by serum uric acid levels exceeding 6.8mg/dL, and the participants were subsequently categorized into two groups based on this criterion. A descriptive analysis and comparison between both groups was made using both parametric and non-parametric tests followed by univariate and multivariate logistic regression. Statistical significance was set at a p-value ≤ 0.05.
Results: The study comprised a cohort of 166 patients, out of which 46 (27.7%) were identified as having hyperuricemia with a mean serum uric acid of 7.6±0.5mg/dL. Nearly every patient had a personal history of psoriasis (98%). Univariate analysis revealed significant differences when comparing hyperuricemic patients to normal-uricemic patients: hyperuricemic patients were more commonly male (84.8% vs 50.8%, p<0.001), older (60.5 vs 54.3, p=0.002) and had more axial involvement (34.8% vs 20.0%, p = 0.046). Although there were no differences regarding current disease activity and pharmacological treatment, patients with hyperuricemia had more established radiographic damage (50.0% vs 31.7%, p=0.028). Multivariable analysis confirmed that hyperuricemic PsA was indeed independently associated with more radiographic lesions (odds ratio 1.83; 95% CI 1.10–3.76; p = 0.045).
Conclusion: Our study suggests that hyperuricemia is associated with certain clinical features of PsA, including a higher prevalence in males, older age, and increased axial involvement, and may be linked to a more severe and progressive disease phenotype in PsA. Further research is warranted to explore the underlying mechanisms and potential therapeutic implications of this association.

210 - LINFOMA NÃO-HODGKIN COMO COMPLICAÇÃO DA SÍNDROME DE SJÖGREN PRIMÁRIA: UMA SÉRIE DE CASOS DE UM SERVIÇO DE REUMATOLOGIA

Sara Alves Costa[1], Ana Isabel Maduro[1], Filipa Canhão André[1], André Saraiva[1], Beatriz Mendes[1], Adriana Carones[1], Fernando Albuquerque[1], Marcelo Neto[1], Maria João Cadório[1], João Alexandre Oliveira[1], Sara Serra[1]
[1]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal

Introdução: A Síndrome de Sjögren Primária (SSP) é uma doença autoimune sistémica que afeta principalmente as glândulas exócrinas, levando ao comprometimento da sua função. O linfoma não-Hodgkin (LNH) constitui a complicação mais grave do SSP e ocorre em cerca de 5–10% dos doentes, com um risco maior que a população em geral. Várias características clínicas e serológicas têm sido propostas como preditores de linfoma em pacientes com SSP.
Objetivos: Descrever a subpopulação de doentes com SSP que desenvolveram LNH quanto às principais manifestações clínicas e serológicas, incluindo fatores preditores relatados na literatura. Caracterizar os LNH nestes doentes (tipo, local e estadiamento), terapêutica realizada, taxa de remissão total e parcial, bem como taxa de sobrevida e de recidiva.
Métodos: Análise retrospetiva descritiva de doentes com SSP que desenvolveram LNH, seguidos regularmente no Serviço de Reumatologia do Centro Hospitalar e Universitário de Coimbra, através da consulta do processo clínico e Reuma.pt, com dados de 1999 a 2023. Os doentes incluídos cumpriam os critérios de classificação ACR/EULAR 2016 e/ou AECG 2002 para SSP. Foram revistas as características demográficas, clínicas e serológicas (nomeadamente fatores preditores de LNH), assim como as características relacionadas com o LNH.
Resultados: Dos 148 doentes com SSP, 6 (4.1%) desenvolveram LNH (4 do sexo feminino, com uma mediana de idades ao diagnóstico de 64 anos). Relativamente às características clínicas dos doentes com LNH, 3 (50%) apresentavam tumefação parotídea persistente e 4 (66.7%) linfadenopatias. Nenhum doente apresentou púrpura/vasculite cutânea, envolvimento renal ou neurológico. Por outro lado, verificou-se a presença de linfopenia, gamopatia monoclonal e hipocomplementémia em 3 (50%) casos. As alterações imunológicas mais frequentemente encontradas foram, respetivamente, a positividade para ANAs (n=6, 100%), anticorpos anti-SSA (n=5, 83.3%), fator reumatóide (FR) (n=4, 66.7%) e anticorpos anti-SSB (n=3, 50%). No que respeita à caracterização dos LNH, o tempo médio desde o diagnóstico de SSP até ao diagnóstico de LNH variou entre 0.5 e 18 anos. O LNH B de grandes células (n=2, 33.3%) e o linfoma MALT (n=2, 33.3%) foram os subtipos mais frequentes, e o estadio (de acordo com a classificação de Ann Arbor) variou entre II (n=1), III (n=2) e IV (n=3). Destaque para a localização do linfoma nas glândulas parótidas (n=4; 66.7%). O tratamento mais utilizado foi o Rituximab em combinação com agentes quimioterápicos (esquema R-CVP ou R-CHOP) (n=5; 83.3%). Verificou-se uma taxa de remissão total de 33%. A taxa de recidiva foi de 33%, com uma taxa de sobrevida de 83% até à data, com um óbito 3 anos após o diagnóstico de linfoma.
Conclusão: Nesta coorte verificou-se que 4% dos doentes com SSP desenvolveram LNH. Estes doentes apresentavam vários dos fatores preditores clínicos e/ou serológicos descritos na literatura, destacando-se a linfadenopatia, o anticorpo anti-SSA e o fator reumatóide (observados em mais de 50% dos casos). Um dos

LNH mais frequente foi o linfoma MALT e as glândulas parótidas constituíram a localização predominante, também de acordo com o relatado em outros estudos. A evolução para malignidade, particularmente para LNH, é uma complicação que, embora rara, é potencialmente fatal, o que reforça a importância de uma vigilância e monitorização regular destes doentes, especialmente aqueles com fatores preditores de desenvolvimento de LNH.

211 - MACROPHAGE ACTIVATION SYNDROME – CLINICAL FEATURES AND TREATMENT OUTCOMES IN SLE AND AOSD PATIENTS

Vanessa Fraga[1], Sandra Sousa[1], Maria José Santos[1, 2]
[1]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Macrophage Activation Syndrome (MAS) is a life-threatening complication observed in some rheumatic diseases, characterized by uncontrolled immune activation and a cytokine storm. Despite its clinical significance and importance of early diagnosis, there remains a need for a comprehensive characterization and treatment outcomes.
Objectives: We aim to characterize the clinical features, diagnostic approaches, management, laboratory results and outcomes of MAS in adult rheumatic patients, followed in our center.
Methods: We performed a retrospective analysis of adult patients with systemic rheumatic diseases diagnosed with MAS in the previous 10 years in our rheumatology department. Demographic characteristics, clinical manifestations, laboratory findings, treatment approach and patient outcomes are reported.
Results: A total of 4 patients with MAS were identified, with a male-to-female ratio of 1:1 and a mean age of 26 ± 5 years at MAS diagnosis. Among these patients, 2 had underlying Systemic Lupus Erythematosus (SLE) and the other 2 had Adult Onset Still Disease (AOSD). Notably, MAS occurred as part of the disease presentation in half of the patients.
Regarding clinical features, all patients presented with high grade fever, arthralgias/arthritis and signs of hemodynamic instability such as hypotension and/or tachycardia. Two patients developed respiratory failure and required transfer to an intensive care unit due to the need for high-flow oxygen or mechanical ventilation. Other clinical features included hepatosplenomegaly, lymphadenopathy, skin rash and odynophagia, present in AOSD patients. Central nervous system was

Table 1. Demographics, clinical features, treatment and outcomes in MAS, MAS-SLE and MAS-AOSD

	MAS (n=4)	
Demographics		
Sex (female)	2 (50%)	
Age, mean ± SD, years	26.25 ±5.5	
Previous immunosuppression	2 (50%)	
Hydroxychloroquine	1 (25%)	
Mycophenolate mofetil	1 (25%)	
Sulfasalazine	1 (25%)	
Clinical features		
Fever	4 (100%)	
Arthralgias/arthritis	4 (100%)	
Hypotension and/or tachycardia	4 (100%)	
Lymphadenopathy	3 (75%)	
Hepatosplenomegaly	2 (50%)	
Skin rash	2 (50%)	
Odynophagia	2 (50%)	
Respiratory failure	2 (50%)	
Central Nervous System involvement	1 (25%)	
Laboratory findings during MAS (left) and 3-months follow-up (right), mean ± SD		
Hemoglobin (g/dL)	8.07 ± 0.71	12.57 ± 0.45
Platelets (10^9/L)	154.25 ± 63	345.75 ± 73
Leukocytes (10^9/L)	5.67 ± 2.35	7.4 ± 2.64
ESR (mm/h)	75.25 ± 40.12	62 ± 29.51
CRP (mg/dL)	4.13 ± 3.07	0.4 ± 0.35
D-Dimer (ug/mL)	4.53 ± 4.59	N/A
Fibrinogen (mg/dL)	387 ± 369	N/A
AST (UI/L)	163.75 ±71.19	18.75 ± 3.2
ALT (UI/L)	76.5 ± 32.37	22 ± 8.52
Triglycerides (mg/dL)	290 ± 92.04	129.5 ± 95.29
Ferritin (ng/mL)	6886.5 ± 4626	56 ± 46.85
LDH (IU/L)	673 ± 94.23	219 ± 35.55
CD25 soluble[1] (U/mL)	6933 ± 642*	N/A
Diagnostic criteria		
HScore[2], mean ± SD, points	213.24 ±24.66	
HLH 20043	1 (25%)	
Treatment		
High dose glucocorticoids[4]	4 (100%)	
Anakinra	2 (50%)	
Cyclophosphamide	1 (25%)	
Rituximab	1 (25%)	

1Only available in 2 patients. 2 Diagnosis of MAS was made when HScore > 169 points. 3 Number of patients who fulfilled ≥ 5 out of 8 items of Hemophagocytic lymphohistiocytosis (HLH) 2004 criteria. 4 Methylprednisolone pulses followed by prednisolone 1mg/kg/day, except 1 patient that did not require glucocorticoid pulse therapy first. AST: Aspartate aminotransferase, ALT: Alanine transaminase, CRP: C Reactive Protein; ESR: Erythrocyte Sedimentation Rate; LDH: Lactate Dehydrogenase, N/A: not available;

involved in 1 patient, who presented with headache and altered mental status. The demographics, clinical features, treatment, laboratory results and outcomes are presented in table 1.
Regarding laboratory results, all patients showed anemia, significantly elevated ferritin levels, elevated inflammatory markers, liver enzymes, triglycerides and LDH. Diagnosis of MAS was established using HScore, with a mean score of 213 ± 24.66 points. Only 1 patient fulfilled the 2004 Hemophagocytic Lymphohistiocytosis (HLH) diagnostic criteria. All patients were promptly treated with systemic glucocorticoids, 75% received pulsed therapy, with clinical and analytical improvement. Regarding immunosuppression, AOSD patients received anakinra and SLE patients received rituximab or cyclophosphamide. One AOSD patient later experienced an underlying disease flare while on anakinra and a switch to tocilizumab was performed. All patients are in clinical remission with a mean follow-up of 29 ± 6 months after the MAS episode.
Discussion: MAS is a life-threatening complication seen in some rheumatic diseases such as SLE and AOSD and can be part of the initial clinical presentation. Common symptoms include high-grade fever, joint pain, low blood pressure, lymphadenopathy, and hepatosplenomegaly. Laboratory findings often reveal cytopenias, elevated inflammatory markers, ferritin, triglycerides, and liver enzymes. In our cohort, HScore assessment demonstrated higher sensitivity than HLH 2004 criteria, for diagnosing MAS. Treatment with high-dose glucocorticoids and immunosuppression was effective. High clinical suspicion and early treatment are fundamental for good outcomes.

212 - CAN IDIOPATHIC CARPAL TUNNEL SYNDROME BE ASSOCIATED WITH CARDIAC AMYLOIDOSIS IN ELDERLY PATIENTS? RESULTS FROM CARPOS STUDY

Ana Martins[1, 2], Sofia Pimenta[1, 2], Bárbara Pereira[3], Micaela Gonçalves[4], Miguel Carvalho[5], Luís Santos[5], Elsa Fonseca[6], Isabel Pinto[4], António Sousa[4], Pedro Madureira[1], Isabel Fidalgo[1], Lúcia Costa[1], Jorge Pereira[3], Teresa Faria[3], Elisabete Martins[2, 5]

[1]Serviço de Reumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [3]Serviço de Medicina Nuclear, Centro Hospitalar Universitário de São João, Porto, Portugal, [4]Serviço de Ortopedia e Traumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [5]Serviço de Cardiologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [6]Serviço de Anatomia Patológica, Centro Hospitalar Universitário de São João, Porto, Portugal

Background: Carpal tunnel syndrome (CTS) is the most prevalent entrapment neuropathy, affecting 1 to 5% of the general population, mostly patients older than 50 years. Several conditions can cause or increase the risk of CTS. However, most of the cases are considered idiopathic. According to previous studies, the prevalence of amyloid deposits, especially wild-type transthyretin (TTRwt) amyloid, in the tenosynovium of patients with idiopathic CTS is high (up to 10%) and CTS is one of the most common manifestations of amyloidosis and frequently appears in earlier stages of the disease, prior to cardiac involvement.
Objectives: To explore the association between idiopathic CTS, amyloid deposits in the tenosynovium and the presence of cardiac amyloidosis.
Methods: A prospective study of patients aged 60 years or older, with bilateral symptomatic CTS proposed for carpal tunnel (CT) release surgery and followed in a tertiary hospital was conducted. Patients younger than 60 year, with unilateral CTS, diabetes mellitus, hypothyroidism, chronic renal failure under haemodialysis, inflammatory arthritis, multiple myeloma and previous trauma of the wrist were excluded. All patients were submitted to a physical examination, laboratory work-up, nerve conduction studies, wrist ultrasound, electrocardiogram and cardiac scintigraphy with Technetium-99m-DPD before the CT release surgery. Wrist ultrasound was performed by the same rheumatologist and the measure of the median nerve cross-sectional area at the tunnel inlet was calculated. Tenosynovial tissue was collected at the time of the surgery, whenever possible, and Congo red staining to identify amyloid deposition was performed. Diagnosis of transthyretin cardiac amyloidosis was based on a high-grade cardiac uptake (Perugini grade 2 or 3) on cardiac scintigraphy.
Results: A total of 20 patients (12 females, 60.0%, mean age of 72.5 ± 8.8 years) were included. The mean age at onset of CTS was 65.9 ± 9.8 years and the mean age at diagnosis was 68.5 ± 11.0 years. Most of the patients had moderate to severe bilateral CTS (n=14, 70.0%), based on nerve conduction studies, and a median nerve cross-sectional area at the tunnel inlet at the wrist ultrasound superior to 10 mm2 in both hands (n=12, 60.0%). Three of the 9 (33.3%) patients submitted to a tenosynovial biopsy had amyloid deposits. Four (20.0%) patients had a cardiac scintigraphy with a high-grade cardiac uptake (Perugini grade 2 in 1 patient and grade 3 in 3 patients). Of these patients, two was submitted to a tenosynovial biopsy and one of them had amyloid deposits.
Conclusion: In our study, 3 of the 9 patients with bilateral idiopathic CTS had amyloid deposits in tenosynovial biopsy and 4 of the 20 patients had cardiac amyloidosis. Although these results are still preliminary and

TO 212 – Table 1. Sociodemographic data, characteristics of the carpal tunnel syndrome, ECG abnormalities, cardiac scintigraphy findings and results of the tenosynovial tissue biopsy of the included patients.

	Participants (N=20)
Female, n (%)	12 (60.0)
Age (years), mean ± SD	72.5 ± 8.8
Current/Former smoker, n (%)	3 (15.0)
Alcohol consumption, n (%)	1 (5.0)
Comorbidities, n (%)	
Hypertension	13 (65.0)
Dyslipidemia	11 (55.5)
Cerebrovascular disease	1 (5.0)
Coronary heart disease	2 (10.0)
Osteoarthritis	4 (20.0)
Characteristics of Carpal Tunnel Syndrome	
Age at onset (years), mean±SD	65.9 ± 9.8
Age at diagnosis (years), mean ± SD	68.5 ± 11.0
Severity based on EMG	
Right hand (n=19)	3 (15.8)
Mild, n (%)	7 (36.8)
Moderate, n (%)	9 (47.4)
Severe, n (%)	
Left hand (n=20)	
Mild, n (%)	5 (25.0)
Moderate, n (%)	8 (40.0)
Severe, n (%)	7 (35.0)
Wrist ultrasound	
Median nerve cross-sectional area at the tunnel inlet	
Right hand (n=19)	
< 10 mm^2, n (%)	5 (26.3)
10-15 mm^2, n (%)	12 (63.2)
>15 mm^2, n (%)	2 (10.5)
Left hand (n=19)	
< 10 mm^2, n (%)	6 (31.6)
10-15 mm^2, n (%)	9 (47.4)
>15 mm^2, n (%)	4 (21.1)
Previous CT release surgery, n (%)	4 (20.0)
Laboratory work-up	
Troponin levels (ng/L), n (%)	
Negative (<16)	18 (90.0)
Positive (>16)	2 (10.0)
ECG abnormalities, n (%)	
Normal	13 (65.0)
First-degree atrioventricular block	1 (5.0)
Nonspecific ventricular repolarization abnormalities	1 (5.0)
Left anterior fascicular block	2 (10.0)
Low QRS voltage	4 (20.0)
Scintigraphy with Technetium-99m-DPD – Perugini Grading System, n (%)	
0	16 (80.0)
2	1 (5.0)
3	3 (15.0)
Tenosynovial tissue biopsy (n=9)	
Presence of amyloid deposits, n (%)	3 (33.3)

Legend: CT. Carpal tunnel, ECG. Electrocardiogram, EMG. Electromyography

with a small sample, patients with bilateral idiopathic CTS seem to have a high prevalence of amyloid deposition in the tenosynovium and a high prevalence of cardiac amyloidosis. This study highlights the importance of screening for cardiac amyloidosis in patients with CTS, especially in patients aged 60 years or older with bilateral idiopathic CTS.
CTS may be a possible predictor of cardiac amyloidosis and the screening for cardiac amyloidosis may allow the diagnosis at an earlier stage, which will significantly improve the prognosis of these patients. Further prospective studies with larger samples are needed.

213 - IGA NEPHROPATHY ASSOCIATED WITH SPONDYLOARTHRITIS – CLINICAL AND IMMUNOPATHOLOGICAL FEATURES OF A PORTUGUESE COHORT

Susana P. Silva[1, 2], Marília Rodrigues[3], Carolina Ochôa Matos[4], Bruna Silva[5], Miguel Bernardes[6], Gisela Eugénio[1, 2], Anabela Barcelos[1, 2]
[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar de Leiria, Leiria, Portugal, [4]Serviço de Reumatologia, Centro Hospitalar Lisboa Norte - Hospital de Santa Maria, Lisboa, Portugal, [5]Serviço de Endocrinologia do Centro Hospitalar Tâmega e Sousa, [6]Rheumatology Department, Centro Hospitalar Universitário de São João, Porto, Portugal

Introduction: Spondyloarthritis (SpA) is a heterogeneous group of inflammatory rheumatic diseases, infrequently associated with IgA nephropathy (IgAN). However, its pathophysiology remains not fully understood, and it is unknown whether SpA IgAN differs from primary IgAN in its clinical presentation, renal function, and prognosis. Besides, literature also describes a controversial relationship between IgAN and biotechnological therapy. Here, we describe the prevalence of spondyloarthritis-associated IgAN in our population.
Material and Methods: Retrospective, multicenter observational study including patients meeting ASAS criteria for axial spondyloarthritis and peripheral spondyloarthritis and/or CASPAR criteria for Psoriatic Arthritis, diagnosed with IgA nephropathy by biopsy. Sociodemographic data, disease characterization, and treatment information were collected between May and December 2022.
Results: Five patients were included (4 were male) with 58.5 (22.04) years old, of which 3 had Ankylosing Spondylitis (AS) and 2 had Psoriatic Arthritis (PsA). All patients had extra-articular manifestations, including psoriasis (n=1), uveitis (N=2), dactylitis (n=1), and enthesitis (n=1). Regarding comorbidities and risk factors for kidney injury, 3 patients had hypertension, 2 had dyslipidemia and hyperuricemia, and another had a history of renal lithiasis. None had chronic kidney disease or previous history of acute kidney lesions. Before the beginning of nephropathy, patients were treated with non-steroid anti-inflammatory drugs (NSAIDs) (n=1), corticosteroids (n=1), methotrexate (n=1), and leflunomide (n=1). Most of the patients were treated with anti-tumor necrosis factor (anti-TNF): 2 with adalimumab and one with golimumab. In all patients, rheumatic disease preceded nephropathy, except for one case, which started 12 months before the beginning of axial symptoms. In 3 patients, kidney disease was asymptomatic, with the fortuitous discovery of proteinuria (n=4) and microhematuria (n=3). Two patients had new-onset hypertension and one of them also reported macroscopic hematuria. All patients had an initial estimated glomerular filtration rate (eGFR) superior to 60ml/min/1.73m2. The mean proteinuria at diagnosis was 1110 ± 648 mg/24h. All patients had IgA Nephropathy confirmed by biopsy and 4 (80%) were treated with corticosteroids. NSAIDs were completely stopped in one patient. Anti-TNF (adalimumab) was also suspended in another patient. Two patients presented with nephrotic proteinuria during the disease course. Two patients started adalimumab during follow-up, including the patient who re-initiated it, without effects on kidney function. In the last evaluation, most patients (n=3) had eGFR between 30 and 60 ml/min/1.73m2 and only one had eGFR of more than 90 ml/min/1.73m2. None of the patients presented with rapidly progressive renal dysfunction or developed end-stage renal disease during 9 ± 8.83 years of follow-up.
Discussion/Conclusion: This retrospective study highlights the low prevalence of spondyloarthritis-associated IgA nephropathy, although it can also be underdiagnosed due to the absence of renal biopsy in possible cases. Unlike the literature, it was not associated with poor outcomes and anti-TNF didn't seem to influence its course.

216 - RHEUMATOLOGY AND PATHOLOGY MULTIDISCIPLINARY CLINIC: INNOVATION IN RHEUMATIC PATIENTS CARE THROUGH SYNOVITIS DIAGNOSTIC PRECISION

Vanessa Sílvia Fernandes[1], Carolina Ochôa Matos[1, 2, 3], Mafalda Pinho[4, 5], Rita Luís[4, 5, 6], Emília Vitorino[4], Elsa Vieira-Sousa[1, 2, 3]
[1]Clínica Universitária de Reumatologia, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas,

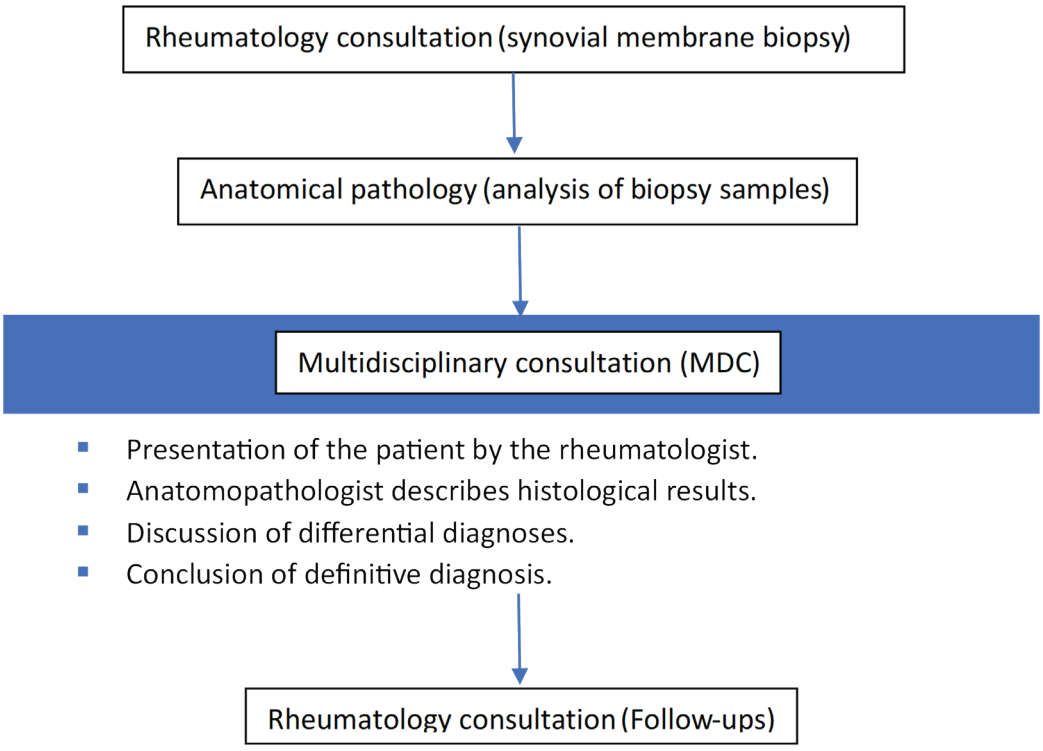
Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Unidade de Investigação em Reumatologia - JE Fonseca Lab, Instituto de Medicina Molecular João Lobo Antunes, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [4]Serviço de Anatomia Patológica, Centro Hospitalar e Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [5]Instituto de Anatomia Patológica, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [6]Serviço de Anatomia Patológica, Centro Hospitalar e Universitário Lisboa Central, Lisboa, Portugal

Background: Synovitis is considered the primordial pathology in many rheumatic diseases. Synovial biopsies can be of help to establish a definite diagnosis of synovitis. Pathologists assist rheumatologists through the analysis of the histology of the synovial membrane, which is often difficult to interpret. Multidisciplinary care of rheumatic patients across these two specialities is seen as having good potential for clinical benefit, but very little research has been published in this field. The Centro Hospitalar e Universitário de Lisboa Norte (CHULN) Rheumatology and Pathology Multidisciplinary Clinic (MDC) was implemented in 2016 to facilitate the discussion about synovitis classification and improve rheumatic patients' care.

Objectives: To describe the workflow of the Rheumatology-Pathology MDC established at CHULN. To report for the first time, the outcomes from this joint clinic on a proof-of-principle cohort.

Methods: Approximately every two months, rheumatologists and pathologists meet to discuss referred patients (2-5 patients on average) submitted to a synovial biopsy performed either by miniarthroscopy or ultrasound-guided techniques. Clinical, imaging, and histopathology data are integrated in these discussions. The workflow of these MDC consultations is presented in Figure 1. Patients' demographic data, histopathology parameters, pre- and post-discussion diagnoses, and pre- and post-medications were collected and analysed.

Results: A total of 30 patients were discussed from 2016 to 2021 at the rheumatology-pathology MDC of CHULN. The patients' ages varied between 16 and 87 years, the average being 50 years, with a gender distribution of 53% male and 47% female. Of the 37 biopsies analysed, 48.6% were obtained by ultrasound guidance, 37.9% by miniarthroscopy, and one (2.7%) using blind needle, totalizing 35 synovial tissue samples. Exceptionally, one pericardial (2.7%) and one salivary gland biopsies (2.7%) were also discussed. For the synovial membrane cases, the predominantly biopsied joints were the knee (48.6%), the wrist (18.8%), and the elbow (16.2%). Pre-MDC diagnoses were ruled out in 12.5%, confirmed in 52.5%, changed in 35%, and



TO 216 – Figure 1. Methodology of the MDC consultations at CHULN

new pathologies were identified in 15% of patients. Consequently, the pre-MDC medications were discontinued in 12.2%, confirmed in 81.6%, changed in 6.1%, and added in 20.4% after MDC.
Conclusion: This work underlines the added value of a Rheumatology and Pathology multidisciplinary consultation through the description of a proof-of-principle cohort assessed over a period of 6 years. It also highlights the relevance of the study of the synovial membrane and its potential to establish rheumatic diseases' definite diagnosis.
Acknowledgments: To Prof. João Eurico Fonseca, Rheumatology Department Director, and Dra. Cristina Ferreira, Pathology Department Director, for their support in the implementation and dynamization of this MDC clinic.

217 - TRATAMENTO COM ABATACEPT NAS DOENÇAS REUMÁTICAS INFLAMATÓRIAS: A EXPERIÊNCIA DE UM CENTRO TERCIÁRIO

Carla Campinho Ferreira[1], Paulo Pereira[1], Ana Margarida Correia[1], Emanuel Costa[1], Diogo Esperança Almeida[1], Joana Sousa-Neves[1], Marcos Cerqueira[1], José Redondo[1], Ana Ribeiro[1], Joana Leite Silva[1]
[1]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal

Introdução: O Abatacept é uma proteína de fusão recombinante, constituída pelo domínio extracelular do CTLA-4 humano e a região Fc modificada da IgG1 humana, que atua inibindo seletivamente a ativação das células T, através da ligação específica ao CD80 e CD86. Pertence à classe dos bDMARDs, sendo que na Europa se encontra aprovado para o tratamento da Artrite Reumatoide (AR), Artrite Psoriática (APso) e Artrite Idiopática Juvenil (AIJ) Poliarticular.
Objetivos: Caracterizar os doentes com doença reumática tratados com Abatacept, seguidos num serviço de reumatologia de um centro terciário português, até maio de 2023.
Métodos: Estudo retrospetivo e unicêntrico. Foram incluídos todos os doentes com mais de 18 anos, com diagnóstico de doença reumática inflamatória, que foram tratados com Abatacept até maio de 2023. Foram colhidas e analisadas retrospetivamente variáveis sociodemográficas, os dados clínicos e analíticos à baseline (data de início do fármaco), aos 3, 6 e 12 meses. As variáveis contínuas são apresentadas como média ± desvio padrão para os dados normalmente distribuídos ou mediana ± intervalo interquartil se ausência de distribuição normal. As variáveis categóricas são apresentadas em número absoluto ou percentagem.
Resultados: Foram identificados um total de 12 doentes com tratamento prévio ou em curso com Abatacept, sendo 7 (58,3%) do sexo feminino, com idade de 56,7 ± 7,0 anos e um tempo de evolução de doença desde o diagnóstico de 5 ± 14,8 anos. Quanto à presença de comorbilidades, 5 (41,7%) tinham hipertensão, 6 (50%) dislipidemia e 3 (25%) diabetes mellitus tipo 2. Dos 12 doentes incluídos, 10 tinham diagnóstico de AR e 2 de APso. O Abatacept foi o bDMARD de 1ª linha em 3 doentes (25%) e 3ª linha ou mais nos restantes. Relativamente aos doentes com AR, 60% apresentavam fator reumatoide e anticorpos anti peptídeos citrulinados positivos, sendo os restantes 40% seronegativos. O DAS28 PCR 3v à baseline foi de 4,01 ± 1,17, com todos os doentes medicados com pelo menos 1 fármaco modificador de doença reumática convencional sintético (csDMARD) e uma dose de prednisolona de 6,75 ± 4,57 mg/d. Três meses após o início do Abatacept, segundo os critérios de resposta EULAR, 50% dos doentes apresentaram boa resposta

TO 217 – Tabela 1. Caracterização da evolução clínica e analítica dos doentes com AR e APso nos primeiros 12 meses de seguimento.

	0 meses	3 meses	6 meses	12 meses
Artrite Reumatoide	n=10	n=8	n=6	n=6
VS, $\bar{x}$ ± s mm/h	24,2 ± 16,5	22,6 ± 19,0	17,2 ± 7,6	14,3 ± 8,5
PCR, $\bar{x}$ ± s mg/L	11,7 ± 11,0	5,1 ± 4,0	5,0 ± 3,4	5,1 ± 4,4
DAS28 PCR 3v, $\bar{x}$ ± s	4,01 ± 1,17	2,61 ± 1,26	2,11 ± 0,48	2,60 ± 1,26
Dose de PDN, $\bar{x}$ ± s mg/d	6,75 ± 4,57	5,16 ± 2,20	4,58 ± 2,46	4,38 ± 2,47
ARTRITE PSORIÁTICA	n=2	n=2	n=2	n=2
VS, $\bar{x}$ ± s mm/h	4,5 ± 3,5	11,5 ± 14,8	9,0 ± 8,5	7,0 ± 8,5
PCR, $\bar{x}$ ± s mg/L	0,9 ± 0,3	1,95 ± 0,9	9,7 ± 8,0	1,0 ± 0,3
DAS28 PCR 3v, $\bar{x}$ ± s	3,00 ± 1,39	2,31 ± 0,40	2,61 ± 0,50	1,29 ± 0,19
Dose de PDN, $\bar{x}$ ± s mg/d	2,50 ± 3,54	5,00 ± 7,07	5,00 ± 7,07	2,50 ± 3,54

ao tratamento e 50% uma resposta moderada. Na avaliação aos 6 meses, 1 doente tinha suspendido o fármaco, sendo que 3 doentes ainda não atingiram esta duração de tratamento. O DAS 28 PCR era de 2,11 ± 0,48 e a dose de prednisolona de 4,58 ± 2,45 mg/d. Não se verificou suspensão do fármaco entre os 6 e os 12 meses, sendo nesta data o DAS 28 PCR 3v de 2,60 ± 1,26. Na tabela 1, encontra-se detalhada a evolução clínica e analítica, durante os primeiros 12 meses de seguimento, nos doentes com AR. Os 2 doentes com APso sob tratamento com Abatacept encontravam-se em remissão aos 12 meses de tratamento. À data de recolha dos dados, o Abatacept tinha sido suspenso em 3 doentes com AR (uma falência 1ª aos 3 meses, uma falência 2ª aos 12 meses e uma remissão clínica aos 12 anos). Os restantes 9 doentes encontram-se sob tratamento mensal com Abatacept, com um tempo de tratamento médio de 23,0 ± 17,8 meses. Não foram registados efeitos adversos da terapêutica em nenhum dos doentes.

Conclusão: O Abatacept é um bDMARD com aprovação na AR e na APso, apesar de não constar na recomendação ACR/EULAR para o tratamento da APso. Da experiência do nosso centro, o fármaco parece constituir uma opção eficaz no tratamento de doentes com AR e APso, destacando-se o perfil de segurança, uma vez que não foi registado nenhum efeito adverso.

221 - RHEUMATOLOGISTS' AND PULMONOLOGISTS' ATTITUDES AND PRACTICES REGARDING RHEUMATOID ARTHRITIS-ASSOCIATED INTERSTITIAL LUNG DISEASE: NATIONAL SURVEY

Hugo Parente[1], Catarina Soares[1], Sara Paiva Dinis [2], Emanuel Costa[3], Maria Pontes Ferreira[1], Anita Cunha[1], Daniela Santos-Faria[1], Soraia Azevedo[1], Francisca Guimarães[1], José Tavares-Costa[1], Carmo Afonso[1], Filipa Teixeira[1]
[1]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [2]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [3]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal

Introduction: The interstitial lung disease (ILD) is a Rheumatoid Arthritis' (RA-ILD) complication with great morbimortality - the quality of life is comparable to that of idiopathic pulmonary fibrosis[1]. Yet, we lack consensual guidelines on its practical approach, and rheumatologists might undervalue RA-ILD's risk and need for a specialized evaluation[2].

Objectives: To determine and compare rheumatologists' and pulmonologists' opinion/knowledge regarding RA-ILD's risk factors, screening, diagnosis, and treatment.

Methods: Online survey sent via e-mail to the Portuguese Rheumatology Society and the Portuguese Pulmonology Society associates. Comparison between both groups was performed.

Results: We amounted 112 respondents. The response rate was 27% (81/297) for the Rheumatology group and 2.6% (31/1200) for the Pulmonology group. Table 1 depicts both groups' features. Fifty-eight (71.6%) rheumatologists had an interest/differentiation on RA; 25 (80.6%) pulmonologists felt similarly about ILD. Overall, 64.3% respondents have a multidisciplinary rheumatology-pulmonology consultation; 80.6% pulmonologists have a multidisciplinary pulmonology-radiology consultation. Most pulmonologists found usual interstitial pneumonia as the main computed tomography (CT) pattern (64.5%). Seventy-eight (96.3%) rheumatologists routinely screen respiratory symptoms, 79 (97.5%) screen smoking habits, and 63 (77.8%) request chest radiography in newly diagnosed RA. Twenty-nine (93.5%) pulmonologists request a chest CT to newly diagnosed RA-ILD and 29 (93.5%) request lung function tests. More than 90% of respondents estimated RA-ILD's prevalence to be 2-30%; 100% thought ILD increased RA's mortality. More rheumatologists found high titers of rheumatoid factor/anti-citrullinated protein antibodies to be a risk factor (97.5% vs 61.3%, p<0.001), as well as smoking (96.3% vs 77.4%, p=0.004), but more pulmonologists found methotrexate use to be harmful (22.6% vs 6.2%, p=0.019). Thirty-six percent of rheumatologists and 48% of pulmonologists avoid it in RA-ILD. For inflammatory RA-ILD (inf-RA-ILD), more rheumatologists found rituximab (91.4% vs 71.0%, p=0.013) to be effective; and for non-inflammatory RA-ILD (Ninf-RA-ILD), more pulmonologists found mofetil mycophenolate (29.0% vs 9.9%, p=0.018) and azathioprine (22.6% vs 3.7%, p=0.004) effective. Comparing to younger practicing doctors (0-5 years of practice, as well as 6-10, 11-15 and 15-30 years), physicians working >30 years more commonly selected RA-ILD's prevalence to be 2% (44.4%, p=0.015, adjusted residues 2.6). Physicians working for 15-30 and >30 years more commonly considered nintedanib ineffective in Ninf-RA-ILD (p=0.005, 26.3% and 33.3% vs 4.8% – adjusted residues 2.4 and 2.3). For inf-RA-ILD, those working for <15 years more commonly found rituximab effective (p=0.006, 90.5% vs 84.2% and 44.4%, adj residues 2.5).

Conclusion: There is some variability in disease assessment among physicians caring for RA-ILD, namely in judging risk factors, screening methodologies and medications. Greater education/multidisciplinarity might increase evidence-based knowledge and align strategies to minimize this condition's impact.

REFERENCES

1. Natalini JG et al. Understanding the determinants of health-re-

TO 221 – Tabela 1. Demographic of respondents

	Rheumatologists (n=81)	Pulmonologists (n=31)	Total (n=112)	P value[a]
Gender, male	21 (25.9%)	13 (41.9%)	34 (30.4%)	0.099
Age, median (IQR)	35 (12)	38 (18)	36 (13)	0.225
Professional status, specialist	59 (72.8%)	23 (74.2%)	82 (73.2%)	0.885
Years in practice				
0-5	28 (34.6%)	9 (29.0%)	37 (33.0%)	0.727
6-10	23 (28.4%)	7 (22.6%)	30 (26.8%)	
11-15	12 (14.8%)	5 (16.1%)	17 (15.2%)	
15-30	13 (16.0%)	6 (19.4%)	19 (17.0%)	
>30	5 (6.2%)	4 (12.9%)	9 (8.0%)	
Practice location				
Aveiro	6 (7.4%)	3 (9.7%)	9 (8.0%)	0.038
Braga	6 (7.4%)	2 (6.5%)	8 (7.1%)	
Bragança	1 (1.2%)	0	1 (0.9%)	
Castelo Branco	2 (2.5%)	1 (3.2%)	2 (2.7%)	
Coimbra	7 (8.6%)	2 (6.5%)	9 (8.0%)	
Faro	3 (3.7%)	3 (9.7%)	6 (5.4%)	
Guarda	0	3 (9.7%)*	3 (2.7%)	
Leiria	2 (2.5%)	0	2 (1.8%)	
Lisboa	27 (33.3%)	8 (25.8%)	35 (31.3%)	*adj res 2.8
Porto	5 (6.2%)	6 (19.4%)*	11 (9.8%)	
Açores	4 (4.9%)	0	4 (3.6%)	
Setúbal	8 (9.9%)	2 (6.5%)	10 (8.9%)	*adj res 2.1
Viana do Castelo	7 (8.6%)	0	7 (6.3%)	
Vila Real	0	1 (3.2%)	1 (0.9%)	
Viseu	3 (3.7%)	0	3 (2.7%)	
Hospital setting, tertiary	44 (54.3%)	12 (38.7%)	56 (50.0%)	0.139

aP-value compares rheumatologists to pulmonologists using Qui2/Fisher's exact testing as appropriate. IQR – interquartile range.

lated quality of life in rheumatoid arthritis-associated interstitial lung disease. Respir Med 2017;127:1–6
2. Solomon JJ et al. The attitudes and practices of physicians caring for patients with rheumatoid arthritis-associated interstitial lung disease: an international survey. Rheumatology (Oxford). 2022 Apr11;61(4):1459-1467

222 - LUPUS ERYTHEMATOSUS-LIKE SYNDROME INDUCED BY ANTI-TNF THERAPY IN RHEUMATIC DISEASES AND INFLAMMATORY BOWEL DISEASE: DESCRIPTION OF A FOLLOW-UP COHORT

Carlos Marques-Gomes[1, 2], Mariana Diz-Lopes [1, 2], Ana Martins[1, 2], Daniela Oliveira[1, 2, 3], Bruno Miguel Fernandes[1], Fernando Magro[3, 4, 5], Miguel Bernardes[1, 2], Lúcia Costa[1]
[1]Rheumatology Department, Centro Hospitalar de São João, Porto, Portugal, [2]Department of Medicine, Faculty of Medicine, University of Porto, Porto, Portugal, [3]Center for Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Porto, Portugal, Porto, Portugal, [4]Gastroenterology Department, Centro Hospitalar Universitário de São João, Porto, Portugal, [5]Pharmacology and Therapeutics Department, Centro Hospitalar Universitário de São João, Porto, Portugal

Background: Anti-tumor necrosis factor-α (anti-TNF) therapy is often part of the treatment strategy for rheumatic diseases (RD) and inflammatory bowel disease (IBD). An association between anti-TNF agents and the development of lupus erythematosus (LE)-like syndrome has been established and some patients may have a definitive diagnosis of systemic lupus erythematosus (SLE), requiring appropriate treatment. To date, no studies have been conducted to compare follow-up between rheumatic and IBD patients who have developed LE-like syndrome induced by anti-TNF agents.

Objectives: To describe and assess clinical and serological markers of LE-like syndrome in patients with RD and IBD receiving anti-TNF therapy. To characterize the development of SLE induced by anti-TNF therapy in patients with RD and IBD.

Methods: In this descriptive study with a minimum 3-month follow-up, we reviewed the clinical and serological parameters of 7 patients with RD and 9 patients with IBD, all diagnosed with LE-like syndrome induced by anti-TNF therapy. The study was conducted at Centro Hospitalar e Universitário de São João, in the Rheumatology Department.

Results: The mean age at the diagnosis of LE-like syndrome was 47,81 (±16,58) years (with most patients falling between 40 and 60 years old). Of the observed

TO 222 – Table 1. Relevant sociodemographic, clinical and laboratory data of all patients with lupus erythematosus (LE)like-syndrome (n=16).

	Age	Sex	Disease	Anti-Tnf (Duration of Therapy/ Months)	Ana At Diagnosis (Title)/ Pattern*	Anti-Dsdna At Diagnosis (Iu/L)	Anti-Histone At Diagnosis	Clinical Manifestation	Switch	Follow-Up (Duration/Months)**
RD	66	female	RA	Infliximab (16)	1/320 AC-1	247,9	Negative	Polyarthrits and constitutional symptoms	Adalimumab	Clinical manifestations resolution, Negative anti-dsDNA and normal complement at 17 months aMer discontinuation of anti-TNF (192)
	43	female	RA	Adalimumab (41)	1/320 AC-1	133,1	Positive	Skin rash	Golimumab	Clinical manifestations resolution, Negative anti-dsDNA and normal complement at 18 months aMer discontinuation of anti-TNF (132)
	53	male	AS	Infliximab (24)	1/640 AC-1	280	Negative	Polyarthritis, serosytis	Etanercept	Clinical manifestations resolution, Negative anti-dsDNA and normal complement at 11 months aMer discontinuation of anti-TNF (240)
	43	male	PsA	Etanercept (52)	1/320 AC-1	251,7	Positive	Polyarthritis, cytopenia (anemia) and constitutional symptoms	Tofactitinib	Clinical manifestations resolution; the patient remained Positive for ANA and antidsDNA (84)
	42	female	SpA	Adalimumab (10)	1/1000 AC-1	129,9	Positive	Polyarthritis	Secukinumab	Negative anti-dsDNA and normal complement at 4 months aMer discontinuation of anti-TNF (60)
	69	male	AS	Etanercept (48)	1/100 AC-2/4/5	105,5	Positive	Polyarthritis	Certolizumab	Clinical manifestations resolution, Negative anti-dsDNA and normal complement at 8 months aMer discontinuation of anti-TNF (53)
	55	female	SpA	Adaliumumab (7)	1/320	259,7	Negative	Polyarthritis; cytopenia (anemia and lynfopenia)	No switch	No clinical manifestations resolution (4)
IBD	44	female	CD	Infliximab (120)	1/100 AC-1	116,5	Negative	Polyarthritis and skin rash	No switch	Clinical manifestations resolution, Negative anti-dsDNA and normal complement aMer 48 months of MTX therapy (76)
	40	female	UC	Infliximab (9)	> 1/1000 AC-1	151,9	Negative	Polyarthritis	Vedolizumab	Clinical manifestations resolution, Negative anti-dsDNA and normal complement aMer 12 months of AZA therapy (62)
	51	female	CD	Infliximab (24)	> 1/1000 AC-1	164,8	Negative	Polyarthritis	No switch	Clinical manifestations resolution, Negative anti-dsDNA and normal complement aMer 24 months of HCQ therapy (45)
	20	female	CD	Infliximab (7)	1/320 AC-1	136	Negative	Polyarthritis	Adalimumab	Loss to follow-up
	59	female	CD	Infliximab (6)	> 1/1000 AC-1	192,7	Negative	Polyarthritis and constitutional symptoms	Vedolizumab	Clinical manifestations resolution, Negative anti-dsDNA and normal complement aMer 5 months of HCQ therapy (34)
	25	female	CD	Adalimumab (33)	1/1000 AC-2/4/5	Negative	Negative	Polyarthritis, hand cutaneuous vasculitis	No switch	Clinical manifestations resolution and Negative ANA at 5 months of treatment with low-dose prednisolone; C4 remained low (26)
	27	female	CD	Infliximab (23)	> 1/1000	Negative	Positive	Polyarthralgias	Ustekinumab	Symptoms control with low-dose prednisolone; dactilitis (PsA onset) (21)
	83	male	CD	Infliximab (24)	> 1/1000	Negative	Positive	Polyarthritis, serosytis	No switch	Clinical manifestations resolution at 6 months aMer discontinuation of anti-TNF and aMer 2 months of treatment with low-dose prednisolone (16)
	45	male	CD	Infliximab (144)	1/320 AC-2/4/5	360,1	Negative	Polyarthritis	Vedolizumab	AMer 5 months of HCQ therapy and anti-TNF stop, the patient remained Positive for anti-dsDNA and with low complement (6)

ANA: an'nuclear an'body; an'-dsDNA: an'-double-stranded DNA an'body; an'-TNF: an'-tumor necrosis factor-α therapy; AS: ankylosing spondyli's; AZA: azathioprine; CD: Crohn's disease; HCQ: hydroxychloroquine; IBD: inflammatory bowel diseases; MTX: methotrexate; PsA: psoria'c arthri's; RA: rheumatoid arthri's; RD: rheuma'c diseases; SpA: spondyloarthri's; UC: ulcera've coli's. *According to Report of the First Interna'onal Consensus on Standardized Nomenclature of An'nuclear An'body Hep-2 Cell PaVerns 2015. ** The an'-TNF was discon'nued at the 'me of LE-like syndrome diagnosis.

patients, 11 were female (68,75%). While 8 IBD patients received infliximab, only 2 patients from the RD group were treated with this anti-TNF agent. Almost all patients developed polyarthritis (n=14), 2 patients developed skin rash, 3 patients had constitutional symptoms (such as fatigue or fever), 2 patients had cytopenia (1 with anemia and 1 with anemia and leukopenia), 2 patients developed serositis (pleural effusion) and 1 patient had vasculitis-like skin lesions in the hands. At the time of diagnosis, all patients tested positive for antinuclear antibodies (ANA; n=16), with higher titers observed in the IBD group, and almost all patients (n=13) tested positive for anti-double-stranded DNA (anti-dsDNA). Except for 1 patient, positive anti-histone antibodies were accompanied by negative or low-positive anti-dsDNA antibodies. Complement levels were mostly normal.

Four IBD patients needed immunosuppressive therapy (methotrexate, azathioprine or hydroxychloroquine) to resolve their symptoms (in 3 patients, ANAs and anti-dsDNA were negative only after at least 1 year of treatment). These patients continued to receive immunosuppressants. In the other 12 cases, including all RD patients, discontinuation of anti-TNF therapy (and sometimes using glucocorticoids) was sufficient for symptoms resolution and antibody negativity. Five patients switched to another anti-TNF without experiencing relapse of symptoms. The relevant clinical and laboratory data are detailed in table 1.

Discussion: Studies show that LE-like syndrome frequency has been increasing and recognition of the condition is very important as anti-TNF therapy is commonly used. This study, although limited by its descriptive nature and small sample size, revealed differences between the RD and IBD groups regarding the specific anti-TNF agents used and the necessity of immunosuppressive therapy, more prevalent in the IBD group. On the other hand, almost all patients improved after discontinuing therapy and tolerated the switch to another anti-TNF-α agent without SLE recurrence.

Further studies are required to establish the impact of definitive SLE diagnosis secondary to anti-TNF therapy.

223 - THE ASSOCIATION OF CARDIOVASCULAR COMORBIDITIES OTHER THAN OBESITY ON KNEE OSTEOARTHRITIS' PAIN, FUNCTION AND OVERALL QUALITY OF LIFE – AN EPIREUMAPT COHORT ANALYSIS

Hugo Parente[1], Emanuel Costa[2], Sara Paiva Dinis [3], Catarina Soares[1], Maria Pontes Ferreira[1], Anita Cunha[1], Francisca Guimarães[1], Soraia Azevedo[1], Daniela Santos-Faria[1], Carmo Afonso[1], José Tavares-Costa[1], Filipa Teixeira[1], Carlos Vaz[4, 5]

[1]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [2]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [3]Serviço de Reumatologia, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal, [4]Serviço de Reumatologia, Centro Hospitalar de São João, Porto, Portugal, [5]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal

Background: Osteoarthritis (OA) has distinct phenotypes[1], one of them being metabolic, with growing evidence of OA's association with cardiovascular risk factors (CVRF)[2].

Objectives: Determine the association of CV comorbidities with knee OA (KOA), namely on the domains of pain, function and quality of life.

Methods: Cohort retrospective study among non-obese patients with KOA who participated in the national health survey EpiReumaPt. Sociodemographic and clinical data were collected, namely CVRF (Essential Arterial Hypertension- EAH, Dyslipidemia- DLP and Diabetes Mellitus- DM), Kellgren-Lawrence (KL) staging, and the Knee and Osteoarthritis Outcome Score (KOOS). The survey conducted 4 interviews (timed at 2012-13[t1], 2014-15[t2], 2015-16[t3], and 2021[t4]) with a serial collection of the EuroQol-5 Dimension (EQ5D), and the Health Assessment Questionnaire (HAQ) scores. Comparison of both cohorts, with and without CVRF, was performed.

Results: A total of 179 patients with knee OA were eligible. Table 1 depicts both groups. CVRF did not associate with KL staging, but it did with lower t2 and t3 EQ5D (p=0.041 and 0.011), and higher t3 HAQ scores (p=0.031). EAH among KOA patients was associated with lower (t1-4) EQ5D ($\bar{x}$=0.519 vs $\bar{x}$=0.635, p=0.003; $\bar{x}$=0.335 vs $\bar{x}$=0.542, p<0.001; $\bar{x}$=0.494 vs $\bar{x}$=0.650, p=0.006; Md =0.379 vs Md=0.693, p=0.036), and higher (t1-3) HAQ scores (Md=0.875 vs Md=0.625, p=0.011; $\bar{x}$=1.280 vs $\bar{x}$=0.859, p<0.001; $\bar{x}$=1.105 vs $\bar{x}$=0.724, p=0.004), and, according to KOOS, to more symptoms ($\bar{x}$=56.07 vs $\bar{x}$=65.51, p=0.004), pain ($\bar{x}$=56.31 vs $\bar{x}$=63.96, p=0.017), less functionality for sports and recreation (Md=15.00 vs Md=25.00, p=0.008) and less overall quality of life ($\bar{x}$=40.71 vs $\bar{x}$=47.94, p=0.033). It also associated with a greater decrease in EQ5D score from t1 to t2 ($\bar{x}$=-0.200 vs $\bar{x}$=-0.104, p=0.031). However, hypertensive patients were older ($\bar{x}$=71.30 vs $\bar{x}$=66.64, p=0.002), more likely to be women (p=0.008, adjusted residues=2.6), and to have a total of 2 (p<0.001, adjusted residues=6.9) and 3 (p<0.001, adjusted residues=3.2) CVRF. DM among KOA patients associated with lower (t1) EQ5D ($\bar{x}$=0.469 vs $\bar{x}$=0.598, p=0.010) and higher (t1) HAQ scores (Md=1.188 vs Md=0.750, p=0.007), more

TO 223 –Table 1 – Demographics of the participants

	Total (n=179)	With CV risk factors (n=144)	Without CV risk factors (n=35)	p-value
Age at diagnosis (years), Mean (SD)	69.45 (10.00)	70.05 (9.43)	67.00 (11.93)	NS
Gender, female, % (n/N)	79.9 (143/179)	82.6 (119/144)	68.6 (24/35)	NS
CV risk factors				
- Essential Arterial Hypertension, % (n/N)	58.2 (103/177)	72.0 (103/143)	-	-
- Dyslipidemia, % (n/N)	59.0 (102/177)	73.4 (102/139)	-	
- Diabetes Mellitus, % (n/N)	19.2 (34/177)	23.6 (34/142)	-	
Number of CV risk factors				
- 0, % (n/N)	19.6 (35/179)	-	100 (35/35)	-
- 1, % (n/N)	34.1 (61/179)	42.4 (61/144)	-	
- 2, % (n/N)	39.1 (70/179)	48.6 (70/144)	-	
- 3, % (n/N)	7.3 (13/179)	9.0 (13/144)	-	
EQ5D				
- T1*, Mean (SD)	0.571 (0.261)	0.554 (0.266)	0.636 (0.232)	NS
- T2*, Mean (SD)	0.423 (0.265)	0.401 (0.274)	0.513 (0.201)	0.041
- T3*, Mean (SD)	0.564 (0.299)	0.527 (0.303)	0.703 (0.243)	0.011
- T4*, Mean (SD)	0.588 (0.314)	0.559 (0.303)	0.699 (0.345)	NS
HAQ				
- T1*, Mean (SD)	0.888 (0.689)	0.938 (0.703)	0.682 (0.592)	NS
- T2*, Mean (SD)	1.109 (0.732)	1.158 (0.716)	0.903 (0.775)	NS
- T3*, Mean (SD)	0.937 (0.694)	1.008 (0.715)	0.650 (0.520)	0.031
- T4*, Mean (SD)	1.017 (0.779)	1.109 (0.791)	0.656 (0.633)	NS
KOOS				
- Symptoms, Mean (SD)	60.21 (21.38)	58.64 (20.85)	66.56 (22.61)	NS
- Pain, Mean (SD)	59.66 (20.59)	58.18 (21.09)	65.56 (17.54)	NS
- Function in daily living, Mean (SD)	60.49 (21.71)	59.47 (21.94)	64.61 (20.58)	NS
- Function in sport and recreation, Mean (SD)	24.41 (25.02)	22.90 (24.26)	24.41 (25.02)	NS
- Quality of life, Mean (SD)	43.72 (21.70)	43.11 (21.75)	30.61 (27.49)	NS
Radiographic scoring through KL classification system				
- 1-2, % (n/N)	37.2 (29/78)	34.8 (23/66)	50.0 (6/12)	NS
- 3-4, % (n/N)	62.8 (49/78)	65.2 (43/66)	50.0 (6/12)	NS

* T1 to T4 relates to the timings of the 4 conducted interviews, for which the total number of participants declined, as follows: T1=179, T2=149, T3=110, T4=63
CV: cardiovascular; EQ5D: EuroQol- 5 Dimension; HAQ: Health Assessment Questionnaire; KL: Kellgren Lawrence; KOOS: Knee and Osteoarthritis Outcome Score; NS: non-significant; SD: standard deviation.

KOOS-reported pain ($\bar{x}$=52.94 vs $\bar{x}$=61.32, p=0.034) and less functionality in daily activities ($\bar{x}$=52.33 vs $\bar{x}$=62.45, p=0.015). Yet, these patients were older ($\bar{x}$=73.50 vs $\bar{x}$=68.40, p=0.007) and more likely to have 3 CVRF (p<0.001, adjusted residues=7.7). DLP among KOA patients associated with higher t3-4 HAQ scores (Md=1.000 vs Md=0.630, p=0.040; $\bar{x}$=1.500 vs $\bar{x}$=0.500, p=0.007). These patients were also more likely to have 2 (p<0.001, adjusted residues=6.4) or 3 (p<0.001, adjusted residues=3.1) CVRF.

Conclusion: CVRF seem to contribute to patient-reported (PR) pain, dysfunctionality and lower quality of life in KOA. EAH, among the conventional CVRF, may be the greatest contributor to this effect. The number of CVRF did not worsen these PR outcomes, and, in this sample, CVRF did not associate with higher KL staging. This study highlights the importance of acting on preventive measures for CVRF, in overall OA management.

REFERENCES

1. Deveza et al. Clinical and experimental rheumatology vol. 37 Suppl 120,5 (2019): 64-72.
2. Orcha et al. Revue du Rhumatisme vol. 87 Suppl 1 (2020): A244.

224 - FATORES PREDITORES DE SWITCH AO PRIMEIRO FÁRMACO BIOTECNOLÓGICO EM DOENTES COM ARTRITE PSORIÁTICA

Carla Campinho Ferreira[1], Paulo Pereira[1], Ana Margarida Correia[1], Emanuel Costa[1], Diogo Esperança Almeida[1], Joana Sousa-Neves[1], Marcos Cerqueira[1], José Redondo[1], Ana Ribeiro[1], Joana Leite Silva[1]

[1]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal

Introdução: A Artrite Psoriática (APso) é uma doença inflamatória crónica cujo tratamento de 1ª linha são

TO 224 – Tabela 1. Distribuição das variáveis demográficas, clínicas e laboratoriais dos dois grupos em estudo.

Características	Non-switchers	Switchers	p-value	OR [IC]
Sexo (N=83), n sexo feminino/N (%)	25/55 (45.5)	15/28 (53.6)	.485	-
Profissão (N=69), n trabalho pesado/N (%)	18/47 (38.3)	11/22 (54.5)	.207	-
Comorbilidades	3/23 (13.0)	1/10 (10.0)	.806	-
Tabagismo (N=33), n/N (%)	10/55 (18.2)	7/28 (25.0)	.468	
HTA (N=83), n/N (%)	13/55 (23.6)	5/28 (17.9)	.547	
Dislipidemia (N=83), n/N (%)	5/55 (9.1)	1/28 (3.6)	.376	
DM tipo 2 (N=83), n/N (%)	2/21 (9.5)	0/18 (0)	.999	
Hiperuricemia (N=39), n/N (%)				
Idade ao diagnóstico da APso (N=81), $\bar{x}$ ± s anos	41.0 ± 10.61 (n=54)	45.1 ± 9.47 (n=27)	.101	-
Tempo de evolução dos sintomas até diagnóstico (N=68), $\bar{x}$ ± s anos	46.0 ± 80.5 (n=44)	58.6 ± 65.32 (n=24)	.514	-
Subtipo de APso				-
AR-like (N=83), n/N (%)	23/55 (41.8)	17/28 (60.7)	.106	
Oligoarticular (N=83), n/N (%)	16/55 (29.1)	7/28 (25.0)	.694	
Distal (N=83), n/N (%)	7/55 (12.7)	0/28 (0)	.999	
Axial (N=83), n/N (%)	10/55 (18.2)	4/28 (14.3)	.655	
Antecedentes				-
Dactilite (N=83), n/N (%)	10/55 (18.2)	7/28 (25.0)	.468	
Entesite (N=83), n/N (%)	5/55 (9.1)	2/28 (7.1)	.763	
Uveíte (N=83), n/N (%)	0/54 (0)	2/28 (7.1)	.999	
Psoríase cutânea (N=80), n/N (%)	41/53 (77.4)	18/27 (66.7)	.307	
Psoríase ungueal (N=80), n/N (%)	7/53 (13.2)	2/27 (7.4)	.444	
HLA-B27 (N=40), n/N (%)	7/27 (26.0)	3/13 (23.1)	.857	-
Ao diagnóstico da APso				-
VS (N=42), $\bar{x}$ ± s mm/h	21.1 ± 18.49 (n=30)	37.42 ± 30.32 (n=12)	.055	
PCR (N=44), $\bar{x}$ ± s	17.5 ± 21.52 (n=32)	21.1 ± 21.86 (n=12)	.618	
mg/L AD (N=39), $\bar{x}$ ± s	3.0 ± 2.86 (n=29)	4.4 ± 2.71 (n=10)	.198	
AT (N=40), $\bar{x}$ ± s	2.4 ± 2.32 (n=30)	3.9 ± 2.69 (n=10)	.112	
Dactilites (N=60), $\bar{x}$ ± s	0.3 ± 1.04 (n=41)	0.5 ± 1.43 (n=19)	.683	
Entesites (N=64), $\bar{x}$ ± s)	0.1 ± 0.29 (n=44)	0.1 ± 0.22 (n=20)	.577	
Erosões (n=54), n/N (%)	10/35 (28,6)	3/19 (15,8)	.301	
csDMARDs prévios ao início de bDMARD (N=81), $\bar{x}$ ± s	1.55 ± 0.789 (n=55)	1.58 ± 0.643 (n=26)	.858	-
Idade no início de bDMARD (N=81), $\bar{x}$ ± s	45.9 ± 10.20 (n=54)	48.3 ± 8.88 (n=27)	.293	-
Tempo de evolução até início de bDMARD (N=80), $\bar{x}$ ± s	60.1 ± 60.80 (n=54)	39.27 ± 39.79 (n=26)	.125	-
bDMARD				
ADA (n=49), n/N (%)	37/55 (67.3)	12/28 (42.9)	.035	.365 [.143-.931]
ETN (n=19), n/N (%)	11/55 (20.0)	8/28 (28.6)	.382	-
IFX (n=6), n/N (%)	2/55 (3.6)	4/28 (14.3)	.099	-
GOL (n=5), n/N (%)	3/55 (5.5)	2/28 (7.1)	.000	-
SCK (n=3), n/N (%)	1/55 (1.8)	2/28 (7.1)	.254	-
CTZ (n=1), n/N (%)	1/55 (1.8)	0/28 (0)	.761	-
No início de bDMARD	4/53 (7.5)	3/26 (11.5)	.560	-
Nº csDMARDs (N=79)	35/53 (66.0)	18/26 (69.2)	.777	-
0 csDMARDs, n/N (%)	14/53 (26.4)	5/26 (19.2)	.484	-
1 csDMARD, n/N (%)	17.5 ± 17,15	33.1 ± 19.71	.011	1.043 [1.010-1.077]
2 csDMARDs, n/N (%) VS (N=56), $\bar{x}$ ± s mm/h	9.2 ± 7.21 (n=42)	21.9 ± 23,83 (n=16)	.033	1.078 [1.006-1.154]
PCR (N=58), $\bar{x}$ ± s mg/L AD (N=56), $\bar{x}$ ± s	3.6 ± 3.95 (n=39)	5.3 ± 5,47 (n=17)	.176	-
AT (N=56), $\bar{x}$ ± s	3.1 ± 3.3 (n=39)	4,9 ± 5,50 (n=17)	.150	-
Dactilites (N=58), $\bar{x}$ ± s	0.5 ± 1.19 (n=42)	0,3 ± 0,78 (n=16)	.409	-
Entesites (N=58), $\bar{x}$ ± s	0.3 ± 0.643 (n=42)	0,2 ± 0,75 (n=16)	.537	-

HTA – Hipertensão arterial; DM – Diabetes mellitus; APso – Artrite Psoriática; AR – Artrite reumatoide, VS – Velocidade de sedimentação; PCR – Proteina C reativa; AD – Articulações dolorosas; AT – Articulações tumefactas; csDMARDs – fármacos modificadores de doença reumática sintéticos convencionais; bDMARD - fármacos modificadores de doença reumática biológicos; ADA – Adalimumab; ETN – Eternecept; IFX – Infliximab; GOL – Golimumab; SCK – Secucinumab; CTZ – Certolizumab, $\bar{x}$ - média; s – desvio padrão

os anti-inflamatórios e os fármacos modificadores de doença reumática sintéticos convencionais (csDMARDs). Nos doentes em que a remissão ou a baixa atividade de doença não é atingida, são utilizados os DMARDs biotecnológicos (bDMARDs). No entanto, é comum haver doentes refratários, com necessidade de efetuar switch para outro bDMARD. Uma vez que a probabilidade de sucesso terapêutico decresce à medida que se avança nas linhas terapêuticas, é importante reconhecer que fatores tornam um doente mais suscetível a switch, com o objetivo de melhor otimização terapêutica.

Objetivos: Avaliar fatores preditores de switch de bDMARD, comparando as características dos doentes que se mantiveram no primeiro bDMARD com aqueles em que foi necessário alterar o fármaco.

Métodos: Estudo retrospetivo, unicêntrico, com inclusão de doentes com mais de 18 anos com diagnóstico de APso, seguidos em consulta de Reumatologia, que tenham efetuado terapêutica com bDMARD. Foram colhidos dados demográficos, clínicos e laboratoriais relativos ao diagnóstico, à data de início do bDMARD, duração do tratamento até switch e motivos de descontinuação. As variáveis foram comparadas entre o grupo de doentes que efetuou switch de bDMARD ("switchers") com o grupo de doentes que manteve o primeiro bDMARD ("non-switchers"). A análise estatística foi efetuada com recurso ao SPSS, utilizando as regressões logísticas uni e multivariada para identificar quais os fatores preditores de switch do primeiro bDMARD. Foi considerado estatisticamente significativo um valor de $p<.05$.

Resultados: Foram incluídos um total de 83 doentes, sendo 40 (48.2%) do sexo feminino, com idade média de 53.2 ± 11.80 anos. A análise descritiva das variáveis avaliadas neste estudo encontra-se representada na tabela 1. Os anti-TNFα foram a classe de bDMARD mais usada, sendo que a maioria (n=49; 59.0%) efetuou Adalimumab (ADA) como primeiro fármaco. Em 28 doentes foi observado switch de bDMARD, sendo o tempo médio até descontinuação de 37 ± 34.9 meses. Os motivos para switch foram: falência primária (n=7); falência secundária (n=14), evento adverso (n=7). Comparando os dois grupos, os doentes do grupo switchers tinham em média VS e PCR à data de início de bDMARD mais elevadas (p.011; OR 1.043; IC95% [1.010-1.077] e p=.033; OR 1.078; IC95% [1.006-1.154], respetivamente). Por outro lado, quando o primeiro bDMARD utilizado foi o ADA, verificou-se uma menor frequência de switch quando comparado com os restantes bDMARD utilizados (p=.035; OR .365; IC95% [.143- .931]). Na análise multivariada, a VS e a PCR à data de início de bDMARD, de forma isolada, foram identificadas como fatores preditores de switch (p=.023; OR 1.041; IC95% [1.005-1.078] e p=.024; OR 1.092; IC95% [1.012-1,179], respetivamente) independentes do sexo, sub-tipo (AR-like vs outros) e tempo de evolução até início de bDMARD

Conclusão: A elevação dos parâmetros inflamatórios à data de início do primeiro bDMARD constituiu um fator preditor de switch na nossa população. Na análise univariada, parece ainda existir tendência para um aumento de probabilidade de switch nos doentes com trabalho pesado, idade jovem ao diagnóstico, subtipo AR-like e menor tempo de evolução até início de bDMARD.

228 – CURE BY RADIATION: A RETROSPECTIVE CASE SERIES ASSESSING EFFICACY AND SAFETY OF RADIOSSINOVECTOMY

André Saraiva[1], Ana Isabel Maduro[1], João Rebelo[2], Inês Lopo[2], Margarida Coutinho[1, 3], Sara Serra[1], Gracinda Costa[2, 3]

[1]Serviço Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Serviço de Medicina Nuclear, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal, [3]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal

Introduction: Radiosynovectomy (RS) is a minimally invasive procedure employed in the treatment of inflammatory joint diseases and intra-articular tumours. (1) By injecting a radiopharmaceutical into the affected joint (Yttrium-90), it selectively targets and irradiates the synovial tissue, aiming to reduce inflammation and alleviate symptoms. Adequate documentation of the outcome of treatment with this technique is sparse. Available literature shows relatively short follow up times and often combine different locations and pathologies with inconsistent results.

This is a procedure that is performed in a relatively small number of centres worldwide. Our goal was to evaluate the clinical outcomes and safety of radiosynovectomy in a small cohort of patients that underwent the procedure in our department.

Methods: Retrospectively, the clinical records of patients who underwent this procedure between 2016 and 2023, were revised and clinical features, treatment, recurrences, and pain were evaluated. All the patients were treated at our clinic with collaboration from the nuclear medicine department.

Results: In total, 4 patients were included (age 36 ± 4.97 years old; female 75%; duration of symptoms 22.8 ± 18.3 months.) None of the patients had a previous inflammatory rheumatic disease diagnosis, three had villonodular pigmented synovitis (SVNP), confirmed ei-

ther by biopsy or MRI, and one had a chronic unspecific synovitis (biopsy). The knee was the affected joint in all the patients and most had prior treatment elsewhere (75%), namely surgical synovectomy (arthroscopic), with recurrence in all patients. One of the patients with SNVP underwent 5 surgical synovectomies, in total. All of them presented to our clinic with pain and swelling of one knee for more than 3 months. After being approved for the procedure, Yttrium was injected in the joint with either ultrasound or fluoroscopic guidance and bone scintigram was performed both prior to the injection as well as post procedure to ensure adequate placement in the joint space. The mean follow-up was 38.3±24.9 months and only the patient with the chronic unspecific synovitis had a recurrence after 6 months; all others were asymptomatic or had big improvements in pain and function. No major side effects were reported, however mild to moderate pain after the procedure was common.

Conclusion: Surgical treatment is the GOLD STANDARD for treatment of SVNP, however recurrence is high (20-40%) (1). RS remains as a treatment option as most patients achieved remission with no major side effects, highlighting the potential efficacy and favourable safety profile of RS, mainly in SVNP. The only patient with a recurrence ended up achieving remission after being started on methotrexate, which might advocate for an alternative rheumatic inflammatory disease diagnosis.

230 - DIFFICULT-TO-TREAT JUVENILE IDIOPATHIC ARTHRITIS: INSIGHTS INTO A CHALLENGING PATIENT POPULATION

Vanessa Fraga[1], Catarina Abreu[1], Sandra Sousa[1], Maria José Santos[1, 2]

[1]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Juvenile Idiopathic Arthritis (JIA) is a heterogeneous group of chronic inflammatory joint diseases with onset before the age of 16 years. While many patients achieve disease control with standard treatment, a subset of patients experience persistent disease activity or inadequate response. Understanding the characteristics and treatment outcomes of this difficult-to-treat JIA (D2T-JIA) population is crucial for developing tailored therapeutic approaches and improving patient outcomes.

Objective: Characterize the D2T-JIA population and compare with the non-D2T-JIA.

Methods: We performed a retrospective study of JIA patients followed in our pediatric rheumatology outpatient clinic registered in Reuma.pt. D2T-JIA was defined according to the European Alliance of Associations for Rheumatology criteria for D2T Rheumatoid Arthritis. Demographic characteristics, clinical manifestations, disease activity, laboratory findings and treatment options are reported and compared in both groups.

Results: A total of 91 JIA patients were included, 58.2% females and mostly Caucasians (82%). The mean age at disease onset was 7.5 ± 4.8 years and the mean age at diagnosis was 8.9 ± 6.6 years. The most common JIA subtypes were persistent oligoarticular (n=23) and extended oligoarticular arthritis (n=16) .

The D2T-JIA group represented 4.4% (n=4) of all JIA patients, with a 1:1 male-to-female ratio. The median age at disease onset and diagnosis were 11 ± 7.4 years and 11.5 ± 7.8 years, respectively. Each patient belonged to a different JIA category: extended oligoarthritis, persistent oligoarthritis, systemic, and rheumatoid factor (RF) positive polyarthritis.

Comparison between D2T-JIA (n=4) and the non-D2T-JIA group (n=87) revealed no statistically significant differences in demographic characteristics, extra-articular manifestations or immunology markers (table 1). Nevertheless, Juvenile Arthritis Disease Activity Score at first bDMARD's baseline was higher in D2T-JIA (21.6 vs 12, p=0.695).

Regarding treatment, while both groups exhibited similar use of nonsteroidal anti-inflammatory drugs and conventional disease-modifying antirheumatic drugs (DMARD), the D2T-JIA group was more likely to require the use systemic glucocorticoids (p=0.020) and a greater number of bDMARD, with all D2T-JIA patients receiving at least 3 bDMARDs. In contrast, only 40.2% of non-D2T patients required bDMARDs. The mean disease duration until first bDMARD was inferior in the D2T-JIA group (1.6 ± 14.4 vs 10 years, p=0.265). The first bDMARD most commonly used in D2T-JIA was anti-tumor necrosis factor (anti-TNF), 75% Etanercept and 25% Adalimumab. All D2T-JIA patients needed a switch due to secondary treatment failure, with a mean drug persistence of 36.3±39.3 months. In 75% of cases, an alternative mechanism of action, primarily anti-IL6 therapy (n=2), was chosen as the first switch.

Discussion: The comparison between the D2T-JIA and non-D2T-JIA groups did not reveal statistically significant differences in demographic or disease characteristics. However, patients with D2T-JIA were more likely to need systemic glucocorticoids, a greater number of bDMARDs and earlier in the disease course, emphasizing the more aggressive treatment approach required for this challenging population. Further research using a larger sample is needed to elucidate who will more be likely to develop D2T disease and the underlying mechanisms contributing to treatment resistance.

TO 230 – Table 1. Demographics, clinical and laboratory characteristics, disease activity and treatment in D2T-JIA and non-D2T-JIA

	D2T-JIA (n=4)	Non-D2T-JIA (n=87)	p-value
Demographics			
Sex (female), n (%)	2 (50)	51 (58.6)	0.557
Race (caucasian), n (%)	3 (75)	72	0.400
Age at disease onset, median ± IQR, years	11.02 (7.4)	7.26 (9.5)	0.255
Age at diagnosis, median ± IQR, years	11.51 (7.8)	7.963 (9.5)	0.444
Disease duration until 1st bDMARD mean ± SD, years	1.62 (14.4)	10.03	0.265
JIA category, n (%)			
Persistent oligoarthritis	1 (25)	22 (25.3)	-
Extended oligoarthritis	1(25)	15 (17.2)	-
Systemic arthritis	1(25)	8 (0.09)	-
RF-positive polyarthritis	1(25)	5 (5.7)	-
RF-negative polyarthritis	0 (0)	11 (12.6)	-
Enthesitis related arthritis	0 (0)	9 (10.3)	-
Psoriatic JIA	0 (0)	12 (13.8)	-
Undifferenciated	0 (0)	5 (5.7)	-
Immunology, n (%)			
ANA	3 (75)	65 (74.8)	0.572
RF	1 (25)	11 (12.6)	0.490
HLA-B27	0 (0)	3 (3.34)	-
Extra-articular manifestations (yes), n (%)	2 (50)	37 (42.5)	0.768
Fever	1 (25)	16 (18.4)	0.569
Rash	1 (25)	8 (9,2)	0.346
Uveitis	1 (25)	7 (8)	0.312
Enthesitis	0 (0)	9 (10.3)	-
Dactylitis	0 (0)	5 (5.7)	-
Psoriasis	0 (0)	7 (8)	-
Inflammatory back pain	0 (0)	3 (3.4)	-
Disease activity measures*, median (IQR)			
JADAS-27, points	21.6 (0)	12 (8.9)	0.695
Tender joint count	4 (7)	4 (8)	0.624
Swollen joint count	1 (7)	4 (8)	0.218
ESR mm/hr	30 (0)	24.5 (23,25)	0.545
CRP mg/dl	0.90 (0)	0.63 (2.6)	0.635
Treatment, n (%)			
NSAID	4 (100)	81 (93.1)	1.000
cDMARD	4 (100)	71 (81.6)	
Intra-articular GC injections	2 (50)	25 (28.7)	0.579
Systemic GC	4 (100)	31 (35.6)	0.020
bDMARD	4 (100)	35 (40.2)	0.031
Number of bDMARD, n (%)			-
0	0 (0)	52 (59.7)	
1-2	0 (0)	34 (39.1)	
3	4 (100)	1 (1.1)	
1st bDMARD's MoA, n (%)			
Anti-TNF	4 (100)	32 (82)	
Anti- IL1	0 (0)	2 (5.1)	
Anti- IL6	0 (0)	1 (2.6)	
First switch to the same MoA, n (%)	1 (25)	12 (70.6)	0.253
First bDMARD persistence (months)	36.3 (39.3)	48.3 (45.2)	0.616

*Disease activity at the baseline of the first bDMARD. ANA: Antinuclear antibody; bDMARD: Biologic disease-modifying antirheumatic drug; cDMARD: Conventional disease-modifying antirheumatic drug; GC: Glucocorticoids; IQR: Interquartile range; MoA: Mechanism of action; NSAID: Non-steroidal anti-inflammatory drugs; RF: Rheumatoid factor.

231 - DOENÇA INTERSTICIAL PULMONAR EM DOENTES COM ESCLEROSE SISTÉMICA E ANTICORPOS ANTI-U1RNP: UMA ATUALIZAÇÃO DA ANÁLISE DA COORTE EUROPEAN SCLERODERMA TRIALS AND RESEARCH (EUSTAR)

Gonçalo Boleto[1], Corrado Campochiaro[2], Jérôme Avouac[2], Yannick Allanore[2]
[1]Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal, [2]Department of Rheumatology, Cochin Hospital, Paris, France

Introdução: A doença intersticial pulmonar (DIP) é principal causa de mortalidade em doentes com esclerose sistémica (ES). A presença de anticorpos anti-U1RNP está principalmente associada à doença mista do tecido conjuntivo, no entanto, estes autoanticorpos podem ser encontrados em até 10% dos doentes com ES. Um recente estudo multicêntrico mostrou que a presença de anticorpos anti-U1RNP estava independentemente associada a doença mais grave em doentes com ES. No entanto, até agora, no contexto de ES-DIP, pouco se sabe sobre o impacto dos anticorpos anti-U1RNP especificamente na gravidade da doença pulmonar.
Objetivo: Descrever as características clínicas, resultados e prognóstico de pacientes com ES-DIP e anticorpos anti-U1RNP.
Métodos: Doentes com ES com dados disponíveis sobre o perfil de autoanticorpos foram identificados na coorte EUSTAR. Aqueles que preencheram os critérios de classificação da American College of Rheumatology (ACR) de 1980 e/ou os critérios de classificação da ACR/ European League Against Rheumatism (ACR/ EULAR) de 2013 e com DIP identificada por tomografia computadorizada de alta resolução (TCAR) foram incluídos na análise. As características demográficas e clínicas basais foram comparadas entre os doentes positivos e negativos para os anticorpos anti-U1RNP. Para a análise longitudinal dos doentes com provas de função respiratória, a diminuição média anual da capacidade vital forçada em % (%CVF) foi comparada entre os dois grupos. Os preditores associados a morte por qualquer causa ou progressão grave, definida como uma diminuiçao >10% na %CVF/ano, foram avaliados em pacientes com ES-DPI com ou sem anticorpos anti-U1RNP nos 3 primeiros anos de evolução. A progressão leve foi definida como uma perda de 5-10% na %CVF/ano.
Resultados: Um total de 6043 doentes com ES-DIP foi incluído na análise, dos quais 217 (3,6%) eram positivos para anticorpos anti-U1RNP. A idade média era de 56,8 ± 13,2 anos e 4971 (82,3%) eram mulheres.
Os doentes com ES-DIP e anticorpos anti-U1RNP positivos tinham doença cutânea limitada com mais frequência (66,5% vs. 53,3%, p<0,001). Não se observaram diferenças estatisticamente significativas relativamente à frequência de envolvimento articular, muscular, renal ou digestivo.
Os pacientes com anticorpos anti-U1RNP positivos tinham um valor basal médio mais baixo de %CVF (83,5% vs. 86,0%, p<0,001) e um valor basal médio mais baixo de capacidade de difusão do monóxido de carbono prevista em % (%DLCO) (57,3% vs. 60,5%, p=0,003) do que os doentes com ES-DIP e anticorpos anti-U1RNP negativos. Não foram observadas diferenças significativas na TCAR, incluindo o grau de envolvimento pulmonar, a presença de opacidades em vidro fosco, favos de mel, alterações reticulares ou trações.
Durante os 3 primeiros anos de evolução, a diminuição anual da %CVF foi semelhante entre os pacientes positivos e negativos para os anticorpos anti-U1RNP (-0,33% vs. -0,72%, p=0,77). A progressão grave não foi estatisticamente diferente entre os dois grupos (6,9% vs. 13,0%, p=0,40). A progressão moderada também foi semelhante entre os dois grupos (18,5% vs. 17,1%, p=0,76). Contudo, observaram-se estatisticamente menos mortes no grupo de doentes anti-U1RNP positivos (6,9% vs. 13,0%, p=0,007).
Conclusão: Os resultados da análise da coorte EUSTAR mostram que apesar dos doentes com ES-DIP e anticorpos anti-U1RNP positivos terem uma função respiratória basal mais alterada com %CVF e %DLCO mais baixas, a taxa de progressão pulmonar foi semelhante entre os dois grupos com risco de mortalidade não aumentada durante o seguimento.

232 - USO DA VITAMINA K NA POPULAÇÃO OSTEOPORÓTICA PARA PREVENÇÃO DE FRATURAS - QUAL A EVIDÊNCIA?

Benedita Martins[1], Lígia Coutinho[1], Ana João Silva[1]
[1]ACES Grande Porto IV, Maia, Portugal

Introdução e Objetivo(s): A osteoporose é um dos maiores problemas de saúde pública mundiais afetando severamente as mulheres pós-menopausa e indivíduos idosos de ambos os sexos. O impacto clínico da osteoporose assenta na incapacidade provocada pelas mais diversas fraturas associadas a esta doença. A nível mundial ocorre uma fratura osteoporótica a cada 3 segundos. Embora haja diversos fármacos que provaram ser eficazes na prevenção e tratamento da osteoporose, o tratamento ideal para a osteoporose ainda não está estabelecido.
A vitamina K foi identificada inicialmente como um fator essencial para a cascata de coagulação, no entanto, tem sido comprovado a sua importância em diversas

outras áreas. Esta mostrou ser eficaz na osteoporose pela ativação de duas proteínas intracelulares que impedem a calcificação vascular e promovem a mineralização óssea, mas também através da inibição de células apoptóticas de forma a preservar o número de osteoblastos.
O objetivo deste trabalho é rever a eficácia da administração de vitamina K2 associado ou não a outras medidas terapêuticas na prevenção de fraturas na população osteoporótica.
Metodologia: Pesquisa de revisões sistemáticas, meta-análises (MA), ensaios clínicos randomizados (ECR) e normas de orientação clínica, publicados nos últimos 20 anos, em português ou inglês, realizada nas bases PubMed, National Institute for Health and Care Excellence, Canadian Medical Association, Cochrane Library, BMJ Evidence-Based Medicine, Database of Abstracts of Reviews of Effects, Bandolier e National Guideline Clearinghouse, utilizando os termos MeSH "Vitamin K2", "Osteoporosis" e "Fractures". Os critérios de inclusão foram definidos segundo o modelo PICO: P - População adulta com osteoporose; I - Uso da vitamina K2 associada ou não a outros fármacos e/ou suplementos; C - placebo ou quaisquer outras medidas terapêuticas ou preventivas que não a vitamina K2; O -incidência de fraturas. Para avaliação da qualidade dos estudos e atribuição da força de recomendação, foi utilizada a escala Strength of Recommendation Taxonomy (SORT) da American Family Physician.
Resultados: Dos 16 artigos obtidos na pesquisa, 1 foi excluído pelo idioma, 1 foi excluído após leitura do resumo, 4 após leitura integral e 8 excluídos por repetição, tendo sido selecionados 2 artigos (2 MA). No geral, constatou-se uma diminuição da incidência de fraturas nos grupos sob vitamina K2 quando comparado com os grupos controlo.
Discussão: Os estudos incluídos apresentam alguma heterogeneidade a nível das terapêuticas instituídas quer nos grupos de intervenção quer nos grupos controlo, dificultando a análise do impacto da vitamina K2 na prevenção das fraturas nos doentes osteoporóticos. Contudo, foi possível concluir que a vitamina k2 pode ter um impacto positivo na densidade mineral óssea e na prevenção de fraturas osteoporóticas.

233 - MACROPHAGE ACTIVATION SYNDROME – A CASE SERIES REPORT FROM A TERTIARY CENTRE

Fernando Albuquerque[1], Marcelo Neto[1], Ana Isabel Maduro[1], André Saraiva[1], Ana Rita Prata[2], Tânia Santiago[1, 3], Maria João Salvador[1]
[1]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal, [2]Rheumatology Department, Centro Hospitalar do Baixo Vouga, EPE, Aveiro, Aveiro, Portugal, [3]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal

Introduction: Macrophage Activation Syndrome (MAS), a form of secondary hemophagocytic lymphohistiocytosis (sHLH), is a severe and life-threatening complication of rheumatic diseases. Prompt diagnosis is essential to optimize the prognosis. However, the clinical presentation is commonly similar to a flare of a rheumatic disease, so it is essential to have a high grade of suspicion.
Objective: To characterize clinical manifestations, laboratory findings, treatment and outcome of patients with rheumatic diseases who developed MAS in our Rheumatology tertiary centre.
Methods: Retrospective descriptive analysis of MAS cases who were admitted to the Rheumatology ward of Centro Hospitalar e Universitário de Coimbra between January 2007 and May 2023. The patients included fulfilled the HLH-2004 criteria or presented at least 169 points in HScore.
Results: Five patients with MAS were included. Of these, four (80%) patients were female with a mean age of 33,8 years, (23 to 52 years) – table 1. Three patients had Systemic Lupus Erythematosus and two had Still Disease.
At baseline all patients (n=5; 100%) had fever. Other manifestations were serositis (n=4; 80%), encephalopathy (n=4; 80%), diarrhoea (n=4; 80%), splenomegaly (n=4; 80%), hepatomegaly (n=3; 60%) and adenomegaly (n=2; 40%).
Two patients had a rash (related to Still Disease) and none had arthritis.
All patients (n=5; 100%) had anaemia (mean 9,0 ± 0,7 g/dL), elevated lactate dehydrogenase (mean 1134,0 ± 878,0 U/L), elevated C Reactive Protein (mean 8,7 ± 8,0 mg/dL) and elevated ferritin (mean 12103,2 ± 8104,8 ng/mL).
Other findings were leukopenia (n=4; 80%), thrombocytopenia (n=4; 80%), elevated aspartate and alanine transaminases (n=4; 80%), elevated triglycerides (n=4; 80%), elevated erythrocyte sedimentation rate (n=4; 80%) and elevated D-Dimers (n=4; 80%). Hemophagocytosis phenomena were demonstrated in three (60%) patients. Only one patient had an identified infection prior to diagnosis of MAS.
All patients started corticosteroids in high doses, and three patients had another concomitant immunosuppressor. Two patients died (Case 1 and Case 2, see table 1).
The male patient started on Cyclosporin (CsA) and Intravenous Immunoglobulin (IVIG) simultaneously but died one day after. The 52-year-old female started on methylprednisolone pulses and IVIG, needing a rescue

TO 233 – Table 1. Characterization of 5 cases of Macrophage Activation Syndrome.

No.	Case 1	Case 2	Case 3	Case 4	Case 5
Age (years)	30	52	37	23	25
Sex	Male	Female	Female	Female	Female
Rheumatic disease	Still Disease	SLE	SLE	SLE	Still Disease
Maximum Temperature (ºC)	39,4	38,6	39,0	39,4	39,5
Rash	Yes	No	No	No	Yes
Hepatomegaly	No	No	Yes	Yes	Yes
Splenomegaly	Yes	No	Yes	Yes	Yes
Adenomegaly	No	No	No	Yes	Yes
Arthritis	No	No	No	No	No
Encephalopathy	Yes	Yes	Yes	Yes	No
Serositis	Yes	Yes	Yes	Yes	No
Diarrhoea	Yes	Yes	Yes	Yes	No
Leukocytes (G/L)	0,5	2,1	0,8	3,4	8,7
Neutrophils (G/L)	0,0	1,2	0,5	2,8	5,12
Haemoglobin (g/dL)	8,5	9,8	8,2	8,8	9,6
Platelets (G/L)	40	49	50	199	206
Erythrocyte Sedimentation Rate (mm/h)	17	25	24	51	35
C Reactive Protein (mg/dL)	22,8	7,58	3,61	4,74	4,71
Procalcitonin (ng/dL)	Not performed	0,7	Not performed	0,45	Not performed
Aspartate Transaminase (U/L)	18	45	1466	519	191
Alanine Transaminase (U/L)	8	56	577	293	118
Lactate Dehydrogenase (U/L)	426	360	1035	1323	2526
Triglycerides (mg/dL)	Not performed	164	390	169	342
Prothrombin Time (seconds)	40,3	10,7	20,0	10,7	15,3
INR	3,1	0,9	1,7	0,9	1,3
Activated Partial Thromboplastin Time (seconds)	54,3	39,1	51,7	32,0	36,0
D-Dimer (ng/mL)	2320	12144	734	2179	Not performed
Fibrinogen (mg/dL)	390	262	162	294	180
Ferritin (ng/mL)	> 1650	9631	18614	8814	21807
Soluble CD25 (pg/mL)	Not performed	Not performed	387	2482	Not performed
Myelogram (presence of hemophagocytosis)	Yes	Yes	No	No	Yes
Bacterial Cultures	Negative	Negative	Negative	Positive (urine)	Negative
Viral Serologies	Negative	Negative	Negative	Negative	Negative
Treatment					
Corticosteroid	MPDN 100 mg/ day	MPDN 500 mg/ day for 3 days	DX 20 mg/day	MPDN 1 g/day for 3 days	MPDN 1 g/day for 5 days
Concomitant Immunosuppressor	CsA 100 mg 2id + IVIG 20 g/day	IVIG 2g/kg/day for 5 days	No	No	Anakinra 400 mg/ day + CsA 5 mg/ kg/day
Rescue Immunosuppressor	No	CsA 150 mg/day	No	No	No
Complications	Death	Sepsis	No	No	No
Outcome	Death	Death	Improvement	Improvement	Improvement

Note: the 23-year-old Female performed a myelogram which did not demonstrate any hemophagocitosis, but the sample was not representative and there was contamination with peripheral blood. DX: Dexamethasone; IVIG: Intravenous Immunoglobulin; MMF: Mycophenolate Mofetil; MPDN: Methylprednisolone; SLE: Systemic Lupus Erythematosus.

therapy with some improvement of MAS features but developed sepsis, leading to death.
The other three patients achieved resolution of MAS features without any direct complication. The average follow-up time was 28,7 months (12 to 50 months), however one patient died 24 months after MAS diagnosis.
Discussion and Conclusion: Our findings are similar to other reports, and serositis was found in 4 of 5 (80%) patients. Interestingly, diarrhoea was found in 4 of 5 (80%) patients and it is not considered in several studies. These observations may imply that there are some clinical features in MAS that need to be further assessed as well as other treatment options for these patients.

241 - IS JAK-STAT KEY TO DISEASE PROGRESSION IN EARLY ARTHRITIS?

Rui Lourenço Teixeira[1, 2, 3, 4], Catarina Tomé[2], Ana Rita Faísca[2], JE Fonseca[1, 2, 3], Alcobia[4], Rita A. Moura[2]
[1]Clínica Universitária de Reumatologia, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Rheumatology Research Unit, Instituto de Medicina Molecular, Faculdade de Medicina da Universidade de Lisboa, Lisboa, Portugal, [3]Rheumatology and Metabolic Bone Diseases Department, Hospital de Santa Maria, Centro Hospitalar e Universitário Lisboa Norte, Lisboa, Portugal, [4]Instituto de Histologia e Biologia do Desenvolvimento, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal

Introduction: Janus kinase inhibitors (JAKi) suppress the activity of JAK tyrosine kinases, interfering with JAK-signal transducer and activator of transcription (STAT) signaling pathway, which is critical for immune cell proliferation, survival and differentiation. Our group has demonstrated that early treatment with a JAKi, in animal models, abrogates disease and prevent bone damage. We hypothesize that JAK-STAT pathway is key to chronic arthritis onset.
Objectives: The main goal of this study is to characterize the JAK-STAT signaling pathway activation in untreated early arthritis patients.
Methods: Peripheral blood mononuclear cells were isolated from blood samples collected from untreated early arthritis patients (<12 months of disease duration). The frequency, phenotype and STAT phosphorylation (by median fluorescence intensity, MFI) levels were evaluated on T and B cells, monocytes and dendritic cells (DC) by flow cytometry. A group of age and sex-matched healthy individuals was included for comparison.
Results: The frequency of total CD19+ B cells was similar between patients (n=12) and controls (n=7), although patients presented a significantly decreased level of pre-switch memory B cells when compared to controls. No significant differences were observed in naïve, post-switch memory, double negative B cells, transitional B cells and plasmablasts. The frequency of total CD3+ T cells, CD14+ monocytes and DCs was similar, however patients had significantly lower levels of plasmacytoid DCs. In addition, we found that STAT3 phosphorylation levels were significantly higher in B cells and DCs in early arthritis patients. The STAT1, STAT5 and STAT6 phosphorylation levels were similar in T and B cells, monocytes and DCs, when compared to controls.
Conclusions: Alterations in the frequencies of circulating memory B cell subsets and pDCs, but not in T cells and monocytes, are found in untreated early arthritis patients when compared to healthy controls. Changes in STAT3 phosphorylation MFI levels observed in B cells and DCs from early arthritis patients in comparison to controls support an early activation of JAK-STAT pathway in the initial phase of arthritis and a role of these cells in disease pathogenesis.

243 - PLEUROPARENCHYMAL FIBROELASTOSIS ASSOCIATED WITH INFLAMMATORY RHEUMATIC DISEASES - EXPERIENCE FROM A SINGLE CENTER

Joao Paulo Vilas-Boas[1]
[1]Rheumatology Department, Hospital Infante D. Pedro (CHBV), Aveiro, Portugal

Introduction: Pleuroparenchymal fibroelastosis (PPFE) is an idiopathic interstitial pneumonia characterized by fibrosis of the lung parenchyma and pleura, resulting in architectural distortion and loss of respiratory capacity. The etiology is unknown, being most case idiopathic. Conditions associated with secondary PPFE include inflammatory rheumatic diseases (IRD), and there is an increasing recognition of this association, the true prevalence of which is unknown. The present study seeks to characterize the demographic and clinical characteristics of patients with IRD and PPFE followed at a reference center for interstitial lung disease.
Methods: A survey was carried out of patients with a diagnosis of PPFE followed in a Pulmonology – Interstice ambulation center at Centro Hospitalar Universitário do São João between March 2019 and March 2023, selecting patients with associated IRD. The diagnosis of PPFE was made through imaging, histology or after multidisciplinary discussion, while the diagnosis of IRD was made by a rheumatologist. Data was collected at the time of PPFE diagnosis and included demographic variables, type of associated IRD, reported symptoms, presence of other patterns of interstitial in-

volvement, respiratory volumes and diffusion capacity.
Results: 164 patients with PPFE were identified. 9.8% (n=16) had associated IRD. The mean age at PPFE diagnosis was 60±9.6 years, with a predominance of female patients (75%; n=12). The most frequently associated IRD was rheumatoid arthritis (n=7;44%), followed by systemic lupus erythematosus (n=3;19%), undifferentiated connective tissue disease (n=3;19%), idiopathic inflammatory myopathy (n=2;13%) and systemic sclerosis (n=1;6%). Most patients (81%; n=13) had a diagnosis of IRD prior to the diagnosis of PPFE, with a mean follow-up interval of 1.3 years. As for the symptoms at the time of diagnosis, the most frequently reported was dyspnea (n=13;81%), followed by non-productive cough (n=10;63%). All patients had pulmonary functional involvement compatible with a restrictive pattern at diagnosis. In the population studied, the mean vital forced capacity was 72.8%±19.1 and the forced expiratory volume at the first second was 76.1%±25.6. The carbon dioxide diffusion capacity was reduced in 88% (n=14) of the patients, with a mean of 65.6%±23.3. Regarding other patterns of concomitant interstitial involvement, 3(19%) patients had non-specific interstitial pneumonia and 1(6%) usual interstitial pneumonia.
Conclusion: In this study, 9.8% of patients with PPFE had IRD, so autoimmunity should not be considered a rare cause of PPFE. As in the idiopathic forms, the presence of PPFE associated with DRI leads to a decline in the lung function with a restrictive pattern. PPFE can appear before or after the diagnosis of IRD, which is why multidisciplinary collaboration between rheumatology and pulmonology is important in order to identify these patients early. Future prospective studies will be needed to delineate in detail of the natural history and prognosis in this sub-entity.

244 - PROMPT START OF DISEASE-MODIFYING TREATMENT ALLOWS FOR HIGH RATES OF REMISSION IN PATIENTS WITH EARLY ARTHRITIS: ADDED VALUE OF AN EARLY ARTHRITIS CLINIC

Rui Lourenço Teixeira[1, 2, 3, 4], Rita da Silva Vieira[3], Maria João Saavedra[1], Rita A. Moura[2], Alcobia[4], Joaquim Polido Pereira[1, 3], JE Fonseca[1, 2, 3], Vasco C Romão[1, 3]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Rheumatology Research Unit, Instituto de Medicina Molecular, Faculdade de Medicina da Universidade de Lisboa, Lisboa, Portugal, [3]Clínica Universitária de Reumatologia, Faculdade de Medicina da Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [4]Instituto de Histologia e Biologia do Desenvolvimento, Faculdade de Medicina, Universidade de Lisboa, Lisboa, Portugal

Introduction: Early arthritis clinics allow the prompt identification, treatment, and follow-up of patients with recent-onset arthritis. In addition to rheumatoid arthritis (RA), current evidence suggests a window of opportunity to start treatment and improve long-term outcomes that may also exist for other inflammatory arthropathies.
Objectives: To fully characterize our cohort of early arthritis and to evaluate the proportion of patients achieving clinical remission (defined by Disease Activity Score 28-joints with 4 variables C-reactive protein (DAS-28-4V-CRP) at month 6 of follow-up.
Methods: We included patients followed-up in our early arthritis clinic from 2015 to 2022. We collected demographic and clinical variables, including symptom duration, disease activity, treatment, and final diagnosis. Patients were followed-up for a minimum period of 6 months, with visits at months 0, 1, 3, and 6. All patients were treated with short-term glucocorticoids and conventional synthetic disease-modifying anti-rheumatic drugs (csDMARDs) with a treat-to-target approach.
Results: We included 130 patients (74.4% female) with a mean age of 59.2±16.1 years. Symptom duration to first visit and treatment start was 5.59±2.1 and 6.59±2.1 months, respectively. The most common articular syndromes were chronic additive polyarthritis (65.5%), followed by oligoarthritis of medium and large joints. All patients received low-dose oral prednisone, with a mean dose of 7.5±2.5 mg. The most frequent first-line therapy used was methotrexate (92%), followed by leflunomide (4%), sulfasalazine (2%), and hydroxychloroquine (2%). In about 10% of the patients, methotrexate was switched to another drug due to toxicity or intolerance. Rheumatoid factor (RF)/anti-citrullinated protein antibodies (ACPA)-seropositive RA was the most common diagnosis (47%), followed by seronegative RA (26%) and psoriatic arthritis (10%). Of note, more than two-thirds of patients achieved clinical remission at 6 months. Health Assessment Questionnaire (HAQ) improvement was 1.5 ± 0.4 points. (Table 1)
Conclusions: Our early arthritis clinic allowed for the prompt orientation, diagnosis, and treatment of patients with recent onset arthropathies. This has resulted in a high percentage of patients reaching disease remission at 6 months, in accordance with a window of opportunity for timely treatment and improved outcomes. Our work highlights the relevance of early arthritis clinics in tertiary rheumatology centers.

TO 244 – Table 1 – Clinical characterization of early arthritis patients at baseline and 6-month follow-up.

N=130	Baseline	6-months
Age (years), mean±SD	59.2 ± 15.7	
Female, n (%)	74.4	
Caucasian, n (%)	85	
Disease duration (months), mean±SD	5.6 ± 2.1	
Presentation (%) • Polyarticular	64.2	
• Oligoarticular	35.8	
Seropositive for RF and/or ACPA, n (%)		
• RF-positive	46.8	
• ACPA-positive	44.4	
Erosive disease (%)	10.1	16.6
Tender joint count-28 (TJC28) , mean±SD	10.9 ± 7.0	1.9 ± 1.8
Swollen joint count-28 (SJC28) , mean±SD	7.5 ± 5.4	0.8 ± 2.0
Patient global assessment (visual analogic scale 0-100), mean ± SD	71.2± 16.8	16.8± 18.7
C-reactive protein (mg/dL), mean±SD	2.5 ± 1.5	0.5 ± 0.2
Erythrocyte sedimentation rate (mm/h), mean±SD	56.7 ± 32.7	24 ± 14.6
DAS28-CRP, mean±SD	4.8 ± 2.0	2.2 ± 0.8
Disease activity (DAS28-CRP), n (%)		
High disease activity (>5.1)	32 (34.8)	0 (0)
Medium disease activity (3.2-5.1)	47 (51.1)	9 (10)
Low disease activity (2.6-3.2)	13 (14.1)	18 (19.6)
Remission (<2.6)	0 (0)	65 (70.2)
Health Assessment Questionnaire (HAQ), mean±SD	2.5 ± 0.8	1.1 ± 0.2
Treatment, n (%) / [Dose (mg), mean±SD]		
Prednisone	130 (100) [7.5 ± 2.5]	
Methotrexate	120 (92.3) / [17.5 ± 2.3]	
Leflunomide	5 (3.8) [20.0 ± 0]	
Sulfasalazine	3 (2.5) [1500.0 ± 408.2]	
Hydroxychloroquine	2 (1.6) [400.0 ± 0]	
Final diagnosis, n (%)		
RF/ACPA+ rheumatoid arthritis	62 (47.7)	
RF/ACPA+ rheumatoid arthritis	34 (26.2)	
Psoriatic arthritis	13 (10)	
Polymyalgia rheumatica	10 (7.7)	
Systemic lupus erythematosus	4 (3.1)	
Mixed connective tissue disease	4 (3.1)	
Anti-synthetase syndrome	1 (0.8)	
Undifferentiated arthritis	2 (1.6)	

246 - OSTEOPOROSIS TREATMENT ADHERENCE 12-MONTHS IN A PORTUGUESE FRACTURE LIAISON SERVICE

Susana P. Silva[1, 2], Maria Céu Morais[1, 2], Graça Costa[1, 2], Carolina Mazeda[1, 2], Joao Paulo Vilas-Boas[1, 2], Sofia Ferreira Azevedo[1, 2], C. Pinto Oliveira[1, 2], Carolina Vilafanha[1, 2], Pedro Miguel Teixeira[1, 2], Gisela Eugénio[1, 2], Anabela Barcelos[1, 2]

[1]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [2]Centro Académico Clínico Egas Moniz Health Alliance, Portugal, Aveiro, Portugal

Background: Osteoporosis (OP) treatment reduces new fractures and mortality. However, many patients with osteoporotic fractures are not treated adequately. A Portuguese study found that only 12.1% of the patients who suffered a hip fracture initiated treatment1. It is recognized that treatment persistence is crucial to reach optimal clinical outcomes. However, a treatment persistence of 3.4% in a 24-month period was described in our population2. Fracture Liaison Services (FLS) have been established worldwide, with positive effects on treatment prescription and adherence. We aimed to evaluate treatment adherence in our FLS and predictors of its persistence.
Methods: This retrospective study included patients admitted in the Orthopaedic/Traumatology ward, who suffered a fragility fracture between January 2019 to December 2022. OP treatment was started according to the Portuguese recommendations and adherence to treatment, new falls, new fractures, and pain were reassessed over a 1-year period. Patients were divided in two groups: discontinued and maintained OP treatment. Demographic, comorbidities, and clinical data were collected. Predictors of treatment adherence were assessed using a logistic regression, and a p value ≤ 0.05 was considered statistically significant.
Results: A total of 220 patients were included, of which 176 (80%) started OP treatment: 79.3% oral bisphosphonate, 10.7% denosumab and 10.1% intravenous bisphosphonate. Thirty-four patients lost follow-up and 7 patients died. After 12 months of follow-up, 133 (74.7%) patients-maintained therapy. Patients who discontinued treatment were older (75.97±10.69 vs. 79.59±8.80, p=0.050), had more respiratory diseases (3.1% vs. 13.3%, p=0.010), had fewer previous OP treatment (24.8% vs. 8.9%, p=0.023) and had more secondary fractures (p=0.010). Patients that maintained OP treatment received more parenteric/subcutaneous formula (25.8% vs. 6.8%, p= 0.008). We didn't found differences in independent status, early menopause, parental hip fracture, calcium consumption, other comorbidities, smoking and alcohol habits, corticotherapy, previous fractures, vitamin D levels, institutionalization, and mortality. Patients treated with parenteric/subcutaneous OP treatment were more likely to maintain treatment, after adjusted for age and sex (OR 4.319, 95% C.I. 1.128-16.540, p=0.033). On the other hand, patients who had respiratory chronic diseases were less likely to maintain treatment (OR 0.208, 95% C.I. 0.045-0.962, p=0.044). Age at fracture, male gender and previous OP treatment were not significant predictors of adherence. The most common reason for treatment discontinuation was treatment complexity and self-reported adverse events.
Conclusion: In our FLS, 80% of patients started OP treatment after a fragility fracture. After 1-year, 74.7% of patients were adherent to OP. We also observed that parenteric/subcutaneous OP treatment were predictors of adherence to OP treatment. We believe that the adoption of the FLS model, could lead to better OP outcomes. Studies with larger cohorts and longer follow-up periods are needed to confirm these findings.

248 - ULTRASOUND OF THE HAND AND WRIST JOINTS IN SYSTEMIC SCLEROSIS: TEN YEARS OF FOLLOW-UP - SYNOVIAL INVOLVEMENT AND RELATIONSHIP WITH DISEASE FEATURES

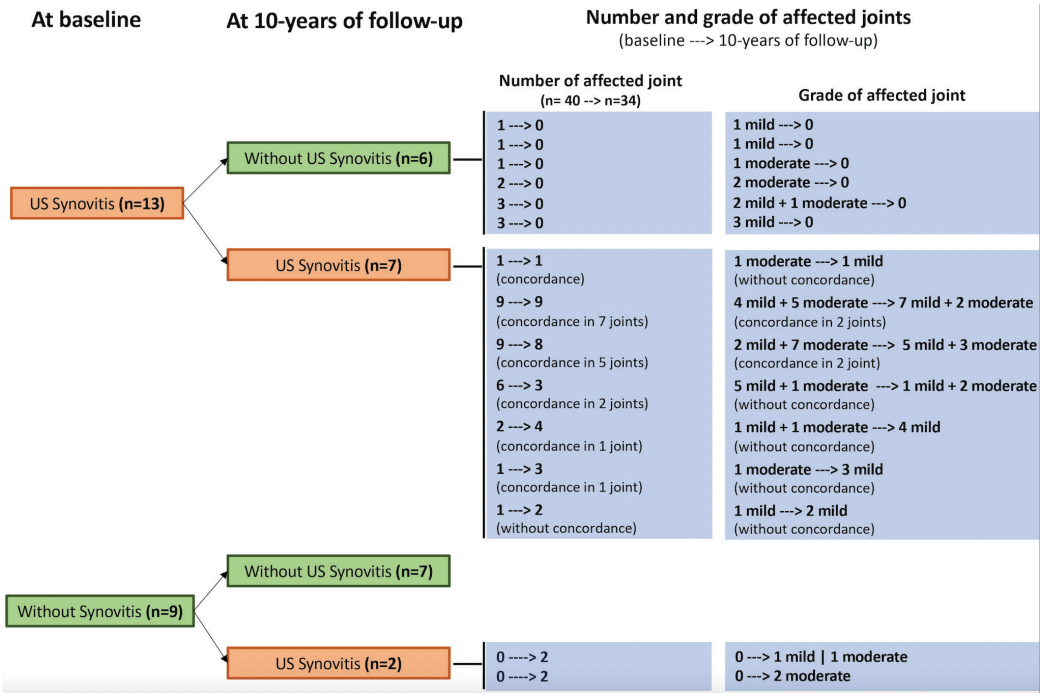
Ana Isabel Maduro[1], Mariana Luis[1, 2], Maria João Salvador[1, 2], JAP da Silva[1, 2], Sara Serra[1]
[1]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal

Background: Musculoskeletal (MSK) hand involvement is common in Systemic Sclerosis (SSc). The skin thickness and contracture make it difficult to evaluate arthritis clinically. It was recognized that ultrasonography (US) is superior to clinical examination in detecting wrist and hand synovitis and tenosynovitis in SSc. However, the US pattern and progression in these patients still need to be understood.
Objective: To describe the main ultrasonographic findings in an SSc cohort followed over a 10-year period; to investigate their correlation with clinical, immunological and radiographic features; and to evaluate the US synovitis progression and associated factors.
Material and Methods: Observational prospective single-center study including SSc patients fulfilling the ACR'1980 or ACR/EULAR'2013 classification criteria, followed up at our university hospital between 2010 and 2022. Patient characteristics were collected, including demographic, clinical and immunological features. MSK hand involvement was assessed by clinical examination, radiography and US at baseline and after 10 years. The same rheumatologist, blinded to the clinical, laboratory and radiographical features, performed US at both times to assess the presence of synovitis, tenosynovitis, erosions, osteophytes and calcifications, according to OMERACT definitions.
Results: We included 22 SSc patients, 90.9% (n=20) females with a median age of 57.50 (IQR 11.7) years at baseline and median disease duration of 11.0 (IQR 7.5) years. Of these, US synovitis was found in 13 (59.1 %) and 9 (40.9%) at baseline and after 10 years, respectively. 8 (36.4%) patients had a positive Power Doppler (PD), at baseline. Only 2 (9.1%) patients had a positive PD at 10 years. The pattern of US synovitis evolution over 10 years (Figure 1) varied among the patients: 7

TO 248 – TABLE 1. Clinical, Ultrasonographic and Radiological findings in SSc patients at baseline and after 10 years of follow-up

	Baseline (n=22)	After 10 years (n=22)	p-value
Clinical articular involvement			
Arthralgias	9 (40.9)	7 (31.8)	0.727
Patients with painful joints (on palpation), n(%)	11 (50)	8 (36.3)	0.096
*Number of painful joints (meanSD)	1.68 (2.1)	0.95 (1.4)	0.127
Patients with ³1 swollen joint, n(%)	4 (18.1)	1 (4.5)	0.250
*Number of swollen joints (meanSD)	0.27 (0.7)	0.1 (0.4)	0.046
Ultrasonographic findings			
Synovitis*	8 (36.4)	9 (40.9)	1.000
Number of joints with synovitis (meanSD)	1.8 (2.7)	1.6 (2.6)	0.457
Distribution of synovitis, n(%)			
Wrist	8 (36.4)	7 (31.8)	1.000
MCP joints	9 (40.9)	7 (31.8)	0.727
PIP joints	4 (18.1)	3 (13.6)	1.000
DIP joints	2 (9.0)	1 (4.5)	1.000
Power Doppler, n(%)	8 (36.4)	5 (22.7)	0.250
Tenosynovitis, n(%)	5 (22.7)	14 (63.6)	0.004
Erosions, n(%)	0 (0)	1 (4.5)	-
Osteophytes, n(%)	10 (45)	12 (54.5)	0.272
Calcifications#, , n(%)	0 (0)	1 (4.5)	-
Radiological findings			
Erosions, n(%)	0 (0)	1 (4.5)	-
Calcinosis, n(%)	7 (31.8)	9 (40.9)	0.180
Osteophytes, n(%)	5 (22.7)	8 (36.3)	0.250
Acroosteolysis, n(%)	1 (4.5)	3 (13.6)	0.250

* Ultrasonographic synovitis was defined as a hypoechoic synovial hypertrophy that was non-displaceable and poorly compressible, with or without PD signal (OMERACT definition). #Calcifications in the tendon, tendon sheath, intraarticular or soft tissue were considered



TO 248 – Figure 1. Progression of US synovitis

did not present synovitis at any time; 6 had complete resolution of the US synovitis (1 of them being medicated with corticoids, due to pulmonary involvement); 2 did not have US synovitis at baseline, but presented it at 10 years of follow-up. 7 maintained US synovitis at baseline and after 10 years, although with variation in the number of affected joints. In many cases with persistent US synovitis, the joints involved were the same, with mild synovitis. In three patients with apparent clinical synovitis, US synovitis was not confirmed. At 10 years of follow-up, we found US and radiographic erosions in one patient, respectively. These 2 patients also had osteophytes; 1 of them had US synovitis at baseline and 10 years. Comparing patients with and without US at baseline, patients with US synovitis have a higher body mass index [27.5(IQR 5.5) vs 23.1(IQR 1.9), p=0.001]. No statistically significant difference was found concerning general clinical and immunological characteristics of the disease or hand disability.

Conclusion: In this cohort, we found US synovitis in 59.1% and 40.9% of patients at baseline and after 10 years, respectively. The pattern of joint involvement is very variable between patients at both time points, with some experiencing persistent synovitis while others had migratory involvement. However, in most cases, US synovitis is mild and does not cause any significant joint damage or functional impairment. Patients with US synovitis tended to have a higher BMI, but we did not find any association between US synovitis and clinical (such as organ involvement) and immunological characteristics, or hand disability, despite the limited sample size.