COMUNICAÇÕES ORAIS

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007 - MENTAL HEALTH BURDEN IN IDIOPATHIC INFLAMMATORY MYOPATHIES: A COMPARATIVE POPULATION-BASED STUDY

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Background: Idiopathic inflammatory myopathies (IIMs) are chronic systemic diseases that affect patients' quality of life. While muscular and extramuscular involvement are well characterized, the psychological burden associated with this group of disorders – particularly anxiety and depression – remains poorly understood.

Objectives: To assess the prevalence and severity of anxiety and depression symptoms in adults with IIM, compared to a matched control group without rheumatic disease. We also aimed to explore whether comorbidities, clinical features, and laboratory markers could predict psychological distress among IIM patients.

Methods: We conducted a cross-sectional, case-control study with prospective data collection from adults with IIM followed at the Myositis Outpatient Clinic at ULSSM (March–June 2025), using the validated Hospital Anxiety and Depression Scale (HADS). A control group, matched 3:1 by sex and age, was selected from the Portuguese population-based EpiReumaPt study (2011–2013). HADS scores of 8–10 indicated possible cases of anxiety (HADS-A) and depression (HADS-D), and scores ≥11 defined probable cases. Group comparisons were per-

007 – TABLE 1. Demographic, clinical, and mental health characteristics of IIM patients and matched controls

	Patients (n = 65)	Controls (n = 195)	P-value
Demographic data			
Female sex, n (%)	51 (78.5)	153 (78.5)	1.000
Age, years, mean ± S.D.	51.8 ±15.3	51.0 ±15.8	0.712
Body Mass Index (BMI), kg/m^2, median (IQR)	24.4 (7.0)	26.0 (5.2)	0.218
Smoking status, n/N (%)			0.503
Smoker	4/59 (6.8)	20/195 (10.3)	
Never smoker	39/59 (66.1)	134/195 (68.7)	
Former smoker	16/59 (27.1)	41/195 (21.0)	
Comorbidities, n (%)			
Arterial hypertension	20 (31.0)	63 (33.0)	0.742
Neoplastic disease	15 (23.1)	21 (10.9)	0.014
Thyroid disease	15 (23.1)	18 (9.3)	0.004
Auto-report of mental disease	12 (18.5)	28 (14.4)	0.437
Osteoarthritis	14 (21.5)	16 (8.6)	0.006
Osteopenia/Osteoporosis	18 (27.7)	9 (4.9)	<0.001
HADS1 score			
HADS-A, median (IQR)	7.0 (6.0)	5.0 (5.0)	0.008
HADS-A ≥ 8, n (%)	32 (49.2)	60 (30.1)	0.007
HADS-A ≥ 11, n (%)	12 (18.5)	30 (15.4)	0.559
HADS-D, median (IQR)	4.0 (7.0)	3.0 (5.0)	0.003
HADS-D ≥ 8, n (%) 2	1 (32.3)	28 (14.4)	0.001
HADS-D ≥ 11, n (%)	7 (10.8)	16 (8.2)	0.528

n: number of patients positive for the variable of interest; N: number of patients without missing information regarding the variable of interest; S.D.: standard deviation; IQR: interquartile range; 1 HADS: Hospital Anxiety and Depression Scale.
*Associations between variables were analysed with appropriate statistical tests, and multiple comparisons were adjusted using the Holm-Bonferroni correction. Values in bold indicate statistical significance at the 0.050 level ($p < 0.050$).

formed using appropriate statistical tests, and multiple comparisons were adjusted using the Holm-Bonferroni correction. Multivariate logistic regression was used to identify predictors of psychological distress.

Results: We included 65 IIM patients, of whom 78.5% were female, with a median disease duration of 7.4 years (IQR 14), and 195 age- and sex-matched controls. No significant differences were found in sociodemographic or lifestyle variables, including BMI (p=0.218) and smoking status (p=0.503) (Table 1).

Compared with controls, IIM patients had a higher percentage of neoplastic disease (23.1% vs. 10.9%, p=0.014), thyroid disease (23.1% vs. 9.3%, p=0.004), osteoarthritis (21.5% vs. 8.6%, p=0.006), and osteoporosis (27.7% vs. 4.9%, p<0.001). IIM patients showed significantly higher HADS-A [7.0 (IQR 6.0) vs. 5.0 (IQR 5.0), p=0.008] and HADS-D scores [4.0 (IQR 7.0) vs. 3.0 (IQR 5.0), p=0.003] than controls. A greater proportion of IIM patients scored ≥8 in HADS-A (49.2% vs. 30.1%, p=0.007) and HADS-D (32.3% vs. 14.4%, p=0.001). No significant differences were observed for the ≥11 cut-off.

Among IIM patients, current corticosteroid use was significantly more frequent in those with anxiety or depression (78.1% vs. 54.5%, p=0.045; and 85.7% vs. 56.8%, p=0.021, respectively). Higher aldolase levels were observed in those with depressive symptoms [13.4 (IQR 13.9) vs. 9.2 (IQR 3.9), p=0.032]. No significant associations were found for disease duration, sex, CK levels, MMT-8 or DAS-skin scores, or extramuscular features.

In multivariable analysis, IIM (OR 2.24, 95% CI 1.08–4.68; p=0.031) and osteoporosis (OR 3.32, 95% CI 1.33–8.24; p=0.010) were independently associated with depressive symptoms (HADS-D ≥8). Neoplastic disease (OR 2.65, 95% CI 1.22–5.77; p=0.014) and osteoporosis (OR 3.65, 95% CI 1.48–9.03; p=0.005) were independently associated with anxiety symptoms (HADS-A ≥8), whereas IIM was not.

Conclusion: Patients with IIM face a significantly greater psychological burden than the general population, with anxiety and depression affecting nearly half and one-third of patients, respectively. Osteoporosis and neoplastic disease emerged as key contributors to psychological distress, while corticosteroid use and elevated aldolase levels were also associated with worse outcomes among IIM patients. These findings underscore the importance of systematic mental health assessment in IIM clinical care.

008 - HOW CALCULATING CONSENSUS CHANGE SCORES CAN GO WRONG: LESSONS FROM MULTI-READER IMAGING ASSESSMENTS IN AXIAL SPONDYLOARTHRITIS

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Background: In research, imaging findings are often assessed by multiple readers and individual readers' scores are combined into aggregate scores to determine the presence of lesions. In longitudinal studies, the focus shifts to change in lesions. Diverse strategies for aggregating change scores are at hand, but may yield different results. We aimed to establish a methodological framework for aggregating change scores in imaging assessments, using the example of syndesmophyte progression in axial spondyloarthritis (axSpA).

Methods: Data from the Sensitive Imaging in Ankylosing Spondylitis cohort were used, including patients with axSpA and established spinal damage. Syndesmophytes were assessed on vertebral corners (hereafter 'level') on conventional radiography (CR) and low-dose computed tomography (ldCT) at baseline and 2 years. CR were scored by 3 central readers and ldCT by 2, blinded for time order. Consensus was determined by majority reader agreement. New syndesmophytes (change score) were assessed after 2 years by two strategies. Strategy 1 calculates change score per reader, then determines consensus on the change per level. Strategy 2 derives consensus change scores from consensus status scores (syndesmophyte present/absent) (Box 1). The means and standard deviations (SD) of the different aggregate change scores were compared. For analyses at the patient level, the total number of new syndesmophytes was averaged across readers, only if patients had ≤25% scores missing per spinal segment at both timepoints.

Results: Complete data from 52 patients were used. Figure 1a provides 3 examples of assessment of a new syndesmophyte. While both strategies do not yield different consensus change scores in examples 1 and 2, they do in example 3. Here, when consensus change score is derived from change scores of individual readers (strategy 1), because only reader C detected a new

008 – TABLE 1. Explanation of two strategies for defining a consensus change score in multi-reader imaging assessments

Strategy	Description	Key steps
1	Consensus change score is based on the agreement of individual readers change scores	Assess delta (progression) of a lesion per level for each reader Calculate consensus change score by assessing the majority reader agreement on change score per level
2	Consensus change score is based on the change of consensus status scores	Calculate consensus status scores by assessing the majority reader agreement on lesion presence at each timepoint per level Calculate change scores based on delta from consensus status scores per level

1a. Examples of calculating new syndesmophytes based on scores per level of three readers according to two strategies

	Reader A	Reader B	Reader C	Consensus status score	Strategy 1: Consensus change score based on individual readers' change score	Strategy 2: Consensus change score based on change of consensus status scores	Average of total new syndesmophytes across readers (patient level)
Example 1							
Syndesmophyte at baseline	0	0	0	0			
Syndesmophyte at 2 years	0	1	0	0			
New syndesmophyte	0	1	0		0	0	
Example 2							
Syndesmophyte at baseline	0	0	0	0			
Syndesmophyte at 2 years	1	1	0	1			
New syndesmophyte	1	1	0		1	1	
Example 3							
Syndesmophyte at baseline	0	1	0	0			
Syndesmophyte at 2 years	0	1	1	1			
New syndesmophyte	0	0	1		0	1	
Total new syndesmophytes	**1**	**2**	**1**		**1**	**2**	**1.33**

1b. Total number of new syndesmophytes at the level and at the patient level in the SIAS cohort calculated according to two strategies

Strategy	Imaging modality	Number of patients	Number of vertebral corners	Total new syndesmophytes: agreement at the level Mean (SD)	Range	Total new syndesmophytes: average across readers at the patient level Mean (SD), n*
1	CR	51	1035	0.69 (0.95)	0 – 4	0.66 (1.10), n=36
2				0.86 (1.00)	0 – 4	
1	ldCT	52	9035	2.35 (4.44)	0 – 18	5.55 (6.65), n=48
2				7.23 (7.46)	0 – 41	

* To calculate the total number of new syndesmophytes at the patient level, only patients with ≤25% scores missing per spinal segment (on ldCT, the cervical and thoracic spine were considered together) at both timepoints were included (with the remaining missing scores considered 'no (new) syndesmophyte').

0: absence of a (new) syndesmophyte; **1:** presence of a (new) syndesmophyte; **CR:** conventional radiography; **ldCT:** low-dose computed tomography; **SIAS:** Sensitive Imaging in Ankylosing Spondylitis cohort.

088 – Figure 1. Comparison of strategies for assessing new syndesmophytes in patients with axial spondyloarthritis after 2 years

syndesmophyte, there is no new syndesmophyte in the aggregated score. In contrast, when consensus status scores are determined first (strategy 2), indicating no syndesmophyte at baseline but one at 2 years, the consensus change score suggests a new syndesmophyte.

Figure 1b illustrates how extrapolating separate level scores by both strategies into patient-sum scores may result in importantly different mean change scores. Strategy 2 is more sensitive, identifying 1.2 times more new syndesmophytes on CR (mean [SD]: 0.86 [1.00] vs 0.69 [0.95]) and 3.1 times more on ldCT (7.23 [7.46] vs 2.35 [4.44]). However, this is accompanied with higher variability. Only strategy 1 represents "true consensus" among readers, however it requires site-level agreement, which compromises sensitivity to change at the patient level. At the patient level, the total number of new syndesmophytes should be calculated as the average of the total number across all readers (CR: 0.66 [1.10]; ldCT: 5.55 [6.65]).

Conclusion: Consensus change scores should be derived from individual readers' change scores. Strategy 1 approaches true change best, while strategy 2 artificially inflates change. At the patient level, the average total number of new syndesmophytes across readers, which has highest sensitivity to change, should be reported. While we focus on syndesmophyte progression in axSpA, this framework is broadly applicable to other imaging findings across multiple diseases. Advanced statistical analysis beyond descriptive purposes should preferably be performed on individual readers' scores, using multilevel models that will properly account for variability in reader assessment.

013 - EMAPALUMAB'S ROLE IN A SEVERE AND TREATMENT-RESISTANT PAEDIATRIC MACROPHAGE ACTIVATION SYNDROME

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Introduction: Macrophage activation syndrome (MAS), a subset of haemophagocytic lymphohistiocytosis (HLH), is a life-threatening condition secondary to rheumatic diseases, characterized by systemic hyperinflammation and subsequent haemophagocytosis. MAS management includes immunomodulation. Emapalumab is an IFNγ directed antibody approved for the treatment of refractory HLH in the USA, but not in Europe.
Case: We report the case of a 15-year-old girl with a severe and highly refractory MAS. She presented with a 5-day history of fever (>39ºC), odynophagia and an erythematous macular rash that accompanied the fever spikes. She was hospitalized and empirical antibiotic therapy started. After 9 days, she was admitted to the intensive care unit for ventilatory and vasopressor support. Blood work showed de novo pancytopenia, hypofibrinogenemia and elevation of AST, ALT, LDH, triglycerides, soluble CD25, serum calprotectin and ferritin (highest 357976ng/ml). Imaging revealed hepatosplenomegaly. A broad infectious screening and immune profile was negative. Myelogram and bone biopsy showed a hypocellular marrow with haemophagocytosis. An exome-based gene panel for inherited immune dysregulation disorders (including 576 genes associated with HLH, autoinflammatory syndromes, and immunodeficiencies) did not identify any pathogenic variants.

She was treated with 5 daily IV pulses of methylprednisolone 1g, followed by oral methylprednisolone 1mg/kg/day, anakinra (IV, maximum 100mg q6h) and cyclosporine (maximum 150mg q12h). Despite an initial response, an infectious complication led to clinical and laboratory worsening of MAS. Cyclosporine was suspended due to drug induced microangiopathy, and anakinra dose was reduced to 100mg q12h due to hepatotoxicity. She was given a new cycle of IV methylprednisolone pulses, 2 IV immunoglobulin perfusions and ruxolitinib was added for a short period. However, pancytopenia persisted, requiring erythrocyte transfusions and G-CSF. On the 41st day of hospitalization, with a ferritin level of 71233ng/ml, emapalumab was started. She received ten 3-day spaced treatments, with progressive and sustained clinical and laboratory improvement, allowing for tapering of corticosteroids and anakinra, and subsequent discharge. She is currently clinically stable with 8 months of follow-up, on canakinumab (switched from anakinra due to local reaction) maintenance treatment for 4 months and reducing steroids.
Discussion: This case highlights the diagnostic and management challenges of MAS at disease onset and supports the role of IFN-γ inhibition in the treatment of severe and resistant disease. It describes a particularly severe and unusually prolonged episode of MAS—lasting over 2 and a half months—as the inaugural manifestation of a likely underlying Still's disease. The diagnosis was initially obscured by the intensity of the hyperinflammatory state, with an exceptionally high ferritin level. MAS was refractory to high-dose corticosteroids, ciclosporin, and IL-1 blockade, with only partial and transient responses. Emapalumab—used here for the first time in Portugal—after 2 and a half months of uncontrolled severe disease, was associated with a rapid and sustained clinical and laboratory remission.

048 - ROMOSOZUMAB IMPROVES BONE MINERAL DENSITY WITHOUT AFFECTING DISEASE ACTIVITY IN MULTIPLE MYELOMA: A 12-MONTH PROSPECTIVE STUDY

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Introduction: Bone damage begins early in the course of plasma cell disorders. Romosozumab (ROMO), a sclerostin inhibitor, has dual anabolic and antiresorptive properties and may offer benefit in this group with high fracture risk.

In this study in patients with multiple myeloma (MM) and osteoporosis, we aimed to evaluate the impact of ROMO over 12 months in bone mineral den-

048 – TABLE 1. BASELINE Characteristics of the 8 patients with SMM (IgG subtype) that received romosozumab for 12 months

	Age	Sex	BMI (kg/m2)	Disease duration (years)	Smoking Status	Previous fracture	Lumbar Spine BMD (g/cm2) T-Score	Femoral Neck BMD (g/cm2) T-Score	Total Hip BMD (g/cm2) T-Score	M-protein (g/L)	Serum FLC ratio (K/λ)	Fractures during FU	Disease progression during FU	After ROMO
1	59	F	19.5	5	No	Yes (peripheric + vertebral)	0.956 -1.9	0.668 -2.6	0.782 -1.8	19.5 (λ)	0.5	No	No	ZOL
2	77	F	28.3	<1	No	Yes (vertebral)	0.810 -3	0.752 -1.9	0.833 -1.4	18 (λ)	0.1	No	No	DMAB
3	77	F	22.2	26	No	No	0.763 -3.4	0.695 -2.4	0.811 -1.6	18 (K)	34.5	No	No	DMAB
4	78	F	22.3	10	No	No	0.905 -3.2	0.826 -1.3	0.821 -1.5	14 (K)	101.5	No	No	DMAB
5	62	F	19.7	<1	No	No	0.550 -5	0.690 -2.4	0.736 -2.2	31 (K)	59.7	No	No	DMAB
6	51	F	21.8	10	Yes, actual	Yes (vertebral)	0.916 -2.2	0.798 -1.5	0.820 -1.5	28 (λ)	0.3	No	No	DMAB
7	71	F	30.9	1	Yes, actual	Yes (vertebral)	0.834 -2.9	0.981 -2	0.866 -1.1	26 (K)	13.3	No	No	DMAB
8	64	F	20.7	2	Yes, former	Yes (vertebral)	0.709 -3.8	0.745 -2	0.872 -1.1	27 (λ)	0.1	No	No	DMAB

Footnote: BMI – body mass index; SMM – smoldering multiple myeloma; BMD – bone mineral density; FLC – free light chains; FU – follow-up; ROMO – romosozumab; ZOL – zoledronic acid; DMAB - denosumab

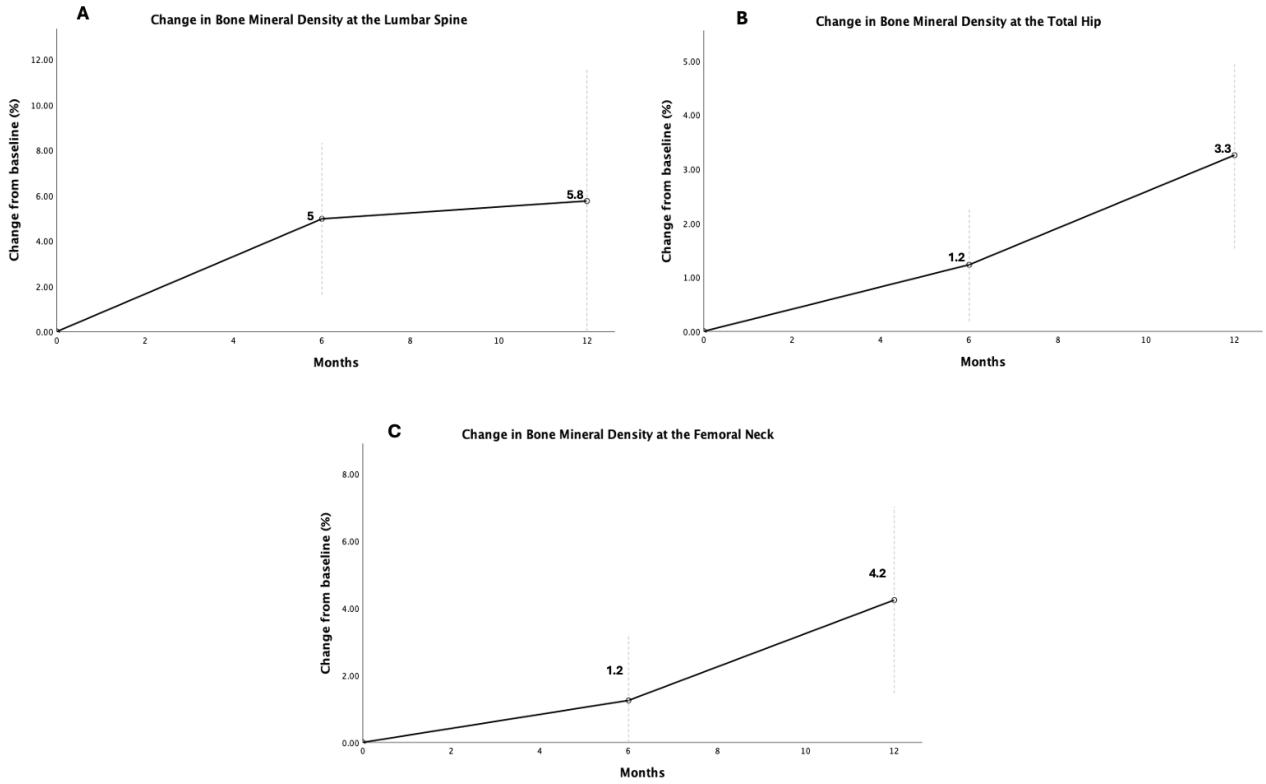
sity (BMD), bone turnover markers (BTMs), high-resolution peripheral computed tomography (HR-pQCT) parameters and MM disease activity.

Methods: We conducted a 12-month prospective observational study of patients with MM and osteoporosis treated with ROMO (210mg/month). Inclusion criteria were: (1) diagnosis of MM according to the International Myeloma Working Group (IMWG) definition, (2) absence of CRAB disease, (3) treatment with ROMO deemed necessary by the treating physician. The primary outcome was bone mineral density (BMD) change at 12 months, other outcomes were bone turnover markers (BTMs) change, high-resolution peripheral computed tomography (HR-pQCT) parameters and MM related parameters (M-protein, immunoglobulins free light chains, β2 microglobulin). Repeated measures were analyzed using a mixed-effects models, with significance set at $p<0.05$.

Results: Eight female patients with MM (mean age 67.4±9.9 years) completed 12 months of ROMO (table 1). A significant BMD increase was observed at all evaluated sites over the study period (figure 1). Lumbar spine BMD increased from 0.822 (0.750-0.908) to 0.832 g/cm² (0.752-0.908) (p=0.046), representing a mean change of 5.8% at 12 months (p=0.048). At the femoral neck, there was an increase from 0.749 (0.694-0.805) to 0.784 (0.729-0.836) g/cm² at (p=0.015), corresponding to a mean change of 4.2% (p=0.020). At the total hip, BMD improved from 0.821 (0.804-0.841) to 0.848 (0.260-0.884) g/cm² (p=0.003), with a percentage change of 3.3% (p=0.002). Bone-alkaline phosphatase (B-ALP) showed a significant decrease by month 12 (13.5 (9.3-17) to 9 (6.12) ng/mL, p=0.022). P1NP rose sharply by month 3, from 60 (49.5-70.3) to 98 (77-115.5) ng/mL (mean change of 117.8%), but then declined, approaching baseline by month 12 (p=0.006). Conversely, CTX showed a tendency for reduction at all time points (p=0.060), with a maximal decrease at the end of the 12 months (-53.6%)

No significant changes occurred in immunoglobulins, M-protein, or light chains. β2-microglobulin showed a downward trend (from 2.35 to 2.1mg/L at month 12, p=0.042), though overall changes were not significant (p=0.092). Regarding HR-pQCT parameters, failure load increased at month 6 (p=0.033) while the other HR-pQCT parameters remained stable throughout the study.

Conclusion: In this exploratory study of patients with SMM and osteoporosis, ROMO significantly improved BMD and modulated BTMs, without any evidence of disease progression over the 12-month period. These findings support ROMO as a potential bone-targeted therapy in patients with osteoporosis and plasma cell disorders.



048 – Figure 1. Percentage Change from Baseline in Bone Mineral Density at the lumbar spine (A), total hip (B) and femoral neck (C)

059 - WHIPPLE'S DISEASE IN RHEUMATOLOGY: INSIGHTS FROM A PORTUGUESE MULTICENTER SERIES

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Background: Whipple's disease (WD) is a rare chronic infection caused by Tropheryma whipplei, typically presenting with a biphasic course. Musculoskeletal (MSK) symptoms, such as migratory arthralgia or arthritis, usually precede gastrointestinal (GI) and systemic manifestations by several years. Immunosuppression may accelerate symptom progression. Owing to its protean features and ability to mimic rheumatic diseases, WD remains a diagnostic challenge in rheumatology. This national series aims to characterize patients with WD initially evaluated for suspected rheumatic conditions.

Methods: Patients diagnosed with WD were retrospectively identified from electronic health records of Rheumatology departments across Portugal. Inclusion required diagnostic confirmation via duodenal histopathology (PAS staining) and/or PCR testing for T. whipplei. Demographic, clinical, and therapeutic data were collected and analyzed using descriptive statistics.

Results: Seven patients were identified (71.4% male), with a mean age at symptom onset of 59.9 ± 8.2 years and a median diagnostic delay of 4 years (IQR 1.4). Regarding MSK symptoms, four had classic migratory arthralgia/arthritis, but presentations were otherwise heterogeneous, mimicking conditions like polymyalgia rheumatica (PMR) (n=1), asymmetric sacroiliitis (n=1), or rheumatoid arthritis (RA) (n=2). Most were RF seronegative and all were ACPA negative. GI symptoms occurred in four patients (57.1%), mainly diarrhea (n=3), and one reported abdominal pain. Systemic features were present in six patients (85.7%), all with weight loss and four with other constitutional symp-

059 – TABLE 1. Demographic and clinical characterization of patients diagnosed with WD (N=7).

Variable	Value
Male sex (N, %)	5 (71.4%)
Age at symptom onset (mean ± SD), years	59.9 ± 8.2
Diagnostic delay (median, IQR), years	4.0 (IQR 1.4)
Delay between musculoskeletal and other involvement (median, IQR), years	2.6 (IQR 3)
Musculoskeletal involvement (N, %)	7 (100%)
Migratory arthralgia/arthritis	4 (57.1%)
Polymyalgia rheumatica	1 (14.3%)
Chronic seronegative polyarthritis	2 (28.6%)
Asymmetric sacroiliitis	1 (14.3%)
Gastrointestinal involvement (N, %)	4 (57.1%)
Diarrhea	3 (42.9%)
Steatorrhea/Malabsorption	2 (28.6%)
Abdominal pain	1 (14.3%)
Systemic involvement (N, %)	6 (85.7%)
Weight loss	6 (85.7%)
Loss of appetite	3 (42.9%)
Fatigue	3 (42.9%)
Fever	1 (14.3%)
Lymphadenopathy	2 (28.6%)
Hepatosplenomegaly	1 (14.3%)
CNS involvement (N, %)	1 (14.3%)
Hemoglobin (mean ± SD), g/dL	10.4 ± 1.2
CRP (mean ± SD), mg/dL	6.1 ± 3.6
ESR (mean ± SD), mm/h	39.7 ± 26.6
RF positive (N, %)	2 (28.6%)
ACPA positive (N, %)*	0 (0%)
Immunosuppressive therapy (N, %)	5 (71.4%)
Steroids	5 (71.4%)
Methotrexate	4 (57.1%)
Biologic therapy	1 (14.3%)
PAS-positive staining on duodenal/jejunal biopsy (N, %)	4 (57.1%)
Positive DNA detection by PCR on duodenal/jejunal biopsy (N, %)*	5 (100%)
Treatment with ceftriaxone plus trimethoprim-sulfamethoxazole	7 (100%)
Treatment duration (range), months	12 to 24

ACPA, anti-cyclic citrullinated peptide antibodies; CNS, Central nervous system; CRP, C-reactive protein; ESR, Erythrocyte sedimentation rate; PAS - Periodic Acid–Schiff RF, rheumatoid factor;. *Performed in 5 patients.

toms. All had anemia (mean Hb 10.4 ± 1.2 g/dL) and elevated inflammatory markers (CRP 6.1 ± 3.6 mg/dL; ESR 39.7 ± 26.6 mm/h). The median interval between MSK and extra-articular symptoms was 2.6 years (IQR 3). Notably, one patient presented with systemic symptoms before MSK involvement, and another developed GI/systemic features only 14 years later. Five patients were immunosuppressed (all with corticosteroids; four with methotrexate; one with biologics). Interestingly, the two who did not receive immunosuppression developed GI or systemic symptoms early (within one year), whereas the only patient treated with biologics did not develop such manifestations. PAS staining was negative in 3 patients (42.9%), with T. whipplei DNA detection by PCR proving essential for diagnosis. All received ceftriaxone followed by trimethoprim-sulfamethoxazole for 12–24 months. Clinical remission occurred within 1–8 weeks.

Discussion: The absence of a uniform MSK presentation highlights WD as a potential "great imitator" in

Sex	Age at onset	Diagnostic delay (yrs)	MSK involvement	GI involvement	Systemic involvement	Delay MSK–GI/systemic (yrs)	Other organ involvement	RF (IU/mL)	Anti-CCP (UA/mL)	Hb (g/dL)*	CRP (mg/dL)*	ESR (mm/h)*	Immunosuppressive therapy	PAS stain (duodenal)	PCR (duodenal)	PCR (other samples)	Antibiotic regimen	Duration (wks)	Time to remission (wks)
M	69	4.0	Polymyalgia rheumatica	NA	Fatigue, anorexia, weight loss	3.9	NA	73.2	0	8.5	4.44	48	PDN 10 mg, MTX 20 mg	Positive	NA	NA	Ceftriaxone 2 g/2 wk + TMP-SMX 960 mg	12	2–4
M	52	4.4	Chronic seronegative polyarthritis	Diarrhea	Fever, hyperhidrosis, fatigue, anorexia, weight loss, generalized lymphadenopathy	4.0	NA	0	0	11	6.67	24	PDN 7.5 mg, MTX 12.5 mg	Positive	NA	NA	Ceftriaxone 2 g/2 wk + TMP-SMX 960 mg	18	6–8
F	47	14.7	Axial and peripheral spondyloarthritis (asymmetric sacroiliitis, migratory arthralgia in lower limbs)	Diarrhea, malabsorption	Weight loss	14.5	NA	1191	NA	9.4	2.48	9	PDN 20 mg	Positive	Positive	NA	Ceftriaxone 2 g/4 wk + TMP-SMX 960 mg	24	4
M	69	4.0	Elderly-onset RA (asymmetric involvement of hands and wrists)	Diarrhea, steatorrhea	Weight loss	–1.0	CNS – ataxia	0	0	9.8	6.12	86	PDN 5 mg, MTX 10 mg	Negative	Positive	CSF	Ceftriaxone 2 g/4 wk + TMP-SMX 960 mg	12	4
F	59	2.0	Migratory polyarthralgia	Abdominal pain	Fatigue, weight loss	1.0	NA	0	NA	11.4	4.36	NA	NA	Negative	Positive	NA	Ceftriaxone 2 g/2 wk + TMP-SMX 960 mg	24	na
M	63	1.5	Migratory polyarthritis	NA	Anorexia, weight loss, lymphadenopathy, mild hepatosplenomegaly	1.2	NA	0	0	10.6	13.62	43	NA	Positive	Positive	NA	Ceftriaxone 2 g/2 wk + TMP-SMX 960 mg	12	1–2
M	60	3.7	Migratory polyarthritis and tibialis posterior tenosynovitis	NA	NA	NA	NA	0	0	11.9	5.12	28	PDN 5 mg, MTX 15 mg, LFN 20 mg, ADA, SEK, UPA	Negative	Positive	Synovial fluid	Ceftriaxone 2 g/2 wk + TMP-SMX 960 mg	Ongoing	1–2

059 – Figure 1. Summary of clinical cases

rheumatology, mimicking RA, spondyloarthritis, and PMR, contributing to diagnostic delays. A high index of suspicion is essential, particularly in patients with refractory rheumatic symptoms, anemia, and weight loss. A longer MSK-to-systemic/GI interval correlated with delayed diagnosis, while early extra-articular features seemed to hasten recognition. This interval was shorter than in previous series. Importantly, non-MSK symptoms preceding joint complaints, as seen in one case, challenge the conventional "MSK-first" paradigm. Our findings suggest early GI/systemic manifestations occurred in patients not exposed to immunosuppression, and the only patient treated with biologics did not develop such symptoms. Thus, the role of immunosuppression in symptom progression may be less predictable than previously thought, warranting further investigation.

070 - PAIN BELIEFS IN ADOLESCENTS: A COMPARATIVE ANALYSIS OF PATIENTS WITH JUVENILE IDIOPATHIC ARTHRITIS AND A POPULATION-BASED COHORT

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Background: Pain beliefs play an important role in pediatric rheumatology, influencing coping strategies and patient-centered outcomes. Adolescents' beliefs about pain can either facilitate adaptive behaviors, such as one's active role in controlling pain, or reinforce maladaptive behaviors, such as exercise avoidance. Little is known about how these beliefs are shaped in the presence of long-term chronic joint inflammation.
Aim: To compare pain-related beliefs between adolescents with and without juvenile idiopathic arthritis (JIA), and to test differences according to the presence of current chronic musculoskeletal pain.
Methods: As part of the FOREUM-funded study SEPIA - Studying Experiences of Pain In Adolescents, we collected data from 21 patients aged 14 to 18 years with a diagnosis of JIA, followed as part of clinical care in 7 rheumatology departments that accepted to participate and reported data to the Portuguese Rheumatic Diseases Register. JIA diagnosis was confirmed based on the International League of Associations for Rheumatology criteria. We compared those with 2266 17/18-year-old adolescents from Generation XXI, a population-based birth cohort from Porto, Portugal. Data were collected using a dedicated mobile app, through which pain was assessed using the Luebeck screening questionnaire. Chronic musculoskeletal pain was considered present when the participant reported a recurrent principal pain in a musculoskeletal site (back, neck/shoulders, upper and lower limbs, hips, unspecified musculoskeletal, or generalized pain) that lasted more than 3 months. Participants also answered a questionnaire including 15 items addressing their beliefs about chronic pain on visual analogue scales scoring from 0 (totally disagree) to 100 (totally agree). Results refer to agreement with each belief and are presented as means and standard deviations and compared using independent samples t-tests.
Results: As seen the Table, the need to continue seeking clinical investigation even in the absence of pathological findings were strongly endorsed by adolescents in the JIA cohort (with chronic musculoskeletal pain: mean 90.9, sd 17.3; no pain: 91.3, 12.8) compared to those in the population cohort (pain: 86.6, 16.8; no pain: 86.9, 18.0). Similarly, participants without recent pain from the JIA cohort agreed more with the belief that physical exercise worsens chronic pain compared to community adolescents (57.3, 34.3 vs. 48.3, 28.0). However, adolescents experiencing chronic pain showed stronger beliefs in their ability to control pain compared to their pain-free peers in both populations (64.9, 25.8 vs. 62.8, 26.0) and JIA (77.7, 30.1 vs. 73.9, 32.5) cohorts. The belief that pain hinders life planning was higher in adolescents without pain in the population cohort compared to their no-pain peers in JIA (40.8, 29.6 vs. 26.1, 28.5). In contrast, optimism about the future was higher in adolescents with no recent pain in the JIA cohort compared to GXXI (72.8, 32.0 vs. 67.2, 28.0).
Conclusion: Pain-related beliefs differ between adolescents with and without chronic pain across both general and clinical populations. Adolescents with JIA, particularly those experiencing pain, adhered more to beliefs on the importance of continued medi-

070 – TABLE 1.

	Population-based cohort G21 (n=2266) mean (SD)			JIA cohort (n=22) mean (SD)		
Question	Chronic musculoskeletal pain (n=518)	No pain (n=1748)	p-value	Chronic musculoskeletal pain (n=11)	No pain (n=11)	p-value
1. If you have chronic pain, it is important to avoid physical exertion until it subsides.	57.60 (29.06)	57.19 (30.29)	0.390	62.45 (33.00)	53.91 (42.37)	0.302
2. Medical examinations should be conducted until the cause of chronic pain is identified.	88.69 (16.31)	89.70 (16.77)	0.108	95.55 (7.46)	90.55 (13.41)	0.148
3. Even if a doctor cannot find the cause of chronic pain, it is important to keep searching.	86.60 (16.81)	86.92 (17.98)	0.354	90.91 (17.33)	91.27 (12.78)	0.478
4. A person with chronic pain will worsen if they engage in physical exercise.	48.68 (26.77)	48.28 (28.03)	0.385	51.82 (31.03)	57.27 (34.32)	0.350
5. When someone experiences chornic pain, family and friends should treat them better.	51.15 (25.49)	55.93 (26.89)	<0.001	57.45 (32.67)	65.91 (34.47)	0.281
6. Thinking about chronic pain worsens the pain.	49.10 (30.99)	52.78 (31.17)	0.009	62.91 (27.40)	62.18 (33.83)	0.478
7. Feeling anxious or stressed worsens chronic pain.	69.18 (25.59)	70.25 (24.80)	0.201	71.82 (21.68)	70.00 (34.38)	0.442
8. Even if pain does not go away, it is possible to enjoy life.	78.46 (22.32)	75.58 (23.23)	0.005	66.55 (29.81)	81.45 (22.77)	0.102
9. Maintaining a normal life at school and during leisure time helps improve chronic pain.	66.18 (25.17)	68.91 (24.78)	0.015	69.00 (30.28)	72.00 (22.48)	0.397
10. Feeling sad or depressed make chronic pain worse.	65.58 (28.89)	69.47 (26.75)	0.003	73.82 (24.71)	61.82 (39.69)	0.203
11. A person with pain lasting several months or years can learn to manage their pain.	64.92 (25.82)	62.80 (26.01)	0.051	77.73 (30.07)	73.91 (32.50)	0.389
12. Experiencing pain for several months or years is a sign that something is wrong with the body.	81.51 (21.31)	83.51 (19.67)	0.029	80.73 (21.62)	88.36 (17.81)	0.188
13. A person with chronic pain (lasting several months or years) can remain optimistic about the future.	72.53 (26.08)	67.17 (27.97)	<0.001	68.18 (32.88)	72.82 (31.97)	0.370
14. Having pain for several months or years makes everything else in life worse.	36.14 (27.23)	40.18 (27.59)	0.002	38.82 (36.79)	35.82 (29.15)	0.417
15. Chronic pain can prevent making plans for the future.	37.58 (29.78)	40.77 (29.55)	0.016	39.82 (36.76)	26.09 (28.54)	0.170

cal care-seeking and endorsed more passive coping for prolonged pain. However, adolescents with JIA that did not experience pain were more optimistic about the future and life planning than those without JIA. This study underlines the importance of pain management among youth with juvenile idiopathic arthritis.

074 - A CASE OF REFRACTORY POLYARTERITIS NODOSA TREATED WITH HIGH-DOSE TOFACITINIB

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Introduction: Polyarteritis nodosa (PAN) is a rare systemic vasculitis that predominantly affects medium-sized arteries. It presents with diverse and potentially severe clinical manifestations. Management involves glucocorticoids (GCs), conventional immunosuppressive agents and biologic disease-modifying anti-rheumatic drugs. Janus kinase inhibitors (JAKi) have emerged as a promising therapeutic strategy in selected refractory cases, although supporting evidence remains limited to a small number of published case reports.

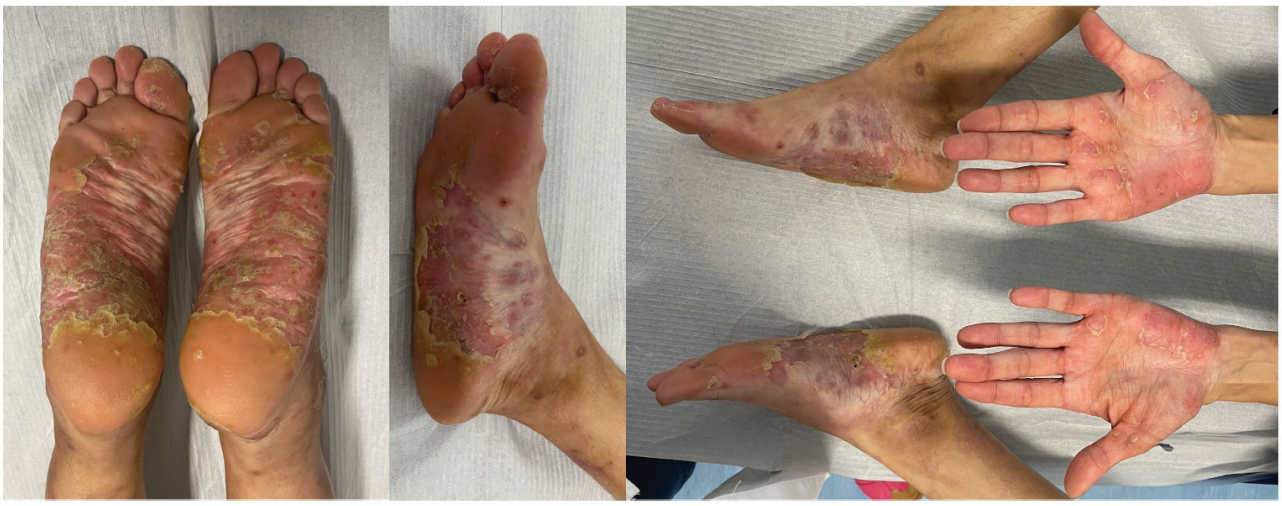
Case Presentation: A 42-year-old Brazilian woman presented with a 2-month history of persistent oligoarthritis of the ankles, paraesthesia, dysaesthesia, livedo racemosa and tender erythematoviolaceous nodules on the lower limbs. Laboratory tests showed elevated acute phase reactants, and ANA, ANCA, ACPA and RF were negative. Electrodiagnostic studies identified a mononeuritis multiplex of the saphenous and external popliteal sciatic left nerves. Skin biopsy confirmed vasculitis of medium-sized deep dermis arteries.

A diagnosis of PAN was assumed. Treatment with prednisolone combined with adjunctive immunosuppression (hydroxychloroquine, azathioprine, and/or methotrexate), achieved good clinical response without GC-free remission (minimum prednisolone effective dose of 10 mg qd). Therapy with IV infliximab (5 mg/Kg q8w) combined with methotrexate led to disease remission, allowing GC discontinuation over a 10-month period.

However, the patient developed infliximab-induced palmoplantar pustular psoriasis. (Figure 1) Infliximab was discontinued and topical GCs and photochemotherapy were started, with partial benefit. Following infliximab withdrawal, a PAN flare ensued with cutaneous and musculoskeletal relapse and elevated acute phase reactants (ESR 120 mm/h, CRP 2.45 mg/dL).

Low-dose prednisolone and tofacitinib 11 mg qd were started. Progressive improvement of the psoriatic lesions was observed until resolution over 6 months. Improvement of PAN lesions was observed but due to residual inflammatory symptoms (mild ankle arthritis, livedo racemosa) tofacitinib was increased to 22 mg qd, resulting in complete clinical remission and normalization of inflammatory markers. As of June 2025, 18 months after having started tofacitinib, the patient remains in remission under tofacitinib 22 mg qd combined with methotrexate.

Discussion: To date, seven cases of PAN treated with JAKi have been reported in the literature. Most refer



074 – Figure 1. Erythematous, desquamating, pruritic lesions of infliximab-induced palmoplantar psoriasis.

to cutaneous-limited PAN, as only two had systemic manifestations. In the majority of cases, JAKi were introduced after failure of multiple immunosuppressants and bDMARDs. Tofacitinib was the most frequently used JAKi, often at a dose of 5mg q12h, with good outcomes reported. The highest dose reported was 10mg q12h, and the longest follow-up was 12 months. (Table 1) Notably, in our case, the patient presented with neurological involvement and demonstrated a dose-dependent response to tofacitinib.

This case adds to the growing body of evidence supporting the potential role of JAKi in refractory PAN. It is, to our knowledge, the first report describing the successful treatment of PAN with neurological involvement using a JAKi. It illustrates the feasibility of optimizing tofacitinib dosing for enhanced efficacy without significant adverse events over an 18-month period.

077 - DIFFICULT-TO-MANAGE AXIAL SPONDYLOARTHRITIS: A TERTIARY CENTRE EXPERIENCE

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Background: In February 2025, the Assessment of SpondyloArthritis International Society (ASAS) published a consensus-based definition of difficult-to-manage (D2M) axial spondyloarthritis (axSpA), including a more stringent subset: treatment-refractory (TR) axSpA. However, data on the clinical profile of D2M/TR-axSpA remains limited.
Methods: We conducted a retrospective cohort study including adult axSpA patients followed in our rheumatology clinic and treated with at least one biological or targeted synthetic DMARD (b/tsDMARD). D2M and TR axSpA were defined per ASAS 2025 criteria. We estimated prevalence, assessed D2M domains, and explored clinical features associated with D2M using univariate logistic regression.
Results: Of 336 patients with axSpA, 140 had received at least one b/tsDMARD and were included. Median age was 46.5 years (range 24–75), 60.0% were female, and median follow-up was 5.8 years (IQR 3.2–9.1). HLA-B27 was positive in 59.2%, and 46.1% had peripheral involvement. At last visit, median ASDAS was 2.5 (IQR 1.8–3.1) and BASFI 3.2 (IQR 1.2–5.9). Fibromyalgia was present in 12.8%, obesity in 21.0%, and 35.2% were not in paid employment. Most patients (84.3%) had received only one b/tsDMARD.D2M-axSpA was identified in 8.6% (n = 12), and 50% of these also met criteria for TR-axSpA. Among D2M patients, the most frequent domain was insufficient disease control (61.4%), followed by problematic management (38.6%) and treatment failure (13.6%).

Univariate logistic regression showed associations with absence of paid work (p = .001), diagnostic delay (p = .014), inflammatory bowel disease (p = .049), fibromyalgia (p = .023), NSAID use (p = .040), glucocorticoid use (p = .018), use of IL-17i (p = .001), other bDMARDs (p = .011), tsDMARDs (p = .001), and higher ASDAS (p < .001), BASDAI (p < .001), BASFI (p = .021), CRP (p = .033), PtGA (p < .001), and PhGA (p = .004). Multivariate analysis was not performed due to the limited number of events. Table 1 summarises these findings.
Discussion/Conclusions: The prevalence of D2M-axSpA (8.6%) was comparable to the Dutch registry (9.7%). Underestimation may have occurred due to missing PtGA and PhGA in six patients with prior failure of at least two bDMARDs and limited iJAK access. Overestimation may reflect the influence of subjective variables, particularly PtGA. The most frequent domain was insufficient disease control, highlighting the weight of subjective assessments. MRI was only available in 4 of 19 with treatment failure, limiting that criterion's application. The role of comorbidities and patient-reported outcomes may deserve further exploration. Unlike the Dutch cohort, where psoriasis and smoking predominated, our data showed associations with fibromyalgia, IBD, diagnostic delay, absence of paid work, higher ASDAS, BASDAI, BASFI, CRP, PtGA, PhGA, and NSAID or glucocorticoid use. Use of IL-17i, tsDMARDs, and other bDMARDs was also more frequent. Absence of paid work was the only consistent factor across cohorts. Despite the small number of D2M cases, the consistency of observed associations suggests potential clinical relevance. Still, some findings should be interpreted with caution due to low event counts. These findings underscore the heterogeneity of D2M and the challenges in applying the ASAS definition, reinforcing the need for further multicentre validation.

081 - NOT JUST HIV: ONE PATIENT, TWO DIAGNOSES

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Background: In people living with HIV, symptoms such as fever, weight loss, cytopenias, and vasculitis are often attributed to opportunistic infections. However, similar features may occur in autoinflammatory syndromes. When symptoms persist despite appropriate antimicrobial therapy and exclusion of common infectious causes, rarer diagnoses should be considered.
Case Description: A 76-year-old man, who lived in West Africa in 1986, had a history of obstructive sleep apnea, type 2 diabetes, and chronic alcohol abuse. In 2021, he developed fatigue, anorexia, and lost 20 kg, which he attributed to alcohol cessation. In December 2023, he presented with fever, productive cough, dyspnea, constitutional symptoms, and purpuric ankle lesions. Laboratory and imaging revealed normocytic anemia (Hb 10.7 g/dL), white blood cell count at the lower limit of normal (3.82 × 10^9/L) with relative lymphopenia, elevated C-reactive protein (24.78 mg/dL), right perihilar consolidation, mediastinal lymphadenopathy, and splenomegaly. He was evaluated by Infectious Diseases and diagnosed with HIV-2 infection. The patient was admitted with pneumonia and started on empirical treatment for Pneumocystis jirovecii and antiretrovirals. Skin biopsy revealed leukocytoclastic vasculitis, which improved with topical corticosteroids and antibiotics. He was discharged on prednisolone 20 mg/day. Over the following year, he had multiple admissions for fever, recurrent arthritis (wrists and MCP joints), purpuric lesions, worsening cytopenias requiring red blood cell transfusions—particularly after tapering prednisolone below 10 mg/day—and persistently elevated inflammatory markers. High-dose corticosteroids (1 mg/kg/day) led to improvement. Due to persistent systemic inflammation with corticosteroid dependence, further investigation was undertaken. Rheumatology and Hematology evaluation, and bone marrow biopsy revealed myelodysplastic syndrome (MDS, IPSS-R 0) with Y chromosome deletion. No vacuolization was observed. Given the recurrent inflammation, cutaneous vasculitis, MDS, and corticosteroid dependence, an acquired autoinflammatory syndrome was suspected, namely VEXAS syndrome. UBA1 gene testing confirmed a somatic variant c.122T>C (p.Met41Thr; allelic fraction 91.5%). He was discharged on prednisolone 7.5 mg/day and weekly erythropoietin. Two months later, he still had arthritis and macrocytic anemia (Hb 7.9 g/dL). Prednisolone was increased to 10 mg/day and azacitidine was started (75 mg/m²/day for 7 days every 28 days). After two cycles, progressive improvement in hemoglobin and platelet count was observed (Hb 6.9 → 8.4 g/dL; platelets 55 → 99 × 10^9/L), with tapering to 7.5 mg/day and transfusion independence, suggesting a possible favorable response, although definitive conclusions regarding its efficacy may be premature.
Discussion and Conclusions: To our knowledge, this is the first reported case of VEXAS in an HIV-positive patient. This case highlights the diagnostic challenge of VEXAS syndrome in people living with HIV, due to overlapping features with infectious complications. The persistence of inflammation, fever, cytopenias, cutaneous vasculitis and rheumatic features despite appropriate treatment raised suspicion. Multidisciplinary collaboration enabled a timely diagnosis.

082 - DRUG-INDUCED SYSTEMIC LUPUS ERYTHEMATOSUS ASSOCIATED WITH TUMOR NECROSIS FACTOR-ALPHA INHIBITORS: A RETROSPECTIVE STUDY USING REAL-WORLD DATA FROM THE EUDRAVIGILANCE DATABASE

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Background: Tumor necrosis factor-alpha inhibitors (iTNF-α) are essential therapeutic agents for the management of diverse medical conditions, including inflammatory rheumatic and intestinal diseases. Suspected adverse reactions (SARs) associated with these treatments have been reported to international pharmacovigilance databases, such as the European EudraVigilance.

iTNF-α-induced systemic lupus erythematosus (iTNF-α-induced SLE) is a well-recognized but rare adverse event associated with these drugs. Although challenging to quantify, the incidence of iTNF-α-induced SLE is estimated to range between 0.10% and 0.22%, with slightly higher rates reported for infliximab (INF), the first iTNF-α introduced into clinical practice [1,2]. Nonetheless, data on the true incidence of iTNF-α-induced SLE remains scarce [3], especially for newer agents.
Objective: To compare the occurrence of iTNF-α-induced SLE in patients medicated with adalimumab

085 – TABLE 1. Characteristics of Individual Case Safety Reports in the EudraVigilance database, containing a suspected adverse reaction of drug-induced systemic lupus erythematosus from 1 January, 2020 to 31 December, 2024, attributed to adalimumab, certolizumab, etanercept, golimumab, and infliximab. a. Each case might meet more than one criterion.

Characteristic	Adalimumab (N, %)	Infliximab (N, %)	Etanercept (N, %)	Certolizumab (N, %)	Golimumab (N, %)
Year of Reporting					
2024	11 (35.5)	13 (22.9)	0 (0.0)	1(16.7)	0 (0.0)
2023	3 (9.7)	5 (8.5)	2 (15.4)	0 (0.0)	1 (25.0)
2022	7 (22.6)	16 (27.1)	4 (30.8)	1(16.7)	1 (25.0)
2021	5 (16.1)	10 (16.9)	5 (38.5)	2 (33.3)	1 (25.0)
2020	5 (16.1)	15 (25.4)	2 (15.4)	2 (33.3)	1 (25.0)
Sex					
Female	26 (83.9)	36 (61.0)	11 (84.6)	5 (83.3)	4 (100.0)
Male	4 (12.9)	21 (35.6)	2 (15.4)	0 (0.0)	0 (0.0)
Not specified	1 (3.2)	2 (3.4)	0 (0.0)	1 (16.7)	0 (0.0)
Age range					
12 to 17 years	0 (0.0)	4 (6.8)	0 (0.0)	0 (0.0)	0 (0.0)
18 to 64 years	26 (83.9)	44 (74.6)	9 (69.2)	4 (66.7)	4 (100.0)
65 to 85 years	3 (9.7)	2 (3.4)	4 (30.8)	1 (16.7)	0 (0.0)
Not specified	2 (6.5)	9 (15.3)	0 (0.0)	1 (16.7)	0 (0.0)
Suspected adverse reaction considered serious	(100.0)	54 (91.5)	12 (92.3)	6 (100.0)	3 (75.0)
Seriousness criteriaa					
Resulting in medically important conditions	28 (90.3)	43 (72.9)	9 (69.2)	6 (100.0)	3 (75.0)
Requiring or prolonging hospitalization	6 (19.4)	13 (22.0)	3 (23.1)	0 (0.0)	0 (0.0)
Resulting in disability/incapacity	0 (0.0)	1 (1.7)	0 (0.0)	0 (0.0)	0 (0.0)
Life threatening	0 (0.0)	1 (1.7)	0 (0.0)	0 (0.0)	0 (0.0)
Reaction outcome at time of report					
Not recovered	5 (16.1)	6 (10.2)	2 (15.4)	0 (0.0)	2 (50.0)
Recovering	6 (19.4)	13 (22.0)	2 (15.4)	0 (0.0)	0 (0.0)
Recovered	5 (16.1)	23 (39.0)	6 (46.2)	2 (33.3)	2 (50.0)
Recovered with sequelae	0 (0.0)	1 (1.7)	0 (0.0)	0 (0.0)	0 (0.0)
Unknown	15 (48.4)	6 (10.2)	3 (23.1)	4 (66.7)	0 (0.0)
Indication for drug use					
Rheumatoid arthritis	9 (29.0)	6 (10.2)	5 (38.5)	1 (16.7)	1 (25.0)
Spondylarthritis	4 (12.9)	5 (8.5)	2 (15.4)	1 (16.7)	1 (25.0)
Psoriatic Arthritis	2 (6.5)	2 (3.4)	2 (15.4)	1 (16.7)	2 (50.0)
Inflammatory Intestinal Diseases	6 (19.4)	32 (54.2)	0 (0.0)	0 (0.0)	0 (0.0)
Ocular Diseases	2 (6.5)	1 (1.7)	0 (0.0)	0 (0.0)	0 (0.0)
Dermatological Diseases	7 (22.6)	3 (5.1)	1 (7.7)	1 (16.7)	0 (0.0)
Other Indications	0 (0.0)	4 (6.8)	2 (15.4)	0 (0.0)	0 (0.0)
Unknown	1 (3.2)	6 (10.2)	1 (7.7)	2 (33.3)	0 (0.0)

(ADA), certolizumab (CER), etanercept (ETA), golimumab (GOL), and INF, by analyzing real-world data from the European EudraVigilance database.

Methods: A retrospective analysis was conducted using the public version of the EudraVigilance database, encompassing reports of suspected adverse reactions (SARs) associated with the administration of ADA, CER, ETA, GOL, and INF between January 2020 and December 2024. This study included only SARs reported by healthcare professionals within the European Economic Area, excluding duplicate cases and those potentially attributable to other medications. Data on patient demographics, the presence of seriousness criteria, outcomes at the time of reporting, and the indications for each iTNF-α use were collected, followed by a descriptive analysis. Finally, the Reporting Odds Ratio (ROR) was calculated for each drug in comparison with the others.

Results: During the specified study period, a total of 161,868 SARs concerning iTNF-α were reported to the EudraVigilance database, including 45,778 related to ADA, 15,836 to CER, 30,604 to ETA, 11,229 to GOL, and 58,421 to INF. Of these, 113 concerned iTNF-α-induced SLE and met our inclusion criteria, comprising 31 related to ADA, 6 to CER, 13 to ETA, 4 to GOL, and 59 to INF.

For all treatments, most patients were females aged between 18 and 64 years (Table 1). Common indica-

tions for iTNF-α use were rheumatoid arthritis and inflammatory intestinal diseases. Most analyzed SARs were classified as serious by the reporter, with hospitalization and the presence of medically important conditions often cited as criteria for seriousness in all groups. At the time of SAR reporting, most patients had either recovered or were recovering.

Concerning the risk of iTNF-α-induced SLE, the analysis based on the ROR indicated a higher risk associated with INF compared to other iTNF-α [ROR: 1.94; 95% CI (1.34–2.80)]. The lowest risk was observed with ETA [ROR: 0.56; 95% CI (0.31–0.99)]. The risks associated with ADA [ROR: 0.96; 95% CI (0.63–1.45)], CER [ROR: 0.52; 95% CI (0.23–1.18)], and GOL [ROR: 0.49; 95% CI (0.18–1.54)] were comparable among these drugs.

Conclusion: In our analysis of real-world data from the EudraVigilance database, INF was associated with a higher risk of iTNF-α-induced SLE, whereas ETA exhibited the lowest risk for this adverse event. These findings are consistent with current scientific evidence derived from other cohorts. iTNF-α-induced SLE accounted for only 0.04% to 0.10% of all reported SARs, further supporting its classification as a rare adverse event.

090 - TERIPARATIDE IN OSTEOPOROSIS TREAT-TO-TARGET APPROACH

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Introduction: A Treat-to-Target (T2T) approach has recently been proposed in osteoporosis (OP) to improve outcomes through individualized care and defined therapeutic goals. Bone mineral density (BMD) remains the most appropriate treatment target due to its accessibility and correlation with fracture risk. Teriparatide, an osteoanabolic agent, promotes rapid and greater BMD gains and enhances fracture risk reduction, particularly when followed by an antiresorptive agent.

Objectives: To evaluate the efficacy of teriparatide in achieving a defined treatment target in a real-world OP cohort.

Materials and methods: This retrospective observational study included patients with OP treated with teriparatide for 18-24 months at our rheumatology center, with available baseline and post-treatment dual-energy X-ray absorptiometry (DXA) scans. Treatment response was defined as achieving T-scores >–2.5 standard deviations (SD) at the lumbar spine (LS), femoral neck (FN) and total hip (TH), as proposed by the American Society for Bone and Mineral Research and the Bone Health & Osteoporosis Foundation in 2024. Wilcoxon's test compared baseline and post-treatment DXA values, while McNemar's exact test assessed response rates, with $p<0.05$ considered significant.

Results: A total of 15 patients were included. Most (93.3%, n=14) were female, with a median age at diagnosis of 65 years (range 32-84). Prior fragility fractures were reported in 86.7% (n=13), mostly vertebral (66.7%). Median BMI was 23.52kg/m² (range 20.1-31.1) and 20% (n=3) were smokers. Most women (92.9%, n=13/14) were postmenopausal at diagnosis; one had early menopause (5 missing data). Teriparatide was initiated a median of 6 months after diagnosis (range 0-189), with 66.7% (n=10) previously treated with oral bisphosphonates. All received calcium and/or vitamin D supplementation. Two cases (13.3%) of transient hypercalcemia occurred without treatment interruption. One (6.7%) sustained a femoral fragility fracture at 12 months. All completed 24 months of therapy, except one who discontinued at 20 months due to pregnancy. Most (86.7%, n=13) transitioned to denosumab. One no longer met OP criteria post-treatment, and another was lost to follow-up.

In patients with baseline T-scores ≤–2.5, significant T-score gains were observed at the LS (+0.60 SD, p=0.036) and FN (+0.65 SD, p=0.017). TH BMD increased significantly (+6.4%, p=0.043), though the T-score improvement did not reach statistical significance. Not all patients exceeded the least significant change (LSC = 0.03), and only T-score data were available in some. After treatment, 35.7% (n=5/14) had T-scores >-2.5 at the LS, 40.0% (n=6/15) at the FN and 69.23% (n=9/13) at the TH; 4 patients (26.7%) met the target at all three sites. Among those with baseline T-scores ≤–2.5, response rates were 18.2% (LS) and 20.0% (FN and TH), above the LSC, but not statistically significant. One patient showed a non-significant decline in FN T-score.

Discussion/Conclusions: In this real-world cohort, teriparatide led to significant T-scores improvements at the LS and FN, with a low fracture incidence. However, the proportion of patients achieving the predefined T2T goal (T-score >–2.5) was modest, aligning with findings from a TOWER trial analysis (21.9% for LS and 14.5% for FN at 72 weeks). These results highlight the challenges of OP management and need for individualized treatment targets. Larger studies are needed to validate these results, assess long-term sequential therapy outcomes and optimize treatment strategies for fracture prevention.

090 – TABLE 1. Baseline and post-teriparatide therapy DXA parameters and treatment response rates according to anatomical site

		Baseline DXA BMD g/cm3 (range)	Post-treatment DXA BMD g/cm3 (range)	BMD difference post-treatment (percentage of improvement)	*p*-value[1]	Baseline DXA T-score SD (range)	Post-treatment DXA T-score SD (range)	T-score SD difference post-treatment (percentage of improvement)	*p*-value[1]	Post-treatment DXA T-score SD		Percentage of patients with post-treatment T-score >–2.5 SD by location	Percentage of patients with post-treatment T-score >–2.5 SD in all three sites	Percentage of patients with baseline T-score ≤–2.5 SD achieving T-score >–2.5 SD	*p*-value[2]
										≤–2.5 SD	>-2.5 SD				
Lumbar spine	≤–2.5 SD	0.690 (0.470 to 0.869) (n=10)	0.796 (0.618 to 0.932) (n=10)	+0.106 (↑15.4%)	0.051	-3.70 (-5.90 to -2.80) (n=11)	-3.10 (-4.70 to -2.30) (n=11)	+0.60 (↑ 16.2%)	0.036	9 (n=11)	2 (n=11)*	35.7% (n=5/14)	26.7% (n=4/15)	18.2% (n=2/11)	0.5
	>-2.5 SD	1.182 (1.090 to 1.396) (n=3)	1.149 (1.070 to 1.438) (n=3)	-0.033 (↓3.3%)	1.000	-0.10 (-0.80 to 1.30) (n=3)	-0.40 (-1.00 to 1.44) (n=3)	-0.30 (↓300%)	1.000	0 (n=3)	3 (n=3)			-	
Femoral neck	≤–2.5 SD	0.567 (0.445 to 0.663) (n=9)	0.631 (0.553 to 0.719) (n=9)	+0.064 (↑11.3%)	0.11	-3.30 (-4.10 to -2.60) (n=10)	-2.65 (-3.50 to -1.80) (n=10)	+0.65 (↑19.7%)	0.017	8 (n=10)	2 (n=10)*	40.0% (n=6/15)		20.0% (n=2/10)	1.0
	>-2.5 SD	0.789 (0.691 to 0.919) (n=5)	0.787 (0.682 to 0.868) (n=5)	-0.002 (↓0.25%)	0.588	-1.60 (-2.40 to -1.20) (n=5)	-1.60 (-2.50 to -1.40) (n=5)	0	0.496	1 (n=5)**	4 (n=5)			-	
Total hip	≤–2.5 SD	0.572 (0.427 to 0.635) (n=5)	0.593 (0.496 to 0.712) (n=5)	+0.021 (↑6.4%)	0.043	-3.40 (-4.80 to -3.00) (n=5)	-3.30 (-4.20 to -2.40) (n=5)	+0.1 (↑2.9%)	0.063	4 (n=5)	1 (n=5)*	69.2% (n=9/13)		20.0% (n=1/5)	1.0
	>-2.5 SD	0.751 (0.686 to 0.956) (n=8)	0.787 (0.685 to 0.969) (n=8)	0.036 (↑ 4.8%)	0.161	-2.00 (-2.40 to -1.00) (n=8)	-1.75 (-2.10 to -0.90) (n=8)	+0.25 (↑12.5%)	0.175	0 (n=8)	8 (n=8)			-	

BMD - Bone Mineral Density; DXA - Dual-energy X-ray Absorptiometry
[1] Wilcoxon's test; [2] McNemar's exact test; A *p*-value of <0.05 was considered significant
* variation higher than the LSC of 0.03; ** variation lower than the LSC of 0.03

113 - CAPILLAROSCOPIC FINDINGS IN PRIMARY RAYNAUD'S PHENOMENON ACROSS AGE GROUPS: DATA FROM CAPRAS REGISTRY

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Background: Primary Raynaud's phenomenon (PRP) is a frequent condition marked by transient vasospasm of the digital arteries, usually in response to cold or stress, without associated disease. Nailfold videocapillaroscopy (NVC) is widely used to detect microvascular changes and screen for connective tissue diseases. However, its role and interpretation in PRP—especially across age groups—remain unclear. Age-related physiological changes may influence findings, potentially mimicking or masking pathological patterns. Understanding these differences is essential to avoid misdiagnosis.

Methods: We conducted a retrospective observational study using data from the CAPillaroscopy Registry Almada-Seixal (CAPRAS), which includes NVCs performed at our center from 2014 to 2024. Examinations were performed using a digital microscope (Dino-Lite CapillaryScope 200 Pro). Of 703 patients in the registry, we selected 118 who met PRP criteria: Raynaud's phenomenon, no autoimmune disease, and negative ANA, ENA, and antiphospholipid antibodies.

Patients were grouped as children (≤18 years), adults (19–64), and older adults (≥65). Descriptive statistics summarized each group. Categorical variables were compared using chi-squared or Fisher's test; continuous variables via Kruskal-Wallis ($p<0.05$).

Results: Among 118 patients, 6 were children, 89 adults, and 23 older adults. Most were female and Caucasian. Mean age at NVC was 16.5 (±1.2) in children, 44.4 (±11.5) in adults, and 76.5 (±11.2) in older adults.

Capillaroscopic abnormalities were observed across all age groups. In children, haemorrhages were found

113 - TABLE 1- Clinical and Capillaroscopic Characteristics of Primary Raynaud's Phenomenon across age groups.

	All patients (n= 118)	Children (n=6)	Adults (n=89)	Older adult (n=23)	p-value
Female sex, n (%)	98 (83.1)	5 (83.3)	78 (87.6)	15 (65.2)	0.036
Caucasian, n (%)	111 (94.1)	4(66.7)	85 (95.5)	22 (95.7)	0.052
Age at NVC, mean (SD)	49.2 (17.6)	16.5 (1.2)	45.1 (11.5)	73.8 (11.2)	<0.001
Hypertension, n (%)	35 (29.7)	0 (0.0)	21(23.6)	14 (60.9)	<0.001
Hypothyroidism, n (%)	7 (5.9)	0 (0.0)	7 (7.7)	0 (0.0)	0.227
Peripheral compression syndromes, n (%)	17 (14.4)	0 (0.0)	15 (16.9)	2 (8.7)	0.451
Nailfold capillaroscopy findings, n (%)					
Haemorrhages	53 (42.3)	5 (83.3)	38 (42.7)	10 (43.5)	0.317
Dilatations (20-50 µm)	30 (25.4)	1 (16.7)	22 (24.7)	7 (30.4)	0.351
Giant capillaries (>50 µm)	3 (2.5)	0 (0.0)	2 (2.2)	1 (4.3)	0.271
Abnormal morphology	21 (17.8)	2 (33.3)	13 (14.6)	6 (26.1)	0.082
Neoangiogenesis	8 (6.8)	0 (0.0)	5 (5.6)	3 (13)	0.216
Decreased density (<7 capillaries/mm)	18 (15.3)	0 (0.0)	15 (16.9)	3 (13.0)	0.326
Nailfold capillaroscopy pattern, n (%)					
Normal pattern	42 (35.6)	0 (0.0)	36 (40.4)	6 (26.1)	0.078
Non-specific abnormalities	67 (56.8)	6 (100)	45 (50.6)	16 (69.6)	0.023
Early scleroderma pattern	1 (0.8)	0 (0.0)	1 (1.1)	0 (0.0)	1.000
Active scleroderma pattern	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1.000
Late scleroderma pattern	1 (0.8)	0 (0.0)	0 (0.0)	1 (4.3)	0.246
Scleroderma-like pattern	5 (4.2)	0 (0.0)	5 (5.6)	0 (0.0)	0.679

n, number. Number of missing data per variable – haemorrhages 2.5% (n=3); dilatations 2.5% (n=3); giant capillaries 2.5% (n=3); abnormal morphology 2.5% (n=3); decreased density 2.5% (n=3); capillaroscopy pattern 1.7% (n=2).

in 83.3%, abnormal morphology in 33.3%, and dilations in 16.7%, with no megacapillaries, density reduction, or neoangiogenesis. In adults, haemorrhages (42.7%) and abnormal morphology (14.6%) predominated, followed by dilations (25.8%) and reduced density (14.6%). Older adults showed a similar pattern: haemorrhages in 43.5%, abnormal morphology in 26.1%, and reduced density in 21.7%.

Although group differences weren't statistically significant for most features, the high rate of haemorrhages and morphological changes in children calls for cautious interpretation.

The most frequent NVC pattern was non-specific abnormalities: 100% of children, 50.6% of adults, and 69.6% of older adults. Scleroderma-specific patterns were rare but observed in adults and older adults—early, late, or scleroderma-like. None were seen in children.

Discussion and Conclusion: In this PRP cohort, non-specific capillaroscopic abnormalities were the most common pattern across all ages. While adults and older adults showed more varied findings, the presence of haemorrhages and morphology changes in children—without autoimmune features—likely reflects physiological variability, trauma, or artifacts rather than microangiopathy.

Scleroderma-specific patterns were absent in children and rare in adults, reinforcing their value in distinguishing primary from secondary Raynaud's. However, isolated findings should prompt careful clinical follow-up.

These findings highlight the need for age-adapted interpretation of NVC in clinical settings, especially in pediatrics. Further studies with larger pediatric samples are warranted.

136 - PARVOVIRUS B19 INFECTION PRESENTING AS POLYARTHRITIS: A NATIONWIDE CLINICAL AND EPIDEMIOLOGICAL STUDY

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Introduction: Viral arthritis represents 1% of acute polyarthritis cases and may be caused by agents like parvovirus B19 (B19V), Epstein-Barr virus, and hepatitis B/C viruses. B19V usually presents with facial rash and fever; joint involvement occurs in around 60% of cases. Although usually self-limited with spontaneous resolution within weeks, it can rarely become chronic. Diagnosis relies on clinical features, B19V serology, and exclusion of other infectious or non-infectious causes. Between March-May 2024, several European countries reported a rise in B19V cases. Building on our previous single-center study (5 cases, 2024), we conducted a multicenter study to further explore the clinical presentation and outcomes of B19V-related polyarthritis.

Materials and methods: This multicenter retrospective study included patients from participating centers with acute-onset arthritis, positive B19V IgM/IgG, and/or relevant epidemiological risk factors. Patients with previous systemic rheumatic diseases were excluded. Cases were identified between March-May 2024. Data was collected from electronic medical records. The study was approved by an institutional ethics committee.

Results: Twenty-eight patients (25 women, 3 men; mean age 44±11.4 years) were included (Table 1). Sixteen (66.1%) had epidemiological risk factors (e.g., childcare work). Twenty-six (92.9%) patients presented with acute, additive, symmetrical inflammatory polyarthralgia worsening over a week (7,5±6,6 days). Fever was reported in 10 (35.7%), and 11 (39.3%) developed typical rash. Additional symptoms (14,3%) included odynophagia, hand pitting edema, and cryoglobulinemic vasculitis. Physical examination showed arthritis of small joints (wrists, hands and feet) in 27 (96.4%) and large joints (knees, ankles, elbows) in 15 (53.6%). All patients had upper limb involvement, while 71.4% had lower limb involvement. Axial inflammatory pain, a rare presentation, was reported in 4. A complete

136 – TABLE 1. Clinical and demographic characteristics of patients

Variable	Mean (±SD) / n (%)	Missings (n)
Total of patients, n (%)	28 (100)	
Women, n (%)	25 (89,3)	0
Age, mean (±SD)	44,0 (± 11,4)	0
Epidemiology		3
Infection in a household contact, n (%)	13 (46,4)	
High-risk occupation, n (%)	3 (19,7)	
No risk factors, n (%)	9 (32,1)	
Clinical presentation		
Fever, n (%)	10 (35,7)	0
Rash, n (%)	11 (39,3)	0
Polyarthritis, n (%)	26 (92,9)*	0
Oligoarthritis, n (%)	2 (7,1)	0
Odynophagia, n (%)	2 (7,1)	0
Hand pitting edema, n (%)	1 (3,6)	0
Cryoglobulinemic vasculitis, n (%)	1 (3,6)	0
Laboratory findings		
Anemia [rv < 12.0 g/dL], n (%)	8 (28,6)	1
Raised AST [rv > 33 U/L], n (%)	8 (28,6)	5
Raised ALT [rv > 32 U/L], n (%)	9 (32,1)	5
Elevated CRP [rv > 0.5 mg/dL], n (%)	17 (60,7)	1
Elevated ESR [rv > 20 mm/h], n (%)	17 (60,7)	2
Immunology		
Positive Parvovirus IgM [rv ≥1.1 UI/mL], n (%)	25 (89,3)	0
Positive Parvovirus IgG [rv > 2.5 UI/mL], n (%)	25 (89,3)	3
IgM and IgG positive	22 (78,6)	3
Positive ANA, n (%)	9 (32,1)	3
Positive CCP [rv > 5 UA/mL], n (%)	0 (0)	6
Positive RF [rv > 15 UI/mL], n (%)	3 (19,7)	5
Clinical course		
Time to full clinical presentation in days, mean (±SD)	7,5 (±6,6)	0
Complete resolution, n (%)	24 (85,7)	0
Time to clinical resolution in days, mean (±SD)	34 (±47,0)	0
Treatment during the acute phase		0
NSAID, n (%)	11 (39,3)	
Metilprednisolone, n (%)	1 (3,6)	
Prednisolone, n (%)	17 (60,7)	
Treatment in chronic presentations		0
cDMARD, n (%)	3 (19,7)	
bDMARD, n (%)	1 (3,6)	

ANA - antinuclear antibody. ALT - alanine transaminase. AST - aspartate transaminase. bDMARD - biotechnological disease-modifying antirheumatic drugs. CCP - Anti–citrullinated protein antibody. cDMARD - conventional disease-modifying antirheumatic drugs. CRP - C-reactive protein. ESR - erythrocyte sedimentation rate. Ig G - Immunoglobulin G. IgM - Immunoglobulin M. n - number of patients. NSAID - nonsteroidal anti-inflammatory drugs. RF - rheumatoid factor. rv – reference value.

workup excluded other etiologies. Baseline laboratory findings included anemia (28.6%), elevated transaminases (28.6–32.1%), and raised C-reactive protein and erythrocyte sedimentation rate (60.7%). Immunological testing revealed positive antinuclear antibodies (ANA) in 32.1%, rheumatoid factor in 19.7%, and all anti-citrullinated protein antibodies were negative. B19V serology (IgM and/or IgG) was positive in 89.3%; diagnosis in seronegative cases relied on clinical and epidemiological data. Regarding treatment, 39.3% received nonsteroidal anti-inflammatory drugs (NSAIDs), 60.7% systemic corticosteroids (prednisolone 10-40 mg/day) and one patient required intravenous methylprednisolone (125 mg) due to severe polyarthritis. Remission was reached in 24 (85.7%) after a mean duration of 34±47.0 days, allowing for corticosteroid tapering. Four patients relapsed, suggesting progression to a chronic inflammatory state. One patient maintained symptom control with intermittent NSAID; three started conventional disease-modifying antirheumatic drugs (csDMARDs), and one of them subsequently required additional therapy with a tumor necrosis factor (TNF) inhibitor.

Conclusion: To our knowledge, this is the largest multicenter study on B19V polyarthritis. B19V infection may present as acute, symmetric polyarthritis mimicking early rheumatoid arthritis. While usually self-limited, some cases progress to chronic disease requiring immunosuppressive therapy. The unusually high case number in a short time suggests a possible epidemiological shift, highlighting the need for clinical awareness and ongoing surveillance.

138 - THE ROLE OF SERUM URIC ACID IN PSORIATIC ARTHRITIS: CAN IT BE USED AS A DISEASE ACTIVITY MARKER?

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Introduction: Psoriatic Arthritis (PsA) is associated with a high prevalence of cardiovascular risk factors, including hyperuricemia, affecting 20-30% of these patients. Although these are strongly linked, the underlying mechanisms remain unclear. Some studies aimed to evaluate the effect of serum uric acid (SUA) levels and the variations of acute-phase reactants (APR), clinical presentation, disease activity and bDMARDs response in PsA patients, raising debate on treating asymptomatic hyperuricemia in this population. However, findings have been controversial. Some studies have shown a decrease in SUA levels with disease activity control and the use of DMARDs, while others found no link or even SUA increase.

Objectives: To assess the role of baseline SUA and their relationship with clinical presentation, severity and disease activity in Portuguese PsA patients, using real-world data.

Methods: We conducted a retrospective observational study including all adult PsA patients from our center registered in the Reuma.pt. Baseline SUA was defined as the last value before bDMARD. Patients lacking SUA records in the year before bDMARD or on hypouricemic therapy were excluded. Patients were divided based on baseline SUA: normouricemia (<6mg/dL) and hyperuricemia (≥6mg/dL). We compared both regarding sociodemographic, clinical characteristics and disease activity indices. Continuous variables were summarized as mean±SD or median±IQR, and categorical variables as frequencies. Comparisons used chi-squared, t-test, or Mann-Whitney U, with statistical significance set as p<0,05.

Results: A total of 95 patients were included. Mean baseline SUA was 4.87±1.48mg/dL. The normouricemia group (n=73; 76.8%) had a mean age of 54.6±11.7 years and median disease duration of 12.1 (IQR 11.75) years; most were female (52.1%) and on anti-TNFα therapy (75.3%). The hyperuricemia group (n=22; 23.2%) was older (61.6±11.95 years; p=0.015) and had a shorter disease duration (8.94 years; IQR 6.35; p=0.056); most were male (68.2%) and on anti-TNFα therapy (81.8%).

No differences were found between groups in predominant involvement (p=0.917), both showing mainly peripheral manifestation. SUA variation (ΔSUA = last – baseline) differed between groups (p=0.018): it was positive in the normouricemia group (0.59±1.06), indicating increased levels, and negative in the hyperuricemia group (-0.35±1.65), indicating a decrease. APR showed no statistically significant differences at baseline [CRP (p = 0.122); ESR (p = 0.698)], at the last appointment [CRP (p = 0.911); ESR (p = 0.639)] or in the changes in their levels [ΔCRP (p= 0,246); ΔERS (p= 0,622)]. Disease activity scores also showed no differences at baseline [DAPSA (p = 0.952); DAS28-4V (p = 0.703); TJC (p = 0.369); SJC (p = 0.621)], at the last appointment [DAPSA (p = 0.342); DAS28-4V (p = 0.650); TJC (p = 0.609); SJC (p = 0.309)] or in the changes in their levels [ΔDAPSA (p = 0,840); ΔDAS28-4V (p = 0,567); ΔTJC (p = 0,754); ΔSJC (p = 0,761)].

Conclusion: In our study, hyperuricemia prevalence in PsA was 23%, with affected patients being older, which is consistent with the literature. We found no relationship between baseline SUA levels and clinical presentation, disease activity, either at baseline or at

138 – TABLE 1. Comparative analysis of disease activity indices between normouricemia and hyperuricemia groups.

	Normouricemia N = 73	Hyperuricemia N = 22	Total N = 95	P - value
CRP, mg/L, Median (IQR)				
Baseline	11,40 (17,45)	7,15 (17,80)	10,55 (17,25)	*0,122*
At last appointment	1,85 (3,22)	1,90 (2,90)	1,90 (3,05)	*0,911*
ΔCRP	-9,00 (19,80)	-3,75 (14,45)	-8,40 (17,45)	*0,246*
ERS, mm/h, Median (IQR)				
Baseline	28,00 (43,00)	23,50 (35,00)	24,00 (42,00)	*0,698*
At last appointment	9,00 (17,75)	12,50 (18,00)	9,00 (17,25)	*0,639*
ΔERS	-11,00 (28,50)	-6,00 (17,50)	-9,00 (26,25)	*0,622*
DAPSA, Mean (SD)				
Baseline	32,76 (13,54)	33,94 (14,64)	33,07 (13,73)	*0,952*
At last appointment	12,71 (10,86)	17,56 (17,34)	13,89 (12,80)	*0,342*
ΔDAPSA	-20,66 (13,25)	-19,62 (15,01)	-20,39 (13,59)	*0,840*
DAS28-4V, Mean (SD)				
Baseline	4,92 (1,48)	4,78 (1,31)	4,88 (1,43)	*0,703*
At last appointment	2,73 (1,30)	3,00 (1,40)	2,81 (1,32)	*0,650*
ΔDAS28-4V	-2,13 (1,55)	-1,88 (1,39)	-2,06 (1,50)	*0,567*
TJC, Median (IQR)				
Baseline	9,00 (14,00)	13,00 (15,00)	10,00 (14,00)	*0,369*
At last appointment	1,00 (5,79)	1,50 (4,25)	1,00 (4,00)	*0,609*
ΔTJC	-5,50 (10,75)	-11,00 (11,50)	-6,00 (11,50)	*0,754*
SJC, Median (IQR)				
Baseline	5,50 (7,00)	6,00 (10,00)	6,00 (8,00)	*0,621*
At last appointment	0,00 (1,00)	0,00 (0,25)	0,00 (1,00)	*0,309*
ΔSJC	-4,00 (7,00)	-5,00 (10,00)	-4,00 (7,75)	*0,761*

CRP: C-Reactive Protein; ΔCRP = CRP last appointment – CRP baseline; ERS: Erythrocyte Sedimentation Rate; ΔERS = ERS last appointment – ERS baseline; DAPSA: Disease Activity Index for Psoriatic Arthritis; ΔDAPSA = DAPSA last appointment – DAPSA baseline; DAS28-4V: 28-joint Disease Activity Score with 4 variables; ΔDAS28-4V= DAS28-4Vlast appointment – DAS28-4Vbaseline; TJC: Tenderness Joint Count; ΔTJC= TJC last appointment – TJC baseline; SJC: Swollen Joint Count; ΔSJC= SJC last appointment – SJC baseline.

the last appointment, nor an association with any of the bDMARD used. SUA levels can be influenced by many factors; however, our results suggest that the SUA levels have an independent course with the PsA activity. This is a controversial topic, and further large-scale studies are needed to validate these results.

142 - PARES PROJECT (PATIENTS SUPPORT TO RHEUMATOLOGY IN SPECIALTY AND SOCIETY) – "STUDENTS' PERCEPTIONS OF THE LEARNING MODEL INVOLVING PATIENT EDUCATORS – A PILOT STUDY"

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Background: Patients hold unique experiential knowledge and expertise that physicians are unable to provide (1). Grounded in the principles of Public and Patient Involvement, active patient engagement is essential to promote patient-centered care. Integrating this expertise into medical education not only enhances future medical doctors' patient-centered skills, but also improves learner engagement, reduces clinical anxiety, and fosters greater confidence and respect in patient interactions.

Objectives: Evaluate students' perceptions about learning from a Patient-Professor with a rheumatic and musculoskeletal disease, the benefits and points for improvement of this model.

Methods: We conducted a quasi-experimental study using a group (N=45) of 4th-year medical students

(N=72). Participants engaged in 1 hour small-group sessions (n≈18) co-facilitated by a Patient-Professor and a Rheumatologist (Doctor-Professor). These interactive sessions simulated clinical consultations while addressing rheumatology diagnostics, management, physical examination and patient perspectives. Post-intervention perceptions were assessed through a mixed-methods survey.

Results: Among 45 respondents (80% female, mean age 22.4 years), Likert-scale responses (1= Totally Disagree, 5= Totally Agree) demonstrated strongly positive perceptions of the Patient-Professor model: 80% agreed it enhanced disease understanding, 73.3% found it useful for improving communication skills, and 100% recognized its clinical relevance. Notably, 77.8% reported increased empathy, while 64.4% and 53.3% noted improvements in history-taking and physical examination confidence, respectively. Diagnostic confidence improved for 66.7% of participants, though some acknowledged limitations. Most respondents (93.3%) considered these sessions equally valuable to traditional teaching, and reported particular benefits for empathy development (86.7%), communication skills (68.9%), and clinical competencies (57.8%).

Additionally, qualitative analysis identified two main topics: (1) the educational advantages of the Patient-Professor model, and (2) potential areas for enhancement. Participants highlighted significant benefits in soft skills development and clinical reasoning, particularly emphasising the dynamic learning environment and authentic patient perspective. The model's relaxed atmosphere was noted to facilitate greater clinical exposure and engagement. Participants provided concrete recommendations to strengthen the model, with clear calls for: (1) content diversification – including a wider variety of Patient-Professors (to represent different clinical experiences) and broader disease coverage, and (2) logistical improvements, specifically, smaller group sizes, extended session durations and the inclusion of visual support.

Conclusions: The Patient-Professor sessions were consistently rated as both educationally valuable and highly engaging for fourth-year medical students. Quantitative results revealed overwhelming recognition of clinical relevance while qualitative feedback highlighted unique benefits in soft skills development and clinical reasoning. Collectively, these findings establish the Patient-Professor model as an innovative and highly effective strategy for clinical learning which make it a compelling candidate for formal adoption into medical curricula.

Keywords: patient education; patient-professor; education model

157 - EXPLORING THE LINK: SYSTEMIC SCLEROSIS AND PERIPHERAL ARTERIAL DISEASE – DATA FROM A TERTIARY CENTER

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Introduction: Systemic sclerosis (SSc) is a connective tissue disease marked by fibrosis and vasculopathy. While microvascular damage is a well-recognized feature, macrovascular involvement, such as peripheral arterial disease (PAD), has received less attention. PAD is a severe consequence of atherosclerosis and confers significant cardiovascular risk. This study aimed to evaluate PAD prevalence in SSc and its association with clinical features.

Methods: We conducted a case-control study in a tertiary center. Patients fulfilled ACR/EULAR 2013 or VEDOSS criteria and were registered in the national rheumatology registry (Reuma.pt). A control group was also included. Sociodemographic and clinical data were collected. PAD was assessed by the Ankle-Brachial Index (ABI), with ABI < 0.9 defining PAD. ABI was measured systematically by the same experienced vascular surgeon. Statistical analysis included χ^2 or Fisher's exact test and logistic regression for significant associations.

Results: We included 88 SSc patients and 60 controls (Table 1). SSc patients were more frequently female (85.2% vs. 43.3%, $p < 0.001$) and had lower rates of regular alcohol consumption (13.6% vs. 55.0%, $p < 0.001$). PAD was significantly more prevalent in the SSc group (10.2% vs. 1.6%, $p = 0.048$). No significant group differences were observed for hypertension, diabetes, dyslipidemia, or tobacco use.

Within the SSc group (table2), 81 patients met the ACR/EULAR 2013 classification criteria, while 7 met the VEDOSS criteria. Limited cutaneous scleroderma

157 – TABLE 1. Comparison of sociodemographic characteristics, traditional cardiovascular risk factors and doppler data in patients with systemic sclerosis (n=88) and controls (n=60)

	All (*n* =148)	SSc group (*n* =88)	Control group (*n* =60)	*p value*
Age (years)* - mean±SD	59.0±13.4	60.0±12.4	57.5±14.7	0.269
Sex - n (%)				**<0.001**
Female	101 (68.2)	75 (85.2)	26 (43.3)	
Male	47 (31.8)	13 (14.8)	34 (56.7)	
Race - n(%)				1.000
White european	147 (99.3)	87 (98.9)	60 (100.0)	
White hispanic	1 (0.7)	1 (1.1)	0 (0.0)	
Alcohol consumption - n(%)*				<0.001
Absent or sporadic	101 (66.9)	74 (84.1)	27 (45.0)	
Regular consumption	45 (30.4)	12 (13.6)	33 (55.0)	
Tobacco use - n(%)*				0.696
Current smoker	25 (16.9)	13 (14.8)	12 (20.0)	
Former smoker	14 (9.5)	8 (9.1)	6 (10.0)	
Non-smoker	108 (73.0)	66 (75.0)	42 (70.0)	
Body mass index (Kg/m²) - n(%)*				
Overweight (>25.0 kg/m2)	44 (29.7)	14 (15.9)	30 (50.0)	0.193
Hypertension diagnosis - n(%)	36 (24.3)	19 (21.6)	17 (28.3)	0.368
Type 2 diabetis - n(%)	16 (10.8)	7 (8.0)	9 (15.0)	0.183
Dyslipidaemia - n(%)	58 (39.2)	31 (35.2)	27 (45.0)	0.253
CHD - n(%)*	2 (1.4)	0 (0.0)	2 (3.3)	0.162
Hystory of stroke - n(%)*	7 (4.7)	5 (5.7)	2 (3.3)	0.701
Ankle brachial index <0.9 - n(%)	**10 (6.8)**	**9 (10.2)**	**1 (1.6)**	**0.048**
Medications at the time ABI - n(%)*				
Aspirin	21 (14.2)	13 (14.8)	8 (13.3)	1.000
CCB	80 (54.1)	72 (81.8)	8 (13.3)	**<0.001**
Fibrate	5 (3.4)	3 (3.4)	2 (3.3)	1.000
Statine	57 (38.5)	32 (36.4)	25 (41.7)	0.490
NOAC	3 (2.0)	2 (2.3)	1 (1.7)	1.000
Imunosupressants				
MTX	23 (15.3)	23 (26.1)		
HCQ	5 (3.4)	5 (5.7)		
MMF	4 (2.7)	4 (4.5)		
RTX	1 (0.7)	1 (1.1)		
Benzodiazepines	6 (4.1)	3 (3.4)	3 (5.0)	0.682
SSRIs	22 (14.9)	19 (21.6)	3 (5.0)	**0.007**
TAD	7 (4.7)	7 (8.0)	0 (0.0)	**0.027**
PDE5i	6 (4.1)	6 (6.8)	0 (0.0)	0.081
Opiates	1 (0.7)	1 (1.1)	0 (0.0)	1.000

Footnote: AC - absent contractility; ACR - American College of Rheumatology; CCB - calcium channel blockers; CHD - Coronary heart disease; HCQ - hydroxichloroquine; IQR - Interquartile Range; NOAC - New oral anticoagulants; MMF - mycophenolate mophetil; MTX - methotrexate; SD - Standard deviation; SSRIs - Selective serotonin reuptake inhibitors; TAD - tricyclic antidepressants; VEDOSS - Very Early Diagnosis of Systemic Sclerosis. Alcohol excessive consumption: >7 units/week **Missing values: 67 for BMI; 3 for medications, 1 for tobacco use and 1 for alcohol consumption; 2 for CHD* and stroke

was the predominant subtype (75.0%). PAD patients were older (66.2±12.2 vs.59.3±12.3), more often male (33.3% vs. 12.7%), and had higher frequencies of digital ulcers (66.7% vs. 20.3%, p=0.019), hypertension (55.6% vs.17.7%, p=0.021), and calcinosis (77.8% vs.10.1%, p<0.001). No associations were found with autoantibody profiles, disease subtype, or other cardiovascular risk factors.

Multivariate logistic regression identified calcinosis (OR = 37.9, 95% CI: 4.6–308.9, p < 0.001) and hypertension (OR = 8.8, 95% CI: 1.1–68.5, p = 0.038) as independent predictors of PAD in SSc. Digital ulcers showed a borderline association (OR = 7.1, 95% CI: 1.0–52.2, p = 0.055), suggesting a potential link with more severe vascular disease. In the full cohort model (SSc + controls), SSc remained an independent

157 – TABLE 2. Association of systemic sclerosis features with peripheral disease (ABI <0.9) in patients with systemic sclerosis (n=88).

	SSc group (*n* =88)	ABI <0.9 (*n* =9)	ABI >= 0.9 (n=79)	*p value*
Age (years)* - mean±SD	60.49±12.5	66.2±12.2	59.32+12.3	0.115
Age at diagnosis (years) - mean±SD	51.85±13.9	59.1±15.8	51.09+13.6	0.125
Sex - n (%)				0.125
Female	73 (83.0)	6 (66.7)	60 (75.9(	
Male	13 (14.8)	3 (33.3)	10 (12.7)	
Race - n(%)				1.000
White european	87 (98.9)	9 (100.0)	78 (98.7)	
White hispanic	1 (1.1)	0 (0.0)	1 (1.3)	
Classification Criteria ACR/EULAR 2013 - n(%)	81 (92.0)	9 (100.0)	72 (91.1)	1.000
Disease subtype - n(%)				0.930
Limited cutaneous scleroderma	66 (75.0)	9 (100.0)	57 (72.2)	
Overlap syndrome	6 (6.8)	0 (0.0)	6 (7.6)	
Sjogren's disease	2 (2.0)	0 (0.0)	2 (2.5)	
Systemic lupus eruthematous	1 (1.1)	0 (0.0)	1 (1.3)	
Rheumatoid arthiritis	3 (3.4)	0 (0.0)	3 (3.8)	
VEDOSS	7 (8.0)	0 (0.0)	7 (8.9)	
Sinescleroderma	5 (5.7)	0 (0.0)	5 (6.3)	
Diffuse scleroderma	4 (4.5)	0 (0.0)	4 (5.1)	
Antinuclear antibodies - n(%)				0.811
Centromere	62 (70.5)	8 (88.9)	54 (68.4)	
Nuclear fine speckled	10 (11.4)	1 (11.1)	9 (11.4)	
Nucleolar	8 (9.1)	0 (0.0)	8 (10.1)	
Homogeneous	7 (8.0)	0 (0.0)	7 (8.9)	
SSc related antibodies - n(%)				
Anticentromere	61 (69.3)	8 (88.9)	53 (67.1)	0.265
CENP-A*	42 (47.7)	6 (66.7)	36 (45.6)	0.126
CENP-B*	46 (52.3)	6 (66.7)	40 (50.6)	0.238
Anti-Scl70*	16 (18.2)	1 (11.1)	15 (19.0)	1.000
Anti-PM/Scl*	11 (12.5)	1 (11.1)	10 (12.7)	1.000
Anti-Ro52*	8 (9.1)	1 (11.1)	7 (8.9)	0.522
Anti-Th/To*	5 (5.7)	0 (0.0)	5 (6.3)	1.000
Anti-SSA	3 (3.4)	0 (0.0)	3 (3.8)	1.000
Anti-Ku*				
Anti-RNA polymerase III*	2 (2.3)	0 (0.0)	2 (2.5)	0.333
Anti-SSB	9 (10.2)	0 (0.0)	9 (11.4)	1.000
Anti-U1-RNP*	1 (1.1)	0 (0.0)	1 (1.3)	1.000
Anti-U3-RNP*	0 (0.0)	0 (0.0)	0 (0.0)	
Anti-U11/U12-RNP*	0 (0.0)	0 (0.0)	0 (0.0)	
Skin fibrosis - n(%)	66 (75.0)	8 (88.9)	58 (73.4)	0.116
Sclerodactyly - n(%)	62 (70.5)	9 (100.0)	53 (67.1)	0.154
Talengiectasias - n(%)	48 (54.5)	7 (77.8)	41 (51.9)	0.174
Raynaud - n(%)	87 (98.9)	9 (100.0)	78 (98.7)	1.000
DU - n(%)	26 (29.5)	6 (66.7)	20 (25.3)	**0.019**
Calcinosis - n(%)	15 (17.0)	7 (77.8)	8 (10.1)	**<0.001**
Nailfold capillaroscopy alterations - n(%)	76 (86.4)	7 (77.8)	69 (87.3)	1.000
Arthritis - n(%)	30 (34.1)	2 (22.2)	28 (35.4)	0.712
Myositis - n(%)	2 (2.3)	0 (0.0)	2 (2.5)	1.000
Esophageal involvment - n(%)	28 (31.)	4 (44.4)	24 (30.4)	0.264
Pulmonary involvment - n(%)	18 (20.5)	2 (22.2)	16 (20.3)	1.000
Cardiac involvment - n(%)	1 (1.1)	0 (0.0)	1 (1.3)	1.000
Renal involvment - n(%)	0 (0.0)	0 (0.0)	0 (0.0)	
Hypertension - n(%)	19 (21.6)	5 (55.6)	14 (17.7)	**0.021**
Type 2 diabetis - n(%)	7 (8.0)	0 (0.0)	7 (8.9)	1.000
Dyslipidaemia - n(%)	31 (35.2)	3 (33.3)	28 (35.4)	1.000
CHD - n(%)	0 (0.0)	0 (0.0)	0 (0.0)	
Hystory of stroke - n(%)	5 (5.7)	0 (0.0)	5 (6.3)	1.000
Alcohol consumption - n(%)*	11 (12.5)	3 (33.3)	8 (10.1=	0.086
Tobacco use - n(%)*	21 (23.9)	3 (33.3)	18 (22.8)	0.681

Footnote: ABI - Ankle brachial index; ACR - American College of Rheumatology; CCB - calcium channel blockers; CHD - coronary heart disease; DU - digital ulcers; IQR - Interquartile Range; SD - Standard deviation; SSc - Systemic sclerosis; SSRIs - Selective serotonin reuptake inhibitors; TAD - tricyclic antidepressants; VEDOSS - Very Early Diagnosis of Systemic Sclerosis. **Missing values: 67 for BMI; 3 for medications, 1 for tobacco use and 1 for alcohol consumption; 8 for CENP-A, CENP-B, Anti-PM/Scl, Anti-Th/To, Anti-Ku, Anti-U1-RNP, Anti-U3-RNP, Anti-U11/U12-RNP; 4 for anti-Ro52; 2 for anti-Scl70, anti-RNA polymerase III*

PAD predictor (OR=22.9, 95% CI: 2.0–258.2, p=0.011), even after adjustments for sex, alcohol, and hypertension.

Discussion: Our findings support that macrovascular disease is an underrecognized feature of SSc. PAD was more frequent in SSc and was strongly associated with calcinosis, hypertension, and potentially digital ulcers. The association with calcinosis and the consistent trend with digital ulcers suggest that PAD may indicate a more severe disease phenotype, possibly linked to poorer outcomes. On the other hand, considering that vascular calcification is a known predictor of PAD and adverse prognosis, the link between calcinosis and PAD may reflect overlapping mechanisms of vascular injury and calcification. This raises the possibility that SSc patients with calcinosis may fall into a higher cardiovascular risk category and could benefit from stricter risk control, including lifestyle measures and possibly early high-dose statin therapy for primary prevention.

Although further studies with larger cohorts are needed to better understand the underlying mechanisms of macrovascular involvement in SSc, ABI screening could serve as a valuable tool for risk stratification and clinical decision-making in this population.

160 - SALIVARY GLAND ULTRASOUND IN SYSTEMIC SCLEROSIS: OVERLAP WITH SJÖGREN'S DISEASE OR JUST A LOOK-ALIKE?

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Introduction: Salivary gland ultrasonography (SGUS) is a valuable tool in autoimmune diseases. While well validated in Sjögren's disease (SD), SGUS has also shown associations with clinical and serological features in systemic sclerosis (SSc). Despite overlapping symptoms like oral dryness, SGUS findings have not been directly compared between SD and SSc.

Objective: To compare salivary gland ultrasonographic, clinical, and immunological parameters between patients with SD and those with SSc.

Methods: Cross-sectional study including 22 SD and 18 SSc patients from a single center. All SD patients met 2016 ACR/EULAR criteria; SSc patients met 2013 ACR/EULAR criteria and were selected based on anti-SSA and/or anti-Ro52 positivity. Clinical, serological, and SGUS data were collected. SGUS was scored using the OMERACT system. Associations between categorical variables were analyzed using chi-square or Fisher's exact test.

Results: Patients with SD were younger at diagnosis and at the time of SGUS compared to those with SSc [median 52 (IQR 16.6) vs. 65.5 (IQR 16.2) years, p = 0.012; mean 57.2±13.7 vs. 66.6±8.8 years, p = 0.022]. Sicca symptoms were significantly more frequent in SD (dry mouth: 100% vs. 61.1%, p = 0.004; dry eyes: 95.5% vs. 33.3%, p < 0.001). Reactive lymph nodes were more common in SD (45.5% vs. 16.7%, p = 0.028). No statistically significant differences were found between groups regarding glandular size, edge definition, hyperechogenic bands, or OMERACT scores. Twenty-five patients (62.5%) had OMERACT scores ≥2, with similar proportions in both groups (59.1% in SD and 62.2% in SSc, p = 0.753). These results are shown in Table 1.

Across the entire cohort, OMERACT ≥2 was significantly associated with ANA patterns (able 2). Centromere (32.0% vs. 14.3%) and homogeneous (8.0% vs. 0%) patterns were more frequent in the OMERACT ≥2 group, while nucleolar pattern predominated in OMERACT <2 (35.7% vs. 0%) (p = 0.010). The speckled pattern was similarly prevalent across subgroups (≈56%). Anti-Ro52 positivity was also significantly associated with OMERACT ≥2 (80.0% vs. 57.1%, p = 0.036). No associations were found with anti-SSB, sicca symptoms, pulmonary involvement, or focus score ≥1 on histology.

Discussion: SGUS abnormalities were observed in both SD and SSc, despite distinct clinical profiles. The similarity in SGUS severity suggests that salivary gland damage also occurs in SSc, particularly in anti-Ro52/SSA-positive patients. The significant association between OMERACT ≥2 and centromere and homogeneous ANA patterns may reflect immunologically driven glandular changes. Interestingly, the speckled ANA pattern, typically linked to anti-SSA/Ro and more frequent in SD, was similarly distributed across OMERACT groups. Although one might expect higher prevalence in OMERACT ≥2, this was not observed. This could reflect sample size limitations or the immunological selection of SSc patients, which may have equalized serological features. Alternatively, SGUS abnormalities in SSc may arise via distinct mechanisms, such as fibrosis or vascular damage, despite overlapping antibody profiles. These findings support SGUS as a useful tool in SSc, capable of identifying glandular involvement that may reflect broader autoimmune activity beyond classic Sjögren's disease.

160 – TABLE 1. Sociodemographic, clinical, immunological, and ultrasonographic characteristics of patients with primary Sgögren's disease (SD) and systemic sclerosis (SSc).

	All (*n* =40)	SD patients (n=22)	SSc patients (*n* =18)	*p value*
Age at diagnosis (years)* - median (IQR)	55 (22.75)	52 (16.6)	65.5 (16.2)	**0.012**
Age at time of gland salivary US - mean±SD	61.2±12.6	57.2±13.7	66.6±8.8	**0.022**
Sex - n (%)				0.642
Female	35 (87.5)	20 (90.9)	15 (83.3)	
Male	5 (12.5)	2 (9.1)	3 (16.7)	
Disease - n(%)				
Sjogren's Disease	22 (55.0)			
Systemic Sclerosis	18 (45.0)			
Sicca symptoms - n(%)*				
Dry mouth	33 (82.5)	22 (100.0)	11 (61.1)	**0.004**
Dry eyes	27 (67.5)	21 (95.5)	6 (33.3)	**<0.001**
Parotid swelling - n(%)*	0 (0.0)	0 (0.0)	0 (0.0)	
Pulmonary involvement - n(%)	8 (20.0)	2 (9.1)	6 (33.3)	0.110
Uultrasound parameters - n(%)				
Glandular involvement (side)				0.277
Without involvement (normal US)	3 (7.5)	5 (22.7)	6 (33.3)	
Unilateral involvement	26 (65.0)	3 (13.6)	0 (0.0)	
Bilateral involvement	11 (27.5)	14 (63.6)	12 (66.7)	
Glandular involvement (type)				0.630
Parotids only	3 (7.5)	2 (9.1)	1 (5.6)	
Submandibulars only	6 (15.0)	2 (9.1)	4 (22.2)	
Predominantly parotids	2 (5.0)	2 (9.1)	0 (0.0)	
Predominantly submandibulars	3 (7.5)	2 (9.1)	1 (5.6)	
Both	15 (37.5)	9 (40.9)	6 (33.3)	
Size*				0.850
Atrophy	12 (30.0)	6 (27.3)	6 (33.3)	
Normal	26 (65.0)	15 (68.2)	11 (61.1)	
Hypertrophy	1 (2.5)	1 (4.5)	0 (0.0)	
Edge*				0.361
Irregular	13 (32.5)	6 (27.3)	7 (38.9)	
Normal	26 (65.0)	16 (72.7)	10 (55.6)	
Hyperechpic bands*	11 (27.5)	7 (31.8)	4 (22.2)	0.725
Lymph nodes				**0.028**
Normal	25 (62.5)	10 (45.4)	15 (83.3)	
Reactive	13 (32.5)	10 (45.5)	3 (16.7)	
Adenomegaly	2 (5.0)	2 (9.1)	0 (0.0)	
OMERACT - n(%)*				0.298
0	11 (27.5)	5 (22.7)	6 (33.3)	
1	4 (10.0)	4 (18.2)	0 (0.0)	
2	11 (27.5)	6 (27.3)	5 (27.8)	
3	13 (32.5)	7 (31.8)	6 (33.3)	
				0.753
OMERACT 0-1	14 (35.0)	9 (40.9)	6 (33.3)	
OMERACT >= 2	25 (62.5)	13 (59.1)	11 (62.2)	
ANAs	40 (100.0)	22 (100.0)	18 (100.0)	
ANA pattern - n(%)				**<0.001**
Speckled pattern	22 (55.0)	18 (81.8)	4 (22.2)	
Centromere pattern	10 (25.0)	0 (0.0)	10 (55.6)	
Nucleolar pattern	6 (15.0)	2 (9.1)	4 (22.2)	
Homogenous pattern	2 (5.0)	2 (9.1)	0 (0.0)	
anti-SSA	36 (90.0)	18 (81.8)	18 (100.0)	0.114
anti-Ro52*	29 (72.5)	15 (68.2)	14 (77.8)	0.108
anti-SSB*	9 (22.5)	6 (27.3)	3 (16.7)	0.704
Histopathology - n(%)*				
Available minor salivary gland biopsies	21 (52.5)	14 (63.6)	7 (38.9)	
FS >=1	14 (35.0)	11 (50.0)	3 (16.7)	0.156

Footnote: SD – Sjögren's disease; SSc – Systemic sclerosis; ANA – Antinuclear antibodies; US – Ultrasonography; OMERACT – Outcome Measures in Rheumatology; FS – Focus score; SSA, SSB, Ro52 – autoantibodies. *Note: Missing data for: Edge, Size, Hyperechogenic bands, OMERACT, anti-SSB (1 patient); minor salivary gland biopsy (2 patients); anti-Ro52 (3 patients).*

160 – TABLE 2. Association between OMERACT salivary glad ultrasound score (≥ 2 vs <2) and clinical, serological, and histopathological features.

	OMERACT <2 (*n* =14)	OMERACT ≥ 2 (n=25)	p value
Sicca symptoms - n(%)*			
Dry mouth	12 (85.7)	20 (80.0)	0.663
Dry eyes	11 (78.6)	15 (60.0)	0.728
Parotid swelling - n(%)*			
Pulmonary involvement - n(%)	3 (21.4)	4 (16.0)	1.000
ANA pattern - n(%)			**0.010**
Speckled pattern	8 (57.1)	14 (56.0)	
Centromere pattern	2 (14.3)	8 (32.0)	
Nucleolar pattern	5 (35.7)	0 (0.0)	
Homogenous pattern	0 (0.0)	2 (8.0)	
anti-SSA	12 (85.7)	23 (92.0)	0.279
anti-Ro52*	8 (57.1)	20 (80.0)	**0.036**
anti-SSB*	2 (14.3)	7 (28.0)	0.438
Histopathology - n(%)*			
FS >=1	6 (42.9)	8 (32.0)	1.000

Footnote: SD – Sjögren's disease; SSc – Systemic sclerosis; ANA – Antinuclear antibodies; OMERACT – Outcome Measures in Rheumatology; FS – Focus score; SSA, SSB, Ro52 – autoantibodies. *Missing values for anti-Ro52 (3 patients), OMERACT, anti-SSB (1 patient), and FS (2 patients)*

161 - FRAILSPA - UNDERSTANDING FRAILTY IN AXIAL SPONDYLOARTHRITIS: RISK FACTORS, PATIENT OUTCOMES AND THE LINK WITH SARCOPENIA AND OSTEOPOROSIS

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Background: Axial spondyloarthritis (axSpA) is a chronic inflammatory disease typically diagnosed in young adults. However, the long-term disease burden and systemic effects may contribute to premature frailty, a multidimensional syndrome marked by exhaustion, weakness, and reduced physical function. Sarcopenia and osteoporosis may further exacerbate vulnerability in this population, reinforcing the concept of osteosarcopenia in inflammatory rheumatic diseases.

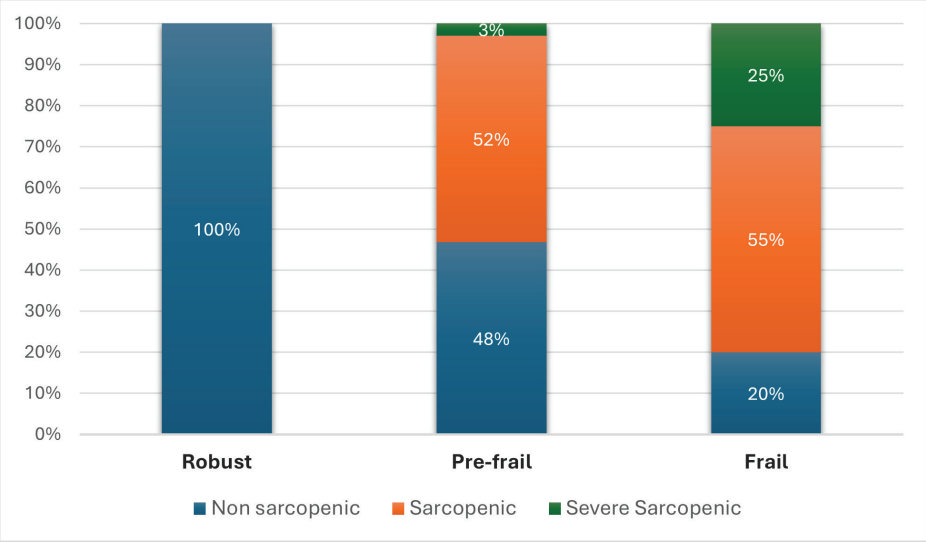
Objectives: To assess the prevalence of frailty and its relationship with sarcopenia and osteoporosis, identify associated risk factors, and evaluate the overall health impact in patients with axSpA.

Methods: We conducted a monocentric, cross-sectional study between October 2024 and May 2025, including patients with axSpA who fulfilled the ASAS criteria. Frailty status was defined using Fried's criteria, while sarcopenia was assessed using the EWGSOP2 definition, incorporating muscle strength (by handgrip dynamometry), muscle mass (by bioelectrical impedance analysis) and physical performance (gait speed). Osteoporosis was defined as a T-score of ≤ –2.5 at the lumbar spine, femoral neck, or total hip, measured by dual-energy X-ray absorptiometry (DXA). Data on demographic and clinical variables, disease-related scores, comorbidities (Charlson Comorbidity Index), physical function (HAQ-DI), quality of life (EQ-5D), and depression/anxiety (HADS-A/D) were collected.

161 – TABLE 1. Demographics, clinical, and patient-reported outcomes associated with frailty in patients with axial spondyloarthritis

Variables	Non-Frail (n=81)	Frail (n=20)	p
Mean age (SD) (years)	50,2 (12,4)	64,2 (14,5)	0,001
Women, n (%)	34 (42%)	11 (58%)	0,324
Body mass index (SD) (kg/m2)	26,9 (3,2)	23,3 (5,2)	0,551
Charlson Comorbidity Index, median (IQR)	1 (0-1)	3 (2-3)	0,014
Radiographic AxSpA, n (%)	34 (63%)	19 (95%)	0,022
Disease duration, median (IQR) (years)	17 (12-27)	34 (28-44)	<0,001
Diagnosis delay, median (IQR) (years)	6 (2-9)	21 (12-28)	<0,001
HLA-B27, n (%)	61 (75%)	13 (65%)	0,401
CRP, median (IQR) (mg/dL)	0,69 (0,18-0,88)	1,00 (0,37-1,31)	0,101
BASDAI, mean (SD)	3,9 (2,0)	4,1 (2,7)	0,613
ASDAS PCR, mean (SD)	2,1 (0,6)	2,6 (1,0)	0,229
BASFI, mean (SD)	3,6 (2,4)	5,2 (2,3)	0.045
BASMI, mean (SD)	3,1 (1,9)	5,0 (1,0)	<0,001
HAQ, mean (SD)	0,488 (0,545)	1,665 (0,622)	<0,001
EQ-5D, mean (SD)	0,566 (0,325)	0,130 (0,432)	0,033
HADS-D, median (IQR)	7 (5-8)	9 (8-13)	0,090
HADS-A, median (IQR)	8 (6-11)	9 (8-15)	0,155
NSAIDs, n (%)	47 (58%)	6 (30%)	0,053
csDMARD, n (%)	9 (11%)	4 (20%)	0,137
b-tsDMARD, n (%)	29 (36%)	6 (30%)	0,798
Osteoporosis, n (%)	18 (22%)	10 (50%)	0,009
Fragility fracture, n (%)	5 (6%)	7 (35%)	<0,001

ASDAS, Axial Spondyloarthritis Disease Activity Score; AxSpA, axial spondyloarthritis; b/tsDMARD, biologic/target synthetic disease-modifying anti-rheumatic drugs; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; BASMI, Bath Ankylosing Spondylitis Metrology Index; CRP: C-reactive protein; csDMARD: conventional synthetic disease-modifying anti-rheumatic drugs EQ-5D, EuroQol 5 dimensions; ESR: erythrocyte sedimentation rate; HADS-D/A, Hospital Anxiety and Depression Scale – Depression/Anxiety; HAQ: Health Assessment Questionnaire; IBD, inflammatory bowel disease; IQR, interquartile range; NSAIDs, nonsteroidal anti-inflammatory drugs; SD, standard difference



161 – Figure 1. Distribution of prevalence of sarcopenia according to Fried's frailty phenotype

Frail patients were compared to non-frail individuals (pre-frail and robust patients) to identify factors associated with frailty status. Univariate analysis, followed by age-adjusted logistic regression, was performed to identify independent associations.

Results: A total of 101 patients were included. Frailty was identified in 20 (19,8%) and pre-frailty in 29 (28,7%), primarily attributed to exhaustion, inactivity, and weakness. Frailty was significantly associated with older age, longer disease duration, delayed diagnosis, radiographic involvement, comorbidities, higher BASMI scores, poorer function, and reduced quality of life compared with non-frail patients (Table 1). Osteoporosis and fragility fractures were also significantly more frequent among frail patients. Sarcopenia was identified in 55% of pre-frail patients and 80% of frail patients, five of whom had severe sarcopenia (Figure 1). After adjusting for age, longer diagnostic delay, history of fragility fracture, and severe sarcopenia were significantly associated with frailty. In multivariate analysis, only diagnostic delay and fragility fractures remained independently associated, possibly reflecting the study's limited sample size.

Conclusion: Frailty affected nearly one in five patients with axSpA in our cohort and was independently associated with both a delayed diagnosis and a history of fragility fractures. Sarcopenia was highly prevalent among frail patients, highlighting its clinical significance in this population. These results underscore the importance of systematically assessing frailty and musculoskeletal health in axSpA, particularly in ageing patients. To more effectively assess frailty, we aim to expand and enhance our cohort to include a broader Portuguese population, enabling the development of targeted interventions to prevent frailty and mitigate its impact on this vulnerable group.

162 - KNOWLEDGE AND PERSPECTIVES ON CANNABINOID-BASED THERAPIES FOR CHRONIC PAIN: A COMPARISON BETWEEN RHEUMATOLOGISTS AND PATIENTS IN PORTUGAL

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Introduction: Cannabinoid-based therapies (CBT) are gaining interest for managing chronic pain in rheumatic diseases. Despite legal approval in Portugal, their clinical use remains limited. Understanding the perspectives of both patients and rheumatologists is crucial for their safe and effective implementation.

Objectives: To assess the knowledge, experiences, and perceptions of rheumatologists and patients regarding CBT in chronic pain management; and to compare patient beliefs with physicians' perceptions.

Methods: A cross-sectional online survey was conducted in the Portuguese population between December 2023 and May 2024, targeting two groups: rheumatologists and patients with rheumatic diseases. The questionnaire assessed demographics, knowledge levels, prior experiences with CBT, and perceptions regarding efficacy and safety. Agreement between patient beliefs and physicians' perceptions was analysed using the Chi-square test.

Results: We obtained 104 physician and 110 patient responses. Most physicians were female (62.5%), aged 41–50 years (42.3%), and 75.0% were rheumatology specialists. Among patients, the majority were female (90.9%), aged 51–60 years (36.4%). The most frequently reported diagnoses were fibromyalgia (42.7%), spondyloarthritis (25.5%) and rheumatoid arthritis (13.6%). Chronic pain was highly prevalent (92.7%), with 66.4% reporting dissatisfaction with current pain management. All physicians were aware of CBT, but only 12.5% had prescribed them, mainly for fibromyalgia (64.3%). Nevertheless, 75% would consider them for opioid-refractory chronic pain. Only 34.6% reported good knowledge of the main indications, while most felt uninformed about available formulations (79.8%) and potential side effects or drug interactions (74%). Consequently, 94.2% expressed interest in further education, and 54.8% supported including CBT in undergraduate medical curricula. Among patients, 92.7% were aware of CBT for therapeutic use, mainly through the media (41.6%). Most (79.1%) supported their use for pain control, although only 39.1% reported awareness of potential adverse effects. Of those who used CBT (30%), only 9.4% obtained them through medical prescription. Most (84.4%) reported pain relief, and 18.2% experienced adverse effects. Only 18.2% had discussed CBT with their rheumatologist, of whom 55% reported a non-receptive response. A large majority (91.8%) expressed interest in further information. A comparison between patient beliefs and physicians' perceptions (Table 1) revealed significant discrepancies: 25.5% of patients agreed that CBT may slow disease progression vs 7.7% of physicians who thought patients held that belief (p=0.002); 31% of patients believed CBT could improve concentration vs 21% of physicians' estimates (p=0.003); 57.3% of patients believed that CBT helped with daily activities vs 43.3% of physicians (p=0.018); and 65.5% of patients supported medical supervision, while only 34.6% of physicians thought patients agreed

162 – TABLE 1. Comparison between the self-reported beliefs of patients with rheumatic diseases regarding cannabinoid-based therapies and rheumatologists' perceptions of those same beliefs.

Question (Q)	Patients (D/N/A) n (%)	Physicians (D/N/A) n (%)	p-value*
Q1. Cannabis-based medicines can cure rheumatic disease	70 (63.6) / 26 (23.6) / 14 (12.7)	72 (69.2) / 17 (16.3) / 15 (14.4)	0.411
Q2. Cannabis can help slow disease progression	55 (50.0) / 27 (24.5) / 28 (25.5)	69 (66.3) / 27 (26.0) / 8 (7.7)	0.002
Q3. Useful for pain control	6 (5.5) / 20 (18.2) / 84 (76.4)	6 (5.8) / 23 (22.1) / 75 (72.1)	0.759
Q4. Useful for anxiety control	14 (12.7) / 28 (25.5) / 68 (61.8)	19 (18.3) / 27 (26.0) / 58 (55.8)	0.496
Q5. Useful for mood improvement	15 (13.6) / 42 (38.2) / 53 (48.2)	19 (18.3) / 34 (32.7) / 51 (49.0)	0.553
Q6. Useful for sleep improvement	14 (12.7) / 37 (33.6) / 59 (53.6)	22 (21.2) / 35 (33.7) / 47 (45.2)	0.220
Q7. Useful for appetite stimulation	30 (27.3) / 50 (45.5) / 30 (27.3)	32 (30.8) / 34 (32.7) / 38 (36.5)	0.143
Q8. Useful for concentration	29 (26.4) / 47 (42.7) / 34 (30.9)	51 (49.0) / 31 (29.8) / 22 (21.2)	0.003
Q.9. Improves social relationships	31 (28.2) / 42 (38.2) / 37 (33.6)	30 (28.8) / 31 (29.8) / 43 (41.3)	0.376
Q.10. Improves ability to perform daily tasks	32 (29.1) / 15 (13.6) / 63 (57.3)	40 (38.5) / 19 (18.3) / 45 (43.3)	0.018
Q.11. Regular use may harm physical health	38 (34.5) / 50 (45.5) / 22 (20.0)	37 (35.6) / 49 (47.1) / 18 (17.3)	0.880
Q10. Regular use may harm mental health	36 (32.7) / 46 (41.8) / 28 (25.5)	33 (31.7) / 45 (43.3) / 26 (25.0)	0.977
Q.13. Use may lead to addiction	33 (30.0) / 43 (39.1) / 34 (30.9)	23 (22.1) / 41 (39.4) / 40 (38.5)	0.341
Q.14. Should only be used under medical supervision	24 (21.8) / 14 (12.7) / 72 (65.5)	52 (50.0) / 16 (15.4) / 36 (34.6)	< 0.001

Responses are presented as absolute numbers followed by percentages for each category: Disagree (D), Neutral (N), and Agree (A).
*Statistical differences were assessed using the Chi-square test ($p < 0.05$ considered significant).

with this ($p<0.001$).

Conclusion: Patients expressed strong interest in CBT, belief in their effectiveness, and willingness to use them. In contrast, physicians tend to be more cautious, often due to limited knowledge regarding formulations and safety. Promoting patient health literacy, enhancing clinicians' education, and developing evidence-based guidelines are essential to ensure the safe and informed use of CBT in rheumatology. A significant mismatch exists between patient beliefs and physician perceptions, underscoring the need for improved communication and education on this topic.

170 - JUVENILE IDIOPATHIC ARTHRITIS (JIA) WITH CHRONIC ANTERIOR UVEITIS: LONG-TERM PROGRESSION AND CLASSIFICATION INSIGHTS FROM A NATIONAL REUMA.PT STUDY

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Background: Chronic anterior uveitis is the most common extra-articular manifestation of juvenile idiopathic arthritis (JIA). Following the recent criteria proposed by the Paediatric Rheumatology International Trials Organisation (PRINTO), which suggest a unique JIA subtype in children at high risk of iridocyclitis, this study investigates whether chronic uveitis defines a distinct JIA subset with consistent characteristics into adulthood, independent of antinuclear antibodies (ANA) status, sex, or age at disease onset.

Objectives: To identify clinical and laboratory factors associated with chronic or recurrent anterior uveitis in JIA patients during long-term follow-up and assess whether this subgroup constitutes a distinct disease entity that persists into adulthood.

Methods: A retrospective, national, multicentre study using data from the National Registry of Rheumatic Patients (Reuma.pt) was conducted. JIA patients aged ≥18 years at their last visit were included. Chronic or recurrent uveitis was defined as a single episode lasting >3 months with relapse occurring within 3 months after treatment discontinuation, or repeated episodes with ≥3 months of inactivity between them. Patients with acute or no uveitis were grouped for analysis. Associations were analysed with appropriate statistical tests, and multiple comparisons were adjusted using the Holm-Bonferroni correction. A weighted logistic regression model was used for multivariate analysis.

Results: Among 778 patients with JIA, 63.8% were female, with a mean disease duration of 23.6 ± 3.1 years. Of these, 51 (6.6%) developed chronic or recurrent anterior uveitis during the follow-up period. No significant sex differences were observed between patients with and without uveitis (p=0.766).

Compared to those without uveitis, patients with chronic or recurrent uveitis had a significantly earlier disease onset [median 4.7 (IQR 6.4) vs. 10.1 (IQR 7.8) years, p<0.001], higher frequency of ANA positivity (66.0% vs. 33.0%, p<0.001), and lower frequency of rheumatoid factor (RF) positivity (2.4% vs. 17.0%, p=0.025). Persistent oligoarthritis was the most common JIA category in the uveitis group compared to those without (52.0% vs. 18.0%, p<0.001) (Table 1). Patients with uveitis were also more frequently treated with biologic disease-modifying antirheumatic drugs (bDMARDs) (62.7% vs. 44.7%, p=0.019) and had a higher rate of treatment switches (p<0.001).

In multivariate analysis, earlier age at onset (OR 0.93, 95% CI 0.89–0.97, p<0.001), ANA positivity (OR 2.64, 95% CI 1.80–3.90, p<0.001), RF negativity (OR 0.13, 95% CI 0.05–0.31, p<0.001), and persistent oligoarthritis (OR 2.38, 95% CI 1.51–3.80, p<0.001) remained independently associated with chronic uveitis (Figure 1).

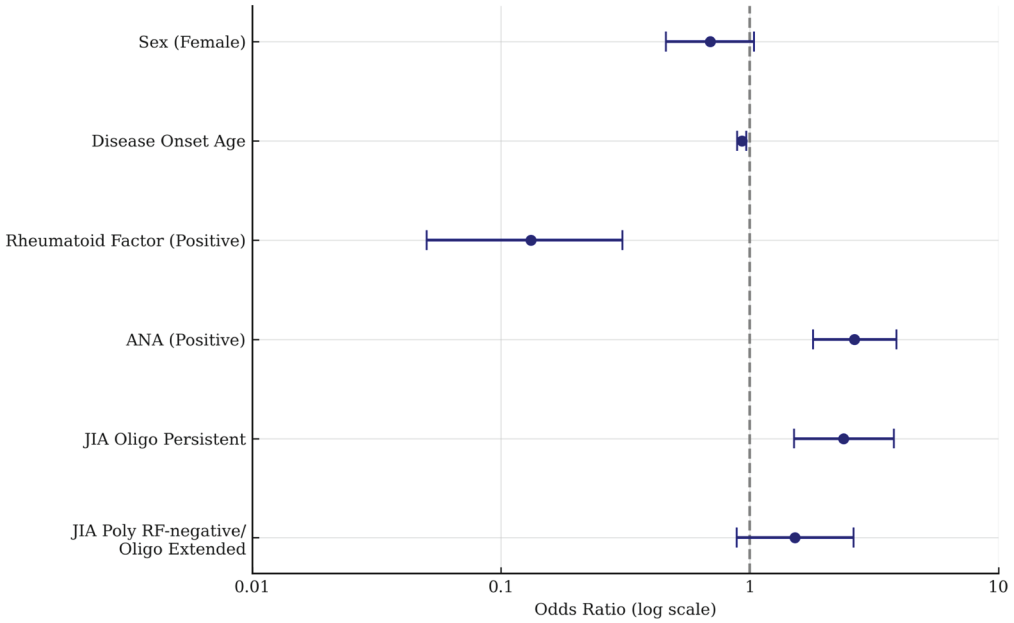
Despite long-term follow-up, only 46.5% of patients with chronic uveitis fulfilled adult disease classification criteria, compared to 73.8% of those without uveitis (p<0.001).

Conclusions: In long-term follow-up into adulthood, chronic anterior uveitis in JIA was associated with early onset, ANA positivity, RF negativity, and persistent oligoarthritis, but not with sex. These features remained consistent over time and defined a group of patients who rarely fulfilled adult classification criteria. This supports the concept of a unique childhood-onset disease that remains clinically distinct in adulthood.

170 – TABLE. Clinical and serological features of JIA patients with and without chronic uveitis.

	All (n=778)	With chronic uveitis (n=51)	Without chronic uveitis (n=727)	*P*-value
Female sex - n (%)	496 (63.8)	34 (66.7)	462 (63.6)	0.766
Age at disease onset - median (IQR)	9.8 (8.3)	4.7 (6.4)	10.1 (7.8)	**<0.001**
Antinuclear antibodies (ANA) status				**<0.001**
Positive - n (%)	225 (35.7)	33 (66.0)	192 (33.0)	
Negative - n (%)	406 (64.3)	17 (34.0)	389 (67.0)	
Rheumatoid factor (RF) status				**0.025**
Positive - n (%)	106 (16.1)	1 (2.4)	105 (17.0)	
Negative - n (%)	551 (83.9)	40 (97.6)	511 (83.0)	
Anti-CCP[1] antibodies status				**0.013**
Positive - n (%)	70 (16.2)	0 (0.0)	70 (17.3)	
Negative - n (%)	363 (83.8)	28 (100.0)	335 (82.7)	
bDMARD[2] treatment ever - n (%)	357 (45.9)	32 (62.7)	325 (44.7)	**0.019**
HAQ[3] (at last visit) - median (IQR)	0.0 (0.5)	0.0 (0.0)	0.0 (0.5)	0.065
JADAS10[4] (at last visit) - median (IQR)	1.7 (4.8)	1.2 (2.8)	1.8 (5.2)	0.131
ILAR JIA[5] Category				**<0.001**
Enthesitis related arthritis (ERA) - n (%)	150 (20.3)	8 (16.0)	142 (20.6)	1.000
Extended oligoarthritis - n (%)	95 (12.9)	10 (20.0)	85 (12.3)	0.628
Persistent oligoarthritis - n (%)	150 (20.3)	26 (52.0)	124 (18.0)	**<0.001**
Psoriatic arthritis - n (%)	38 (5.1)	1 (2.0)	37 (5.4)	1.000
RF-negative polyarthritis - n (%)	133 (18.0)	2 (4.0)	131 (19.0)	**0.028**
RF-positive polyarthritis - n (%)	97 (13.1)	1 (2.0)	96 (13.9)	0.087
Systemic (Still's disease) - n (%)	70 (9.5)	2 (4.0)	68 (9.9)	0.864
Undifferentiated arthritis - n (%)	6 (0.8)	0 (0.0)	6 (0.90)	1.000
Adult Inflammatory Disorders Criteria – n/N (%)	391/546 (71.6)	20/43 (46.5)	371/503 (73.8)	**<0.001**
Ankylosing spondylitis (AS)[6] - n (%)	53 (13.6)	5 (25.0)	48 (12.9)	0.595
Undifferentiated spondyloarthritis - n (%)	82 (21.0)	6 (30.0)	76 (20.5)	1.000
Rheumatoid arthritis (RA)[7] - n (%)	146 (37.3)	3 (15.0)	143 (38.5)	**0.001**
Psoriatic arthritis[8] - n (%)	42 (10.7)	3 (15.0)	39 (10.5)	1.000
IBD[9] associated arthropathy - n (%)	12 (3.1)	1 (5.0)	11 (3.0)	1.000
Still's disease[10] - n (%)	56 (14.3)	2 (10.0)	54 (14.6)	0.296

n: number of patients positive for the variable of interest; N: number of patients without missing information regarding the variable of interest; IQR: interquartile range.
[1] Anti-CCP: anti-cyclic citrullinated peptide; [2] bDMARD: biologic disease-modifying anti-rheumatic drugs; [3] HAQ: Health Assessment Questionnaire; [4] JADAS10: Juvenile Arthritis Disease Activity Score 10; [5] ILAR: International League of Associations for Rheumatology; JIA: Juvenile idiopathic arthritis; [6] According to the Modified New York criteria; [7] According to the 2010 ACR/EULAR classification criteria; [8] According to the CASPAR classification criteria; [9] IBD: inflammatory bowel disease; [10] According to the 1992 Yamaguchi's diagnostic criteria.
* Associations between variables were analysed with appropriate statistical tests, and multiple comparisons were adjusted using the Holm-Bonferroni correction. Values in bold indicate statistical significance at the 0.050 level ($p < 0.050$).



170 – Figure 1. Weighted logistic regression model (odds ratios with 95% CI) evaluating predictors of chronic uveitis in JIA patients.

182 - RADIOFREQUENCY ECHOGRAPHIC MULTI SPECTROMETRY (REMS) TECHNOLOGY IN THE EVALUATION OF GLUCOCORTICOID-INDUCED OSTEOPOROSIS IN SYSTEMIC LUPUS ERYTHEMATOUS PATIENTS

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Introduction: Patients with Systemic Lupus Erythematosus (SLE) have an increased risk of osteoporosis, particularly those undergoing glucocorticoid (GC) therapy. Dual-energy X-ray absorptiometry (DXA) remains the gold standard for bone mineral density (BMD) assessment, while Radiofrequency Echographic Multi Spectrometry (REMS) is emerging as a radiation-free alternative. This study aimed to compare REMS and DXA in SLE patients with and without GC therapy.

Methods: This cross-sectional observational study included 106 SLE patients referred for DXA evaluation. Patients were stratified by GC use at the time of assessment. BMD and T-score values were obtained at lumbar spine (LS), femoral neck (FN), and total hip (TH) using both DXA and REMS. Agreement between methods was evaluated using correlation analysis, Bland-Altman plots, and Cohen's kappa.

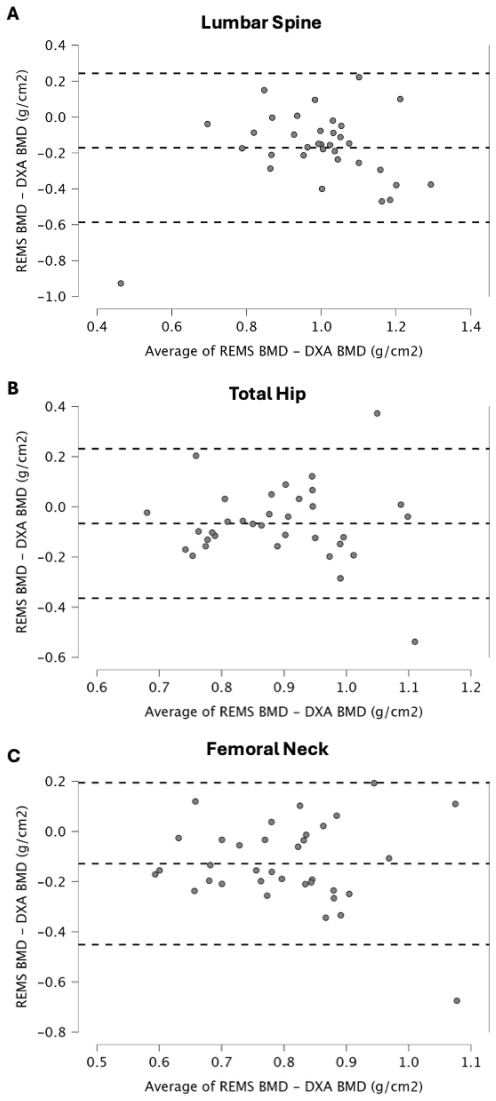
Results: A total of 106 patients with SLE were included, mostly women (88.7%) and with a mean age of 53.6±13.9 years old. Forty-four patients were under GC therapy and the mean cumulative dose (of prednisolone equivalent) in this group was 17 (6.5-31-3) mg and 3 (1-10.3) mg in the group of patients not currently on GC ($p<0.001$). In the GC-treated SLE patients, BMD measurements differed between REMS and DXA in the FN (0.739±0.134 vs 0.867±0.149, $p<0.001$), LS (0.906±0.204 vs 1.069±0.179, $p<0.001$) and TH (0.860±0.130 vs 0.921±0.144, $p=0006$), with the values obtained with REMS being significantly lower. In non-GC patients, significant differences were observed in both BMD and T-scores ($p<0.001$). DXA diagnosed osteoporosis in 27.3% of GC users and 12.9% of non-GC users, compared to 13.6% and 21% by REMS, respectively. REMS demonstrated modest sensitivity (33.3–60%) but high specificity (85.7–96.7%), with the best diagnostic agreement at a the FN.

Bland-Altman plots were obtained to evaluate the differences between BMD values measured with DXA and REMS for each site in the patients under GC therapy (figure 1). The average difference was -0.171±0.211 g/cm2 for LS, -0.066±0.152 g/cm2 for TH, and -0.128±0.165 g/cm2 for FN. The plots also highlighted the absence of any trend linking the BMD values measured by REMS and DXA to their average value, suggesting the accuracy of BMD evaluated by REMS does

182 – TABLE 1 - Results of the REMS accuracy evaluations for for lumbar spine and femoral neck in the GC treated and non treated groups; *p<0.05

	GC treated		No GC	
Anatomical site	Lumbar Spine	Femoral Neck	Lumbar Spine	Femoral Neck
Number of cases	34	33	46	44
Sensitivity (%)	33.3	60	33.3	33.3
Specificity (%)	91.4	94.9	89.8	85.7
k	0.239	0.549*	0.120	0.147
r	0.40*	0.41*	0.59*	0.60*
r2	0.16*	0.17*	0.35*	0.36*
Regression line slope	0.40	0.39	0.60	0.69
SEE (g/cm2)	0.190	0.127	0.190	0.153
Average diferences (mean±1.96 SD, g/cm2)	-0.171±0.211	-0.128±0.165	-0.217±0.176	-0.159±0.110

Footnote: GC – glucocorticoid, SEE - Standard Error of Estimate; SD – standard deviation



182 – Figure 1. Bland Altman plots comparing the REMS and DXA BMD measurements in the GC treated group at the lumbar spine (A), total hip (B) and femoral neck (C)

not depend on the BMD value. When looking at the non-GC treated group, similar results were obtained. The average difference was -0.217±0.176 g/cm2 at the LS, -0.087±0.097 g/cm2 for TH, and -0.159±0.110 g/cm2 at FN.

In the GC treated group, the Cohen's k correlation coefficients for the diagnosis of osteoporosis were: k=0.239 (95% CI -0.105, 0.582) at the LS, k=0.549 (95% CI -0.156, 0.941) at the FN and k=0.407 (95% CI 0.099, 0.716) when considering the worst T-score, reflecting a moderate agreement particularly at the FN. In the patients not currently on GC, the Cohen's k for the diagnosis of osteoporosis was 0.120 (95% CI -0.191, 0.430) at the LS, 0.147 (95% CI -0.154, 0.447) at FN and 0.271 (-0.011, 0.554) at any site.

The full comparison between the results obtained for the accuracy of REMS in each group is summarized in table 1.

Discussion: REMS demonstrated acceptable agreement with DXA for BMD assessment in SLE patients under GC, especially at the FN, but consistently underestimated BMD values. While specificity was high, limited sensitivity suggests that REMS may miss cases of osteoporosis. In this population, REMS could be a complementary tool in clinical practice but is not an ideal replacement of DXA for definitive diagnosis.

205 - FAST-TRACK CLINICS IMPROVE VISUAL OUTCOMES IN GIANT CELL ARTERITIS: A META-ANALYSIS

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Introduction: Giant Cell Arteritis (GCA) is a chronic vasculitis. Delayed diagnosis and treatment can result in severe complications, including irreversible vision loss. Fast-Track Clinics (FTC) have been developed to expedite diagnosis and treatment, potentially improving patient outcomes. This meta-analysis aims to compare the effectiveness of FTC versus conventional practice (CP) in managing GCA.

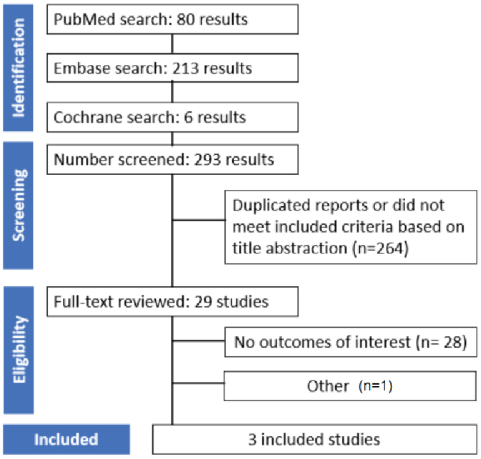
Methods: This systematic review with meta-analysis followed the PRISMA guidelines. We included peer- reviewed studies comparing Fast-Track Clinics with conventional practice in GCA management that reported on at least one of the following outcomes: visual disturbances, permanent sight loss, biopsy rates, or median days to diagnosis. There were no restrictions on publication date or language. A systematic literature search was conducted in MEDLINE (via PubMed), Embase, and the Cochrane Library on January 15, 2025. The search incorporated both free-text terms and controlled vocabulary including Medical Subject Headings (MeSH) and EMTREE terms. Key terms included: 'Giant Cell Arteritis', 'Temporal Arteritis', 'Fast-Track', 'Rapid Access', 'Early Diagnosis', 'Clinical Pathway', 'Visual Loss', and 'Sight Loss'.Extracted data included patient demographics and clinical presentation. Data extraction was performed using a standardized form capturing study characteristics, patient demographics, diagnostic pathways and reported outcomes. Quality assessment was performed using the QUADAS-2 tool. Pooled odds ratios (OR) with 95% confidence intervals (CI) were calculated for binary outcomes. P value ≤ 0.05 was considered statistically significant. Heterogeneity was assessed using the I^2 statistic. A random-effects model was applied due to anticipated heterogeneity. Funnel plots were used to assess publication bias. Statistical analysis was made with IBM SPSS Statistical Analysis. Outcomes were extracted as defined by the original studies. Permanent sight loss was typically defined as visual acuity reduction not recovered by the end of floow-up. Visual disturbances included transient of reversible symptoms such as diplopia ou amaurosis fugax.

Results: The PRISMA Box diagram of study screening and selection is shown in Figure 1. The search strategy in Embase, MEDLINE, and Cochrane yielded 293 results, of which 29 were fully reviewed for inclusion and exclusion criteria. Three studies met the inclusion criteria and were included in the meta-analysis. A total of 348 patients were included, with 173 in the Fast-Track Clinic (FTC) group and 175 in the Conventional Practice (CP) group. The baseline characteristics of patients are described in table 1. QUADAS are found in table 2. The pooled analysis demonstrated a statistically significant reduction in permanent sight loss among patients managed within an FTC group (82/173, 47.40%) compared to conventional practice (114/175, 65.14%) (OR 1.38; 95% CI [-0.01, 3.73]; p = 0.05. Median days since diagnosis was lower in the FTC group (57.6 days) compared to conventional practice (58.3 days), without being statistically significant (OR -1.11; 95% CI [-3.04, 0.86]; p = 0.26). The heterogeneity for permanent sight loss was low (I^2 = 0.00) and visual disturbances (I^2 = 0.25), whereas moderate heterogeneity was observed for biopsy rates (I^2 = 0.78) and median days for diagnosis (I^2 = 0.98).

Conclusion: This meta-analysis provides strong evidence that FTCs improve patient outcomes in GCA by significantly reducing the risk of permanent sight loss and visual disturbances.

205 – TABLE 1.

	Fast-track clinical (N=173)			Conventional Practice (N=175)		
	Patil et al, 2015	Diamantopoulos et al, 2016	Monti et al, 2020	Patil et al, 2015	Diamantopoulos et al, 2016	Monti et al, 2020
Age (Years)	74,1±7.6	72 (70-75)	75,1±7.6	75.4±7.6	74 (71-78)	70.6±8.2
Median days from disease onset to diagnosis	17,5 (0-206)	42 (21-84)	63(15-84)	21 (1-196)	42 (21-152)	60 (20-122)
CRP(mg/L)	35 (1- 286)	55 (27-81)	59 (24- 86)	60 (2- 329)	45 (31-77)	35 (20-99)
ESR (mm/H)	37±24.3	68 (58-78)	78 (63-96)	48±24.0	73 (63-84)	82 (48-102)
Tender temporal artery	52/67	12/43	13/63	35/46	12/32	5/97
Polymyalgic symptoms	28/67	18/43	32/63	16/46	13/32	48/97
Jaw	44/67	14/43	29/63	26/46	13/32	33/97
Claudication						
Biopsy performed	48/67	31/43	5/63	45/46	24/32	45/97
Visual Disturbances	9/67	9/43	17/63	11/46	9/32	37/97
Permanent Sight Loss	5/67	1/43	8/63	11/46	6/32	26/97



207 - CARDIOVASCULAR EVENTS IN PATIENTS TREATED WITH ROMOSOZUMAB AND OTHER ANTI-OSTEOPOROTIC AGENTS: A COMPARATIVE ANALYSIS USING REAL-WORLD DATA

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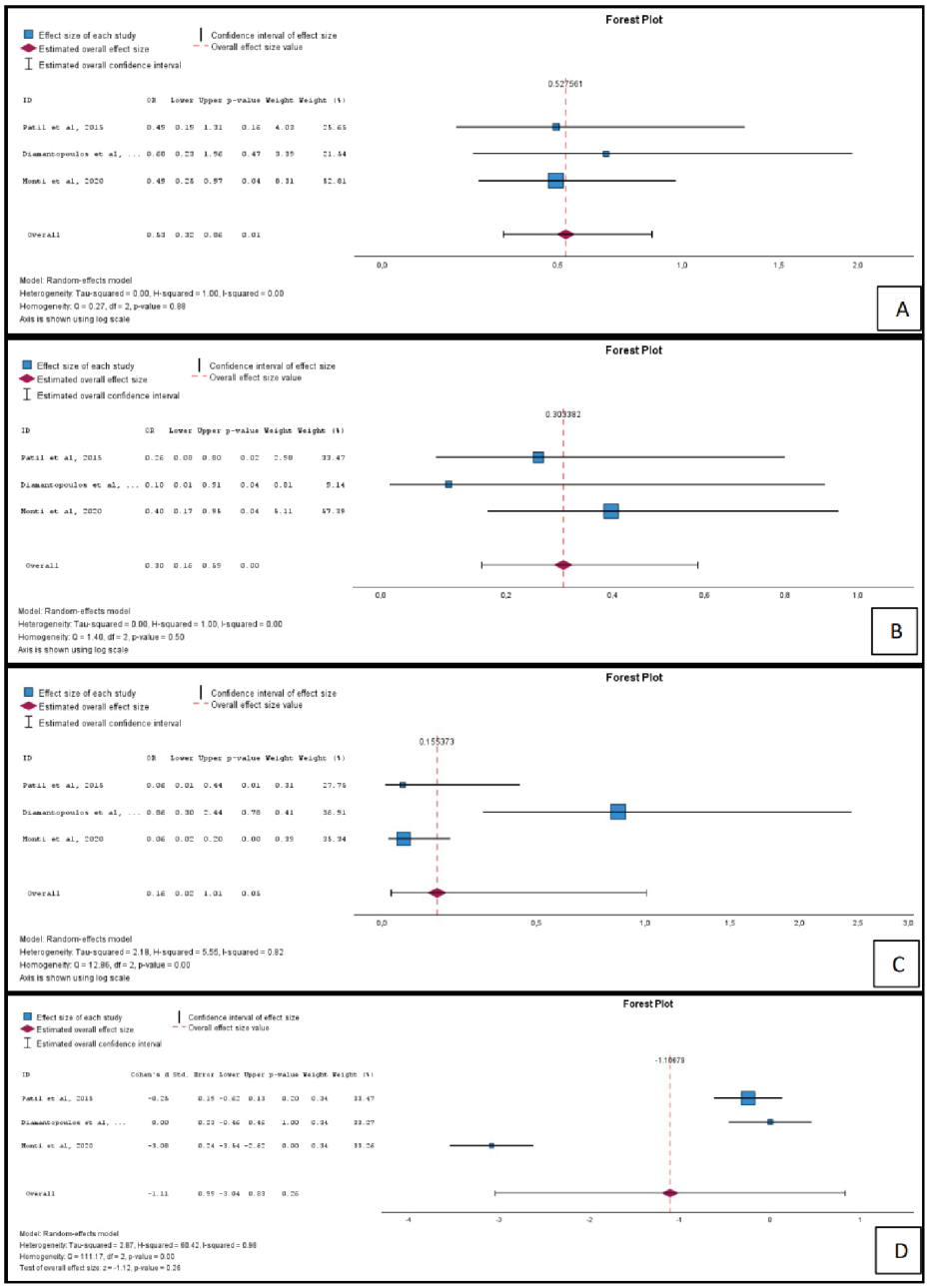

205 – Figure 1A. Figure 1A - PRIMA Box diagram; Figure 1B: Meta-analysis

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Introduction: Romosozumab (RMZ) is a recently approved agent with proven efficacy in treating Osteoporosis. However, its use has been associated with increased cardiovascular (CV) events. This study uses real-world data to describe and compare the occurrence of CV events among patients treated with RMZ and other anti-osteoporotic agents.

Methods and Materials: Retrospective study of EudraVigilance reports on CV events associated with RMZ, bisphosphonates (BP), and denosumab (DN) from January 2020 to December 2024. Reports by non-healthcare professionals, duplicates, or possibly related to more than one anti-osteoporotic treatment were eliminated. Demographics, events' outcomes, severity criteria, and associated actions were analyzed descriptively. Comparative analysis was performed to evaluate differences across treatments, stratifying by age, sex, event type, outcome, severity criteria, event duration, treatment indication, and approach. Reporting Odds Ratio (ROR) was calculated for RMZ compared to the other treatment groups.

Results: Healthcare professionals reported 33,348 suspect adverse reactions (SARs) in the EudraVigilance database (5,722 RMZ-, 12,246 BP-, and 15,380 DN-related SARs). Of these, 84 CV events were reported for RMZ, 8 for BP, and 10 for DN (Table 1).

207 – TABLE 1. Sociodemographic and clinical data.

	Romosozumab	Bisphosphonates	Denosumab
Sex - N (%) ¨			
Female	67 (81.70)	5 (71.43)	10 (100)
Male	15 (18.30)	2 (28.57)	0 0.00
Age Group - N (%)^			
18-64 years	13 (16.88)	2 (33.33)	1 (11.11)
65-85 years	55 (71.43)	3 (50.00)	8 (88.89)
Older than 85 years	9 (11.69)	1(16.67)	0 0.00
Primary Source Country, Non European Economic Area - N (%)	77 (91.67)	5 (62.50)	3 (30.00)
Treatment indication,- N (%) *			
Osteoporosis	73 (100)	4 (80.00)	2 (66.67)
Bone Lesions	0 (0.00)	1 (20.00)	1 (33.33)
Treatment duration, days - Mean ± SD	32.8±60.72	16.00±6.00	5.50±0.50
Type of Suspected adverse event - N (%)			
Acute myocardial infarction	50 (59.82)	4 (50.00)	5 (50.00)
Cerebrovascular Accident	34 (40.48)	4 (50.00)	5 (50.00)
Reaction outcome at time of report - N (%)**			
Recovered/resolved	29 (45.31)	1(16.67)	4 (66.67)
Recovering/resolving	13 (20.31)	2 (33.33)	0 (0.00)
Recovered with sequelae	1 (1.56)	0 (0.00)	0 (0.00)
Not recovered/not resolved	11 (17.19)	0 (0.00)	0 (0.00)
Fatal	9 (14.06)	3 (50.00)	2 (33.33)
Seriousness criteria - n (%)			
Caused/prolonged hospitalization	52 (61.90)	6 (75.00)	2 (20.00)
Disabling	3 (3.57)	0 (0.00)	0 (0.00)
Life Threatening	14 (16.67)	3 (37.50)	0 (0.00)
Other medically important condition	66 (78.57)	4 (50.00)	9 (90.00)
Resulted in death	9 (10.71)	3 (37.50)	2 (20.00)
Action taken - N (%) +			
Treatment withdraw	52 (98.11)	0 (0.00)	0 (0.00)
Treatment unchanged	1 81.89)	0 (0.00)	1 (100.00)
TOTAL	84	8	10

¨ of 82,7, and 5 with available data for romosozumab, bisphosphonates, and denosumab, respectively; *of 73, 8, and 5 with available data for romosozumab, bisphosphonates, and denosumab, respectively; **of 64,6, and 6 with available data for romosozumab, bisphosphonates, and denosumab, respectively; ^ of 77, 6, and 9 with available data for romosozumab, bisphosphonates, and denosumab, respectively; + of 53, 0, and 1 with available data for romosozumab, bisphosphonates, and denosumab, respectively.

The majority of CV SARs involved females aged 65-85 Years. All CV SARs were associated with at least one severity criteria, and the most reported was the presence of other important medical conditions (78.57% of RMZ -, 50% of BP-, and 90% of DN-related SARs), such as the presence of neoplasia. RMZ was stopped in all cases with available data except one. No significant differences were observed among the treatments regarding event type, outcomes, age or sex distribution, severity criteria, or event duration.

Romosozumab was associated with a significantly higher risk of CV SARs [ROR 22.85, CI (13.72-38.05)].
Conclusion: Our EudraVigilance data analysis revealed that RMZ was associated with a substantially increased risk of CV events (1.47% of all SARs). These findings highlight the need to address this safety issue and consider CV risks in Osteoporosis management. Ensuring the accurate reporting of SARs is essential to enhance the reliability of real-world data, ultimately supporting better-informed decisions in clinical practice.

222 - CLINICAL AND PROGNOSTIC DIFFERENCES BETWEEN MALE AND FEMALE PATIENTS WITH GIANT CELL ARTERITIS

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Introduction: Giant cell arteritis (GCA) is a systemic vasculitis affecting adults over 50, with varied clinical manifestations. Recent studies suggest that women tend to present stronger inflammatory responses and relapse rates, while men are more prone to cranial ischemic events. Recognizing these differences may help identify patients needing closer monitoring or early glucocorticoid (GC)-sparing strategies.
Objectives: To investigate how sex impacts disease phenotype, treatment response, and long-term outcomes in patients with GCA.
Methods: Retrospective cohort of GCA patients diagnosed at a tertiary rheumatology department (Jan 1999–Mar 2025), stratified by sex. Categorical and continuous variables were compared using appropriate tests. Multivariable logistic and linear regression assessed associations with clinical and laboratory parameters. Time-to-event outcomes (relapse, GC suspension, mortality) were evaluated using Kaplan–Meier and Cox regression.
Results: A total of 231 patients were included (64.5% female, n=149), mean age at diagnosis of 74.2±8.5 years. Compared to males, females more often had jaw claudication (43.0% vs 29.3%, p=0.029), higher ESR (90.9±27.1 vs 70.7±32.5, p<0.001), lower haemoglobin (11.2 vs 12.5 g/dL, p<0.001), and higher platelet counts (394.0 vs 317.0×10^9/L, p=0.009) (Table 1). In multivariable models, female sex remained associated with higher ESR (B –9.72, p<0.001) and lower haemoglobin (B 0.77, p<0.001), but not with jaw claudication (OR 1.26, p=0.519) or platelet count (B –0.79, p=0.252).

Median follow-up was 36.0 months (IQR 66.3). Females had more relapses (74.4% vs 54.0%, p=0.005), higher cumulative GC doses (12,173.2±7432.4 vs 9730.3±7411.2 mg, p=0.040), but also longer follow-up (46.5 vs 23.5 months, p<0.001). When restricting follow-up to the first two years, sex differences in GC dose (p=0.366) and relapse frequency (p=0.063) were no longer significant. Multivariable models confirmed no sex differences in GC cumulative dose (B –494.0, p=0.559) or relapse risk (OR 2.83, p=0.063) during this period. Kaplan–Meier analysis showed no sex differences in time to first relapse or GC suspension (p=0.846 and p=0.426; Figure 1A–B). However, in Cox regression adjusted for age, GC cumulative dose, relapse frequency, and follow-up time, male sex was associated with a lower hazard of GC suspension (HR 0.45, p=0.004), reflecting earlier GC discontinuation in females. Time to first relapse remained non-significant (HR 0.80, p=0.357). Seventy-three patients died (38 women, 35 men). Mortality was higher in males overall (42.7% vs 25.5%, p=0.004) and within two years (20.7% vs 7.4%, p=0.001). Kaplan–Meier analysis confirmed a significant sex difference in overall survival (p=0.034; Figure 1C). However, in the adjusted Cox model, sex was no longer an independent predictor of mortality (HR 1.50, p=0.359).
Conclusion: In this cohort, we confirmed sex-related differences in GCA inflammatory profile, with women showing higher ESR and lower haemoglobin at diagnosis. Relapse frequency and GC cumulative exposure were higher in females, aligning with previous reports, but these differences lost significance after adjusting for follow-up time. Males were less likely to discontinue GC, a novel finding suggesting differences in treatment response. While male sex has been associated with higher mortality in prior studies, our data confirm this trend descriptively but not independently. These findings reinforce sex as a factor in disease expression and treatment course, though not in major long-term outcomes like relapse or survival.

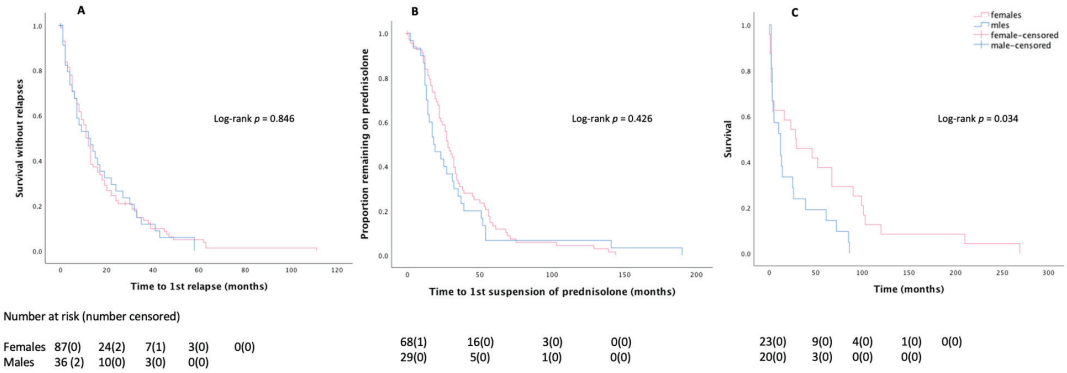
222 – TABLE 1. Patient characteristics stratified by sex at baseline during follow-up.

	All (n=231)	**Female** (n=149)	**Male** (n=82)	***p* value***
Disease baseline				
Age at diagnosis (years), mean ± SD	74.2 ± 8.5	74.4 ± 8.4	73.9 ± 8.9	0.710
Age at symptom onset (years), mean ± SD	73.8 ± 8.5	73.9 ± 8.4	73.4 ± 8.9	0.672
Disease characteristics, n (%)				
Constitutional symptoms	151 (65.4%)	100 (67.1%)	51 (62.3%)	0.469
Polymyalgia rheumatica	106 (45.9%)	69 (46.3%)	37 (45.1%)	0.811
New-onset headache	170 (73.6%)	115 (77.2%)	55 (67.1%)	0.116
Jaw claudication	88 (38.1%)	64 (43.0%)	24 (29.3%)	**0.029**
Limb claudication	4 (1.7%)	3 (2.0%)	1 (1.2%)	1.000
Visual abnormalities[1]	96 (41.6%)	61 (40.9%)	35 (42.7%)	0.835
Stroke/transient ischemic attack	22 (9.5%)	11 (7.4%)	11 (13.4%)	0.145
Abnormalities on temporal artery examination[2]	100 (43.4%)	63 (42.3%)	37 (45.1%)	0.513
GCA phenotype[3]				
• Cranial	153 (69.9%)	97 (69.3%)	56 (70.9%)	0.171
• Large vessel	30 (13.7%)	16 (11.4%)	14 (17.7%)	0.171
• Mixed	36 (16.4%)	27 (19.3%)	9 (11.4%)	0.152
Laboratory findings				
ESR (mm/h), mean ± SD	83.0 ± 30.6	90.9 ± 27.1	70.7 ± 32.5	**<0.001**
CRP (mg/dL), mean ± SD	4.94 ± 6.0	7.1 ± 6.4	6.1 ± 5.2	0.268
Haemoglobin (g/dL), median (IQR)	11.6 (2.5)	11.2 (2.2)	12.5 (2.1)	**<0.001**
Platelets (/uL), median (IQR)	361.0 (190.0)	394.0 (201.0)	317.0 (179.5)	**0.001**
Disease follow-up				
Follow-up time (months), median (IQR)	36.0 (66.3)	46.5 (74.0)	23.5 (41.0)	**<0.001**
Treatment				
IV methylprednisolone pulses, n (%)	78 (33.8%)	48.0 (32.2%)	30 (36.6%)	0.610
Cumulative doses of prednisolone (or equivalent), mean ± SD				
• Total	11273.6 ± 7496.5	12173.2 ± 7432.4	9730.3 ± 7411.2	**0.004**
• At one year	8350.7 ± 3719.1	8501.9 ± 3216.5	8058.0 ± 4562.9	0.554
• At two years	10371.4 ± 4268.3	10645.5 ± 3782.9	9778.6 ± 5174.2	0.366
Time to 1st suspension of prednisolone (months), median (IQR)	27.0 (31.0)	28.0 (32)	18.5 (25)	0.170
Patients who were treated with DMARDs, n (%)	143 (61.9%)	97.0 (65.1%)	46 (56.1%)	0.338
• Methotrexate	114 (49.4%)	77.0 (79.4%)	37 (45.1%)	0.608
• Tocilizumab	35 (24.5%)	25.0 (25.8%)	10 (21.7%)	0.085
Outcomes				
Patients who relapsed, n (%)	124 (53.7%)	90 (60.4%)	34 (41.5%)	**0.005**
Time to first relapse (months), median (IQR)	11 (18.5)	11 (14.0)	8.5 (32.0)	0.508
Number of relapses within the first two years, mean ± SD	0.7 ± 0.9	1.0 ± 1.1	0.5 ± 0.7	0.063
Deaths, n (%)	73 (31.6%)	38 (25.5%)	35 (42.7%)	**0.004**
Deaths within the first two years, n (%)	28 (12.1%)	11 (7.4%)	17 (20.7%)	**0.001**
Time from diagnosis to death (months), median (IQR)	14.0 (64.0)	67.0 (72.0)	43.5 (65.0)	0.249

CRP: C-Reactive protein, DMARDs: Disease-modifying antirheumatic drugs; ESR: Erythrocyte sedimentation rate, GCA: Giant cell arteritis; IQR: Interquartile range; IV: Intravenous; SD: Standard deviation.

1-Transient amaurosis, permanent vision loss or arteritic anterior ischaemic optic neuropathy; 2- Tenderness or thickness of the artery or reduced or absent pulse; 3- According to ultrasound results.

*Categorical variables were compared using chi-square or Fisher's exact tests, and continuous variables with t-tests or Mann–Whitney U tests.



205 – Figure 1. Kaplan–Meier survival curves comparing male and female patients with giant cell arteritis (A) time to first relapse, (B) time to first suspension of prednisolone, and (C) mortality