Amyloid deposition in rheumatoid arthritis patients

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To the Editor,

Secondary amyloidosis or systemic amyloidosis A (AA) is one of the most feared complications of rheumatoid arthritis. Its development is a lengthy process associated with longstanding inflammation and results from tissue deposition of the acute phase reactant SAA (serum amyloid A)[1]. In RA patients, 5 to 29%[2,3] will acquire AA amyloidosis depending upon severity and duration of disease as well as the method used for amyloid search. Some authors claim that the prevalence of amyloidosis AA is declining in recent years due to better treatment by the development of powerful drugs[2]. Others do not agree[4].

Although longstanding inflammation is necessary for the development of AA amyloidosis, it may not be sufficient, as different populations show diverse prevalence of its appearance. More over, some patients have high degree of inflammation for a long time and never develop amyloidosis. Life style, genetic background determinating SAA subtypes, differences in RA treatment and concurrent diseases may explain such differences[2].

We studied retrospectively the prevalence of amyloid deposition through abdominal subcutaneous fat tissue aspiration in RA patients in our service for the past 5 years. This was done in order to know its prevalence in Southern Brazilian patients and its relationship with RA clinical and autoantibody profile.

The sample was formed by 150 RA patients (132 females and 18 males) with at least 4 of American College Rheumatology classification criteria for AR[5] with mean age of 53.01±12.20 years and mean disease duration of 11.89 ± 8.36 years.

The prevalence of positive amyloid biopsy was of 24/150 (16%). None of these patients had any proteinuria, hematuria or any other clinical evidence of amyloidosis. The comparison of demographic data, an-

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TABLE I. COMPARISON OF 150 RA PATIENTS POSITIVE AND NEGATIVE FOR AMYLOID DEPOSITION IN ABDOMINAL SUBCUTANEOUS FAT TISSUE

	Amyloid positive n=24	Amyloid negative n=126	p
Gender	22 females;2 males	110 females;16 males	0,73
Mean age (years)	49.71 ± 13.53	53.85 ± 11.88	0,14
Mean disease duration (years)	13.92 ± 8.03	11.54 ± 8.34	0,12
Rheumatoid Nodules	2/20 (10%)	15/113 (13.2%)	1,00
Interstitial lung disease	3/19 (15.7%)	15/99 (15.1%)	0,56
ANA	5/20 (25%)	29/115 (25.5%)	1,00
RF	19/24 (79.16%)	88/126 (69.8%)	0,35
Anti CCP	10/12 (83.3%)	48/62 (77.4%)	1,00
Sedimentation rate (mean value)	24.94 ± 22.24	33.95 ± 32.53	0,44
C reactive protein (median value)	0.61	1.180	0,28
Mean DAS 28 (4v) (7)	3.40	3.55	0,58
HAQ (6) (mean values)	1.79 ± 0.94	1.19 ± 0.76	0,044

ANA= antinuclear antibody; CCP= cyclic citrullinated peptide; DAS= disease activity score; HAQ= health assessment questionnaire; RF= rheumatoid factor.

tibody and clinical profile of positive and negative patients for amyloid deposition is seen in Table I.

Analysing the table, the only item that will help the clinician suspect of amyloid deposition is a worse functional status. These results showed that patients with and without amyloid deposition are almost equal from clinical and serological point of view so an active search for this complication has to be done in order to make an early diagnosis. Subcutaneous fat tissue aspiration is an easily performed and useful screening method[8].

Until recently, doing a precocious diagnosis of amyloid deposition did not change patient's prognosis. Nowadays, medications such as anti TNF-a[9] and anti interleukin 6[10] have been shown to arrest and even to reverse amyloidosis. In these patients an earlier indication for this kind of drugs may prevent the development of full blown amyloidosis and improve their prognosis.

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