

Table 1
Genetic Characteristics of Patients with Cutaneous Lesions Induced.

ISL	N.p. Total	N.p. HLA-B27+	N.p. HLA-CW6+	N.p. HLA-DR1+	N.p. HLA-DR4+	N.p. HLA-DR7+	LCE 3C-LCE 3B-del D+/D+	LCE 3C-LCE 3B-del D+/D-
Skin psoriasis	7	4	1	1	0	3	3	4
Alopecia areata	3	1	1	0	0	1	1	2
Cutaneous lupus	2	0	1	0	0	1	0	2
Eczema	1	1	0	0	0	1	0	1
Hydradenitis	1	1	0	0	0	0	0	1
Erythema multiforme	1	0	1	0	1	0	0	1
Total	15	7	4	1	1	6	4	11

LCE 3B-3C-LCE del, deletion of the late cornified envelope; ISL, induced skin lesions; N.p., number of patients; +, positive; D+, deleted; D-, non-deleted.

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Conflict of Interest

The authors have no conflict of interest to state.

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Usefulness of Ultrasound in Jaccoud's Arthropathy. A Case Report[☆]



La ecografía en la artropatía de Jaccoud. A propósito de un caso

To the Editor,

Joint involvement is one of the initial manifestations in most patients with systemic lupus erythematosus (SLE); ranging from joint pain to severe deforming arthritis.¹ Within the deforming types, non erosive forms, such as *Rhupus* and others such as Jaccoud's arthropathy (JA),² non-erosive in principle, occurs in 10%–35% of patients with SLE.³

Ultrasound has proven superior to clinical examination in detecting joint and tendon inflammatory activity in patients with SLE.² We report a patient with SLE and JA in which this technique was useful in the assessment of disease.

The patient is a 40-year-old woman from Honduras diagnosed with SLE 4 years prior and, has presented during her evolution, joint pain and arthritis of small proximal joints of the hands, wrists,

knees and elbows; scarring alopecia; Raynaud's phenomenon and subacute cutaneous lupus erythematosus lesions. She had positive antinuclear antibodies (1/1280), anti-dsDNA, anti-Sm, anti-RNP and anti-CCP (high titers); anemia of chronic diseases and complement consumption. From the onset of the disease she has been treated with hydroxychloroquine, methotrexate and prednisone.



Fig. 1. Left hand of the patient with Jaccoud's arthropathy where ulnar deviation of the 5th finger and swan neck deformity of fingers 2–5 is appreciated.

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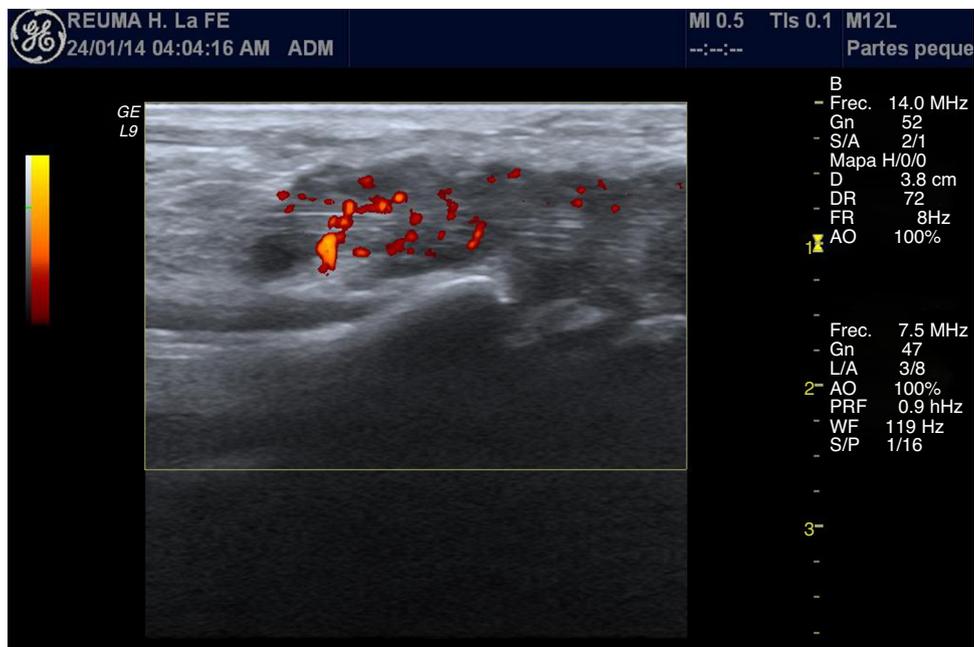


Fig. 2. Longitudinal view of the palmar radiocarpal joint, in which the flexor tendons of the wrist are appreciated. A hypoechoic thickening of the synovial sheaths of the tendons is highlighted, corresponding to tenosynovitis with marked synovial hypertrophy and a pathological Doppler signal, which shows an increase of normal vasculature.

Currently, she is painless and has non reducible deformity of the hands, bilaterally, with a positive score (more than 5 points) for JA in the index proposed by Spronk et al.,³ with predominating “goose-neck” deformities, evident ulnar deviation in some fingers and unilateral thumb ‘Z’ deformity (Fig. 1). Despite being asymptomatic, C-REACTIVE protein (CRP) and erythrocyte sedimentation rate remained persistently positive, so an ultrasound of the wrists and hands was requested, after ruling out infections or other concurrent processes.

Synovial hypertrophy was observed in the radiocarpal and midcarpal joints bilaterally, according to the *Outcome Measures in Rheumatoid Arthritis Clinical Trials* (OMERACT). It is described as an abnormal non-movable, intraarticular hypoechoic tissue, poorly compressible,⁴ 2nd stage,⁵ because it reached the top of the periarticular bone and did not extend to the diaphysis. The power Doppler signal was 2⁶ as confluent vascular signals were observed in the synovial area. Flexor tenosynovitis was also shown on the wrist, corresponding to a hypoechoic thickening of the synovial sheaths of the tendons⁵ with bilateral pathological Doppler (Fig. 2) signal corresponding to an increase of normal vascularization. Similarly, tenosynovitis was seen in all finger flexors with no evidence of bone erosions.

JA is a chronic deforming joint affection,^{7,8} associated with different diseases⁹ which usually affects the metacarpophalangeal and proximal interphalangeal joints, wrists and knees.¹ It leads to deformities that mimic rheumatoid arthritis (RA), predominantly dislocations, although with little pain and little functional impairment. Alterations are reducible⁹ and not the typical radiographic erosions seen in RA,² even in patients with longstanding MR studied disease.¹

We hypothesize that tenosynovitis is a typical primary lesion¹ and deformities result from the involvement of ligaments and tendons, rather than an erosive arthritis with *pannus* formation, as in RA.²

In our patient, antiphospholipid antibodies were negative, contrary to the previously suggested association⁹ and she showed no renal involvement. There is a negative association between JA and kidney damage, despite the prevalence of anti-dsDNA antibody, suggesting a better prognosis in these patients.⁹

The acute phase reactants and ultrasound abnormalities point to sustained synovial inflammation, consistent with previous publications³ in which higher CRP levels were observed in patients with JA compared with patients without it, despite a similar clinical situation and treatment.

We agree with other groups, on the importance and value of ultrasonography in patients with SLE² although it is still not validated as an imaging technique for the assessment of lupus. In our case, it allowed us to detect elementary lesions suggestive of active inflammation, although the patient remained clinically asymptomatic. These alterations detected by ultrasound show, with great probability, subclinical tendon involvement due to the disease, similar to what has been suggested in other studies in patients with lupus, but without reference to JA.¹⁰ Nevertheless, more studies are needed to assess the need for a screening ultrasound or other imaging techniques in all patients with JA, or only those who remain with elevated acute phase reactants, looking for signs of inflammatory activity.

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Rheumatoid Arthritis, a New Focus on Cardiovascular Risk*



Artritis reumatoide, un nuevo enfoque del riesgo cardiovascular

Dear Editor,

Patients with rheumatoid arthritis (RA) have a greater prevalence of traditional risk factors and 68% more risk of developing a myocardial infarction¹ than the general population, with this risk persisting even when the analysis is adjusted for traditional coronary risk factors.^{1,2} EULAR³ recommendations for the evaluation of cardiovascular risk in subjects with RA propose the application of risk evaluation methods such as, for example, the Framingham type. On the other hand, EULAR recommends special attention to subjects with long-standing RA (over 10 years), rheumatoid factor or anti-CCP antibody positivity as well as those with extra articular manifestations.³

Rheumatologists, in their daily clinical practice, must perform different indices: diagnostic, classification, disease activity, radiological progression, risk for fracture due to frailty (FRAX and others), patient quality of life, etc., to which we add the evaluation of coronary risk.

The 2013 ACC/AHA Guidelines on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines⁴ were recently published, which propose “extensive and consistent” evidence on the benefit of the use of statins in order to reduce the cardiovascular risk in subjects with LDLc over 70 mg/dL. Four groups of patients are identified in subjects who would benefit from the use of statins: (1) subjects with clinical cardiovascular disease; (2) subjects with LDLc ≥ 190 mg/dL; (3) diabetics between 40 and 75 years of age with LDLc between 70 and 189 mg/dL and no clinical cardiovascular disease, and (4) subjects with no clinical cardiovascular disease or diabetes, with LDLc between 70 and 189 mg/dL and a 10 year estimated risk of ≥ 7.5%.⁴

Because RA has a cardiovascular risk similar to diabetes mellitus,⁵ that the use of statins provide a modest but significant anti-inflammatory effect,⁶ and that the use of anti-inflammatory drugs (coxibs or non-coxibs) is associated to a greater coronary risk,⁷ we propose that subjects with RA and no clinical cardiovascular disease, with LDLc between 70 and 189 mg/dL and no upper limit for age be considered in group 3 of the four above-mentioned groups, which implies the use of statins in “moderate intensity”⁴ for most of the patients with RA; however, in subjects

with long-standing RA who are rheumatoid factor/anti-CCP positive or have extra articular manifestations who comply with two or more of these criteria, the clinician might consider the use of “high intensity” statin treatment.⁴ These recommendations might be extended to subjects with spondyloarthritis, including psoriatic arthritis.⁸

The use of statins modifies the plasma lipid profile and the cardiovascular risk of subjects with inflammatory arthritis in a similar way than in patients without inflammatory arthropathy and this reduction of extended to RA, spondylitis and psoriatic arthritis⁹; even in subjects with RA who are using statins, the interruption in treatment is associated to an increase in the risk of cardiovascular mortality.¹⁰

In spite of the benefits in cardiovascular risk that, in our judgment, would be provided by statins in subjects with inflammatory arthritides, the clinician must always take into account the possibility of myopathy and especially liver toxicity that may occur in subjects who frequently take other hepatotoxic drugs.

In conclusion, we recommend that all patients with inflammatory arthritis, especially RA, over 40 years of age, with LDLc between 70 and 189 mg/dL and no cardiovascular disease receive statins at a moderate dose and those patients with a particularly high risk (two or more of these conditions: long-standing RA, rheumatoid factor/anti-CCP positivity, extra articular manifestations) receive statins in a high intensity regimen.

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