

but some patients develop erosive arthritis similar to RA, with it associating with calcinosis and acroosteolysis.⁸ However, the development of JA is quite uncommon, with only 4 cases reported to date,^{2–4} so it is considered that it may correspond more to the coexistence of idiopathic JA than a manifestation of SS.³ It has been suggested that JA in SLE associated with inflammatory activity due to prolonged or recurrent low degree swelling of the synovial membrane and joint capsule, and ligamentous laxity cause an imbalance of muscle forces. However, in cases of associated JA and SS there is no clinical or imaging^{2,3} inflammation evident, so other mechanisms could be involved, including pericapsular and tendon fibrosis.¹ A symmetrical erosive arthritis of small joints, often RF positive, has been described in PBC, which can be indistinguishable from RA. Up to 31% of PBC cases develop arthritis, characterized by a non-deforming asymmetric affection (although cases have been reported with deforming/erosive arthritis), negative for RF and anti-CCP, and recently it has been shown that it has a special histopathology regarding synovial infiltration predominantly by B lymphocytes and plasma cells.⁷ However, JA is not described in these patients. Pyrophosphate deposition disease has also been associated with JA,^{9,10} so that may have contributed to its development in our patient because there was evidence of chondrocalcinosis of the right wrist, but not in previous episodes of arthritis at that level. In conclusion, the development of JA in the RS seems to be rather incidental, because it is not properly a joint manifestation of any of the entities that make up this syndrome.

References

1. López Longo FJ. Artropatía de Jaccoud: algo más que lupus. *Semin Fund Esp Reumatol*. 2011;12:36–41.

2. Bradley JD, Pinals RS. Jaccoud's arthropathy in scleroderma. *Clin Exp Rheumatol*. 1984;2:337–40.
3. Spina MF, Beretta L, Masciocchi M, Scorza R. Clinical and radiological picture of Jaccoud arthropathy in the context of systemic sclerosis. *Ann Rheum Dis*. 2008;67:728–9.
4. Spinel N, Montenegro P, Rondón F, Restrepo JF, Iglesias Gamarra A. Artropatía de Jaccoud y fibrosis pulmonar en síndrome CREST. *Rev Colomb Reumatol*. 2010;17:64–8.
5. Rigamonti C, Bogdanos DP, Mytilinaiou MG, Smyk DS, Rigopoulou EI, Burroughs AK. Primary biliary cirrhosis associated with systemic sclerosis: diagnostic and clinical challenges. *Int J Rheumatol*. 2011;2011:976427.
6. Reynolds TB, Denison EK, Frankl HD, Lieberman FL, Peters RL. Primary biliary cirrhosis with scleroderma, Raynaud's phenomenon and telangiectasia. *New syndrome*. *Am J Med*. 1971;50:302–12.
7. Smith MD, Walker JG, Ahern MJ, Roberts-Thomson PJ. Inflammatory arthritis in a patient with primary biliary cirrhosis: B cell mediated synovitis. *J Rheumatol*. 2010;37:212–4.
8. Avouac J, Clements PJ, Khanna D, Furst DE, Allanore Y. Articular involvement in systemic sclerosis. *Rheumatology (Oxford)*. 2012;51:1347–56.
9. Palazzi C, D'Amico E, De Santis D, Petricca A. Jaccoud's arthropathy of the hands as a complication of pyrophosphate arthropathy. *Rheumatology (Oxford)*. 2001;40:354–5.
10. Hajri R, Rezgui L, Ben Miled M, Chekili S, Laatar A, Kassab S, et al. Jaccoud arthropathy and pyrophosphate arthropathy: a rare association. *Tunis Med*. 2004;82:1048–51.

Walter Alberto Sifuentes Giraldo,* Marta Grandal Platero, Carlos de la Puente Bujidos

Servicio de Reumatología, Hospital Universitario Ramón y Cajal, Madrid, Spain

* Corresponding author.

E-mail address: albertosifuentesg@gmail.com

(W.A. Sifuentes Giraldo).

The Lag Time Between Onset of Symptoms, Medical Encounter, and Initiation of Disease Modifying Antirheumatic Drugs in Patients With Rheumatoid Arthritis[☆]

Tiempo entre comienzo de síntomas, acudir al médico e inicio de fármacos modificadores de la enfermedad en pacientes con artritis reumatoide

To the Editor,

Rheumatoid arthritis (RA) is an autoimmune disease and most patients have a chronic, fluctuating progression. If untreated, it leads to progressive joint deformity, disability and premature death. The onset of early treatment with disease modifying drugs (DMARD) reduces disability at 5 years.¹ The objective of treatment in patients with recent onset RA is the suppression of disease activity before joint damage is established; this justifies the importance of opportune therapeutic intervention.^{2,3} Patients treated early (during the first three months since disease onset) have a better prognosis and may go into disease remission.

Studies performed to evaluate time between disease onset and time of diagnosis and start of adequate treatment are scarce^{4–9} and none exist in Mexico. The objective of the study was to evaluate the time since the beginning of disease and the visit to the family physician, the time since this and the referral to the rheumatologist and the time to onset of DMARD treatment.

We included, from January to December 2010, adult patients with clinical manifestations of RA and no previous evaluation by a rheumatologist or DMARD treatment. Patients were sent to different family physicians or from the internal medicine specialist to the rheumatologist of a regional General Hospital of the Instituto Mexicano Seguro Social (IMSS). We defined RA based on the 1987 criteria proposed by the American College of Rheumatology. We evaluated the following timelines: time from onset of symptoms to the visit to the family physician; time from disease onset to the first visit to the rheumatologist and time since disease onset and DMARD treatment onset.

Mean age±SD of the 98 patients was 38±9 years; 85% were female; 49 had an RA disease progression of 1–6 months, the diagnosis established by the family physician was RA in 79% of patients and the mean number±SD of visits the patient received before referral to rheumatology was 6.6±5.8). In 19, 33, 23 and 24% of patients, the onset of DMARD treatment was 1–3, 4–6, 7–12 and ≥13 months after RA symptom onset, respectively. The mean time for the patient to receive medical attention was 2.9 months, and for referral to Rheumatology from primary care was 6.6 months. For DMARD treatment onset it was 9.9 months.

Our results show that only in 19% of patients was DMARD started in the first three months after disease onset and the delay in the prescription of DMARD was mainly due to the delay in referral from family medicine to the rheumatologist. Studies performed in the past 2 decades which evaluate the onset of DMARD treatment in patients with early RA, performed in the US,⁴ Spain,⁵ Canada,⁹ the United Kingdom,⁸ the Middle East,⁶ and in European countries,⁷ show that the mean time since the onset of disease and the onset of DMARD treatment ranges from 6 to 18 months, similar to our findings (mean 11 months). This indicates that the diagnosis of RA after

[☆] Please cite this article as: Zonana Nacach A, Campaña-Parra A, Santana-Sahagún JE. Tiempo entre comienzo de síntomas, acudir al médico e inicio de fármacos modificadores de la enfermedad en pacientes con artritis reumatoide. *Reumatol Clin*. 2014;10:131–132.

the onset of symptoms is delayed and, therefore, the objective of starting treatment early is not achieved in most cases. Less than 30% of RA patients receive DMARD treatment in the first 3 months.^{7,9,10} It is necessary to implement measures that act on early diagnosis and treatment of RA, through the diffusion of knowledge relating to the disease in the general population, advertising campaigns as well as an increase in the level of knowledge regarding RA in primary care physicians.

References

1. Wiles NJ, Lunt M, Barret ME, Bukhari M, Silman JA, Symons PM, et al. Reduced disability at five years with early treatment of inflammatory polyarthritis. *Arthritis Rheum.* 2001;45:1033–42.
2. Van der Heide A, Jacobs JW, Biglisma WA, Heurkens AH, van Booma-Frankfort C, van der Veen MJ, et al. The effectiveness of early treatment with second line antirheumatic drugs: a randomised controlled trial. *Ann Intern Med.* 1996;124:699.
3. Mottonen R, Hammonnen P, Leirisalo-Rapo M, Nissila M, Kauntiamen H, Korpela M, et al. Comparison of combination therapy with single-drug therapy in early rheumatoid arthritis: a randomised trial. *Lancet.* 1997;350:309–18.
4. Chan A, Felson TD, Yood AR, Walker MA. The lag time between onset of symptoms and diagnosis of rheumatoid arthritis. *Arthritis Rheum.* 1994;37:814–9.
5. Hernández-García C, Vargas E, Abásolo L, Lajas C, Bellajdell B, Morado IC, et al. Lag time between onset of symptoms and access to rheumatology care and DMARD therapy in a cohort of patients with rheumatoid arthritis. *J Rheumatol.* 2000;27:2323–8.
6. Zafar S, Badsha H, Mofti A, Delosantos A, Altares J, Matudio G, et al. Efforts to increase public awareness may result in more timely diagnosis of rheumatoid arthritis. *J Clin Rheumatol.* 2012;18:279–82.
7. Raza K, Stack R, Kumar K, Filer A, Detert J, Bastian H, et al. Delays in assessment of patients with rheumatoid arthritis: variations across Europe. *Ann Rheum Dis.* 2011;182:2–5.
8. Kumar K, Daley E, Carruthers DM, Situnayake D, Gordon C, Grindulis K, et al. Delay in presentation to primary care physicians is the main reason why patients with rheumatoid arthritis are seen late by rheumatologists. *Rheumatology (Oxford).* 2007;46:1438–40.
9. Jamal S, Alibhai SM, Badley EM, Bombardier C. Time to treatment for new patients with rheumatoid arthritis in a major metropolitan city. *J Rheumatol.* 2011;38:1282–8.
10. Van der Linden MP, le Cessie S, Raza K, van der Woude D, Knevel R, Huizinga TW, et al. Long-term impact of delay in assessment of patients with early arthritis. *Arthritis Rheum.* 2010;62:3537–46.

Abraham Zonana Nacach,^{a,*} Alfredo Campaña-Parra,^b Jesús Ernesto Santana-Sahagún^b

^a *Unidad de Investigación Clínica y Epidemiología, Hospital General Regional N.º 20, Instituto Mexicano del Seguro Social, Tijuana, Baja California, Mexico*

^b *Consulta externa Reumatología, Hospital General Regional N.º 20, Instituto Mexicano del Seguro Social, Tijuana, Baja California, Mexico*

* Corresponding author.

E-mail address: zonanaa@yahoo.com (A. Zonana Nacach).

Clinical Typology of Chronic Fatigue Syndrome: Classificatory Hypothesis[☆]

Tipología clínica del síndrome de fatiga crónica: hipótesis clasificatoria

Primary (or pure) chronic fatigue syndrome (pCFS) is a complex and severe chronic and disabling disease of unknown causes, excluding secondary chronic fatigue syndrome (sCFS) related to some other medical condition. It is characterized by intense fatigue in addition to cognitive, autonomic, neuroendocrine, immunoallergic and musculoskeletal symptoms, which are of recent appearance and that cannot be explained by other clinical reasons, lasting for at least 6 months, is non-remitting significantly with rest and which worsens with physical or mental activity, with very slow recovery and a reduction of >50% of activities of daily living previously performed by the patient.^{1–4} It is diagnosed according to the 1994 Fukuda criteria,¹ the Canadian consensus document published in 2003² or, more recently, the international consensus criteria of 2011; with the name of myalgic encephalomyelitis that offers a review on its physiopathology, symptoms and treatment.³ Prevalence is estimated to be between 0.5% and 2.5% of the general population.^{1–3,5} In spite of it being recognized as a disease by the WHO since 1989, and classified with the code G93.3 in the ICD-10,^{5,6} and that evidence accumulated from different fields during the past 2 decades, it is possible that pCFS is still largely unknown by most health professionals.

Due to the great heterogeneity in its clinical expression and the lack of standardized instruments to order its different symptoms (according to greater or lesser frequency) and clinical presentation (distinguishing between pCFS, sCFS and Idiopathic CF), we propose a classification into clusters: I, II, IIIa, IIIb and IIIc, subgrouping the main clinical conditions in a consecutive series of 199 patients (Table 1), seen in a period of time between June 2010 and February 2013 at the Chronic Fatigue Specialized Hospital Unit of the Camp de Tarragona belonging to the Hospital Universitario Joan XXIII. 84% of patients (n=167) were women (5:1). Mean age at symptom onset was 41.5 years (range 9–76). The onset of symptoms was insidious in 72.8% of patients and 71.3% had a progressive evolution of disease. At the moment of interview, 51.7% (n=103) were unemployed (65 lost their job while 38 were disabled). 8% (n=16) were retired. The pCFS criteria are valid for the diagnosis in the clinical daily practice independently of other associated criteria.^{6–8} Having a classification of patients with CFS will allow us to identify more homogeneous groups of patients, candidates both for more individualized diagnostic and therapeutic guidelines and which allow for better expectations. In order to correctly classify a patient with CFS, we recommend a multidisciplinary approach. In first place, the CFS diagnosis must be confirmed (family physician, internal medicine specialist and/or rheumatologist), in order to then evaluate the existence of possible associated systemic diseases or chronic local processes that have a large differential diagnosis and, finally, to perform a precise diagnosis of the disease with a psychopathological background (psychologist and/or psychiatrist). Having a clear diagnosis in each one of these 6 areas, as well as the analysis of the chronology of symptoms or processes allows the inclusion of the patient into each one of the different classification subgroups.

[☆] Please cite this article as: Qanneta R, Fontova R, Poveda MJ, Castro S. Tipología clínica del síndrome de fatiga crónica: hipótesis clasificatoria. *Reumatol Clin.* 2014;10:132–133.