but some patients develop erosive arthritis similar to RA, with it associating with calcinosis and acroosteolisis.\textsuperscript{3} However, the development of JA is quite uncommon, with only 4 cases reported to date;\textsuperscript{4–5} so it is considered that it may correspond more to the coexistence of idiopathic JA than a manifestation of SS.\textsuperscript{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;\textsuperscript{4,5} inflammation evident, so other mechanisms could be involved, including pericapsular and tendon fibrosis.\textsuperscript{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.\textsuperscript{7} However, JA is not described in these patients. Pyrophosphate deposition disease has also been associated with JA,\textsuperscript{3,9,10} 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


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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 rheumatologist 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,\textsuperscript{8} Spain,\textsuperscript{2} Canada,\textsuperscript{9} the United Kingdom,\textsuperscript{6} the Middle East,\textsuperscript{6} and in European countries,\textsuperscript{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
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. \cite{7,8,10}

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


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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, immunological 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. It is diagnosed according to the 1994 Fukuda criteria, the Canadian consensus document published in 2003 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. Prevalence is estimated to be between 0.5% and 2.5% of the general population. 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, 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.

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.