Editorial

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Systemic lupus erythematosus is one of the main chronic rheumatic diseases both in adults and in childhood. In this issue of the ARP there are three papers[1-3] concerning several different aspects of this multifactorial, multi-systemic and potentially severe disease.

Musculoskeletal symptoms are the most prevalent symptoms, being present in childhood cases in 87.5% of cases[1]. For this reason SLE is considered worldwide as a ***rheumatic disease***, as everybody knows. Of course its kidney, pulmonary, cardiac, neurologic, cutaneous manifestations are also frequent and, all of them can be severe but, nonetheless, it is appropriately considered a rheumatic disease.

As we all know very well too, Rheumatology is the subspecialty of Internal Medicine, that collects the knowledge to treat these patients, working in team with all the other medical specialties needed specifically in each clinical situation.

Coming back to the current issue of the Acta Reumatol Port., SLE in the pediatric series showed results similar to the ones found by others, in Portugal[4] and elsewhere[5,6], perhaps with less frequent severe neurologic manifestations. In this series[1], near one third of the patients are in long-term clinical remission, some of them with some degree of damage; disease activity and damage were significantly, in the same series, higher in patients with neurolupus and/or lupus nephritis. As in adults, these two manifestations carry a poor prognosis[4-6]. But, even in these cases of poor prognosis, long-term remission can be achieved even more than 30 years after disease onset[7] and only 4 (7%) patients had severe neurologic manifestations[1].

In the interesting study comparing SLE activity and serum bilirubin levels[2], the authors found a negative correlation between indirect bilirubin (IB) levels and SLEDAI, being the values of IB also lower in patients in patients with higher ESR. So, higher IB seem to be somehow protective to SLE activity, and the authors speculate that this can be due to a compensatory mechanism against oxidative stress resulting from inflammatory activity in SLE[2]. The same protective effect of IB has been shown in the Framingham study against myocardial infarction and other cardiovascular events[8] and also in patients with Gilbert's disease that seem to have lower prevalence of ischemic heart disease than the control population[9]. It is interesting to notice that old time rheumatologists are also well aware of the improvement of Rheumatoid Arthritis symptoms during episodes of cholestatic jaundice, that may have a similar physiopathology.

In the cross sectional MRI study of psychiatric disorders in SLE and Behçet's disease[3] (BD), the main issue is the definition of a "neuropsychiatric disorder", since patients (older or younger) with a severe medical disease might well suffer from reactive depression, completely independent from the pathogenesis of the disease in itself.

If any depressive symptoms would account for the definition of a "neuropsychiatric disorder", than the prevalence of such disorders would increase dramatically also in the pediatric series[1] published in this issue. This definition is clearly responsible for the high prevalence of "neuropsychiatric disorders" both in SLE (56%), BD (26%) and in the control group, where it reaches more than 14% of the persons. The problem is completely different in patients with psychosis, present both in SLE and BD groups.

On the other hand, it is interesting to notice that the frequency of MRI abnormalities in BD patients with psychiatric manifestations was higher (with statistical significance) when compared to SLE patients with the same kind of manifestations[3], and that 1/3 of SLE patients with psychiatric manifestations showed no alterations on MRI, probably indicating the cases of reactive depression in the group.

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