Systemic Lupus Erythematosus and Vaccination against Hepatitis B

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Dear Sir,

Systemic lupus erythematosus (SLE) is an autoimmune disease of uncertain etiology. Several factors may act as the instigators of the onset or reactivation of such disease, namely ultraviolet rays, certain drugs, pregnancy, and viral infections [1]. The administration of vaccines has also been associated with outbreaks of the disease [2]. We present a case of SLE which had its onset after vaccination against hepatitis B, a fact which we have not found described in any of the literature on the subject.

A 43-year-old woman, without a particularly relevant medical background, presented edema on both legs for 6 weeks. About 2 weeks before the onset of the symptoms, she had been administered a first dose of recombinant antihepatitis B vaccine (Engerix®-B). Laboratory data showed: hemoglobin 10 g/dl, with a leukocyte and blood platelet count within a normal range; a certain deterioration of the renal function was observed, plasma creatinine 172 $\mu\text{mol/l}$, with proteinuria 1.8 g/24 h, and microscopic hematuria. Electrocardiogram and chest x-ray were normal. Haptoglobin and ferritin were within a normal range, and Coombs' test resulted negative. Immunological study revealed an increase in IgG to 2,070 mg/dl (normal = 800-1,700) with IgM and IgA within normal limits. The dosage of the complement showed a decrease in C3 to 17 mg/dl (normal = 50-120), C4 to 4 mg/dl (normal = 20-50) and CH50 to 2 U/ml (normal = 75-125). Circulating immune complexes were 2 $\mu\text{g/ml}$ (normal < 1.5). The antinuclear antibodies were positive at 1/1,280 homogeneous pattern, and anti-DNA at 1/320, with positive anti-Ro and anti-La antibodies. The serologic test for syphilis and rheumatic serology was negative. A renal biopsy was practiced which showed a diffuse proliferative glomerulonephritis with extracapillary proliferation and positive immunofluorescence for different immunoglobulins and complement factors. With the diagnosis of lupus nephritis type IV, treatment was initiated with low doses of prednisone (0.5 mg/kg/day) and cyclophosphamide (500 mg/m²) in monthly boluses. The subsequent evolution was favorable; renal function went back to normal, the level of antinuclear antibodies and immune complexes decreased, and anti-DNA antibodies were negative again.

The role of vaccination as an inducing factor in outbreaks of SLE has not been established up to now. Nevertheless, there are some facts that suggest that the administration of immunization to these patients may precipitate the clinical manifestations of the disease. Thus, the outbreak of

lupus vulgaris after vaccination with BCG has been reported [3]. On the other hand, in a study on the efficacy of vaccination against influenza in patients with SLE, the appearance of a certain deterioration of the renal affection was observed in 3 of the 202 patients studied [4]. Perhaps, such immunization implies an increase in the number of immune complexes, which is an ill-tolerated phenomenon in these patients since they suffer from a decrease in their capacity of clearing such products. In another recent study on vaccination against hepatitis B in patients with renal failure, the lack of response to immunization was observed in those patients with SLE, which was attributed to the widely-known dysfunction of B cells [5].

In our case, the appearance of the clinical manifestations of the disease after immunization suggests that such immunization may have had a precipitating role in the autoimmune phenomena. The implication of this vaccine in such phenomena has not been reported to the moment.

References

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