Dear Sir,

The beneficial effect of intravenous human gamma-globulin (IVIG) has been reported in patients with idiopathic membranous nephropathy [1, 2], Kawasaki syndrome [3] and lupus nephritis [4-6].

We report a patient diagnosed as having systemic lupus erythematosus (SLE), unresponsive to conventional therapy with methylprednisolone (MP) and cyclophosphamide (CA), who responded clearly to treatment with IVIG.

In February 1990, a 21-year-old woman with SLE and lupus nephritis (IV WHO Class) [7] was admitted to our Center because of fever and arthralgias. At admission she presented anemia, pleural effusion, ascites, nephrotic proteinuria, high titers of anti-dsDNA and low serum C3-C4 levels. Three months before entry, treatment with MP bolus (1 g MP/day intravenously for 3 consecutive days) followed by oral prednisone (2.5 mg/kg weight/day) and intravenous CA bolus had been instituted. She received a CA bolus every 3 months (0.5 g/m² body surface area). In spite of this treatment, the patient still had clinical and serological lupus activity. Blood and urine cultures were negative and no evidence of infectious disease was detected. In the following days, anemia and serositis increased. IVIG, 400 mg/kg/day, was administered for 5 days. By the third day after treatment, the patient showed a dramatic improvement. Fever disappeared and proteinuria decreased from 3.6 to 0.7 g/day. No side effects were detected. MP was maintained at a stable dose of 1 mg/kg/day for 1 month and later reduced to 0.5 mg/kg/day. Intravenous CA was administered every 3 months. Arthralgia, serositis, hypocomplementemia and elevated anti-dsDNA titers improved progressively. Sixteen months later, she remains asymptomatic with oral prednisone (0.3 mg/kg/day and intravenous CA (0.5 g/m² body surface area) every 3 months.

Although the mechanism of IVIG action is controversial [2, 6], it has been proposed that the high IgG serum levels obtained could produce an antibody excess state capable of dissociating IgG deposits. Furthermore, it may be related to the modulation of macrophage T-cell function, enhancement of suppressor T-cell function and elimination of circulating immunocomplexes through anti-idiotype antibodies [4].

Although more experience is needed to confirm the effectiveness of IVIG in SLE therapy, we think that high-dose IVIG could be useful in renal and extrarenal manifestations of SLE unresponsive to conventional therapy.
References

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