Relapse of Systemic Lupus erythematosus after Extracorporeal Immunoabsorption

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Dear Sir,

We report the case of a patient in whom an acute exacerbation of systemic lupus erythematosus (SLE) was associated with staphylo-coccal protein A immunoabsorption which had been performed in order to remove antibodies, against human leucocyte antigens. The patient was born in 1966 and developed SLE at the age of 13 years, presenting with arthralgia. Kidney failure developed at the age of 22 years, renal biopsy showing a segmental and proliferative glomerulonephritis. At the time dialysis was started, he suffered a relapse of SLE with pericarditis and arthralgia. The anti-double-stranded DNA (anti-dsDNA) antibody level was 39.1 (normal range < 7.0) IU/ml. He received oral prednisolone and monthly intravenous cyclo-phosphamide, with a good response. One year later, he received a cadaveric renal transplant. He remained antinuclear antibody positive at a titre of 1/160, but the anti-dsDNA levels were within the normal range. The transplant failed after 2 years, due to biopsy-proven chronic rejection.

On haemodialysis, he was well with an anti-dsDNA antibody level of 7.9 IU/ml. He was placed on the transplant waiting list, and immunosuppressive treatment was tailed off. Eighteen months later he had not received a transplant and had 95% panel-reactive antibodies. Therefore, he was considered for immunoabsorption treatment in order to remove antibody and to facilitate renal transplantation.

He received immunoabsorption (Citem 10 System; Excorim, Lund, Sweden) for a total of eight sessions over a total period of 4 weeks. The total plasma volume administered was 65.9 litres, ranging in each session

from 6.7 to 9.7 litres. Neither cyclophosphamide nor prednisolone were administered.

After 4 weeks of immunoabsorption, he developed malaise which was clinically diagnosed as a viral illness. Immunoabsorption was stopped. There was elevation of alkaline phosphatase (351 IU/l, normal range 30-120) and γ -glutamyl transferase (255 IU/l, normal range 5-55) levels. Extensive testing for viral infection was negative (in particular for hepatitis B and C and for cytomegalovirus). The level of anti-dsDNA antibodies was slightly elevated (13.9 IU/ml), but the symptoms were not reminiscent of his previous SLE. Twelve weeks after the last immunoabsorption treatment he developed an acute arthralgia typical of his previous SLE, and the anti-dsDNA level was 74.4 IU/l. He was treated with prednisolone and azathioprine, with improvement in his symptoms and a fall in anti-dsDNA levels. He remains

well on haemodialysis, does not at present wish to receive more immunoadsorption, and has panel-reactive antibody levels of 100%.

The relapse of SLE was temporally associated with a course of immunoadsorption treatment. His SLE had previously been in remission for over 3 years.

Although patients with severe acute SLE have been treated with immunoadsorption with apparently good results [1,2], there are no reports of patients in remission receiving such treatment. Antibody-mediated control of T cell activity is complex in SLE, and there are antibody networks that may be either sup-pressive or stimulatory [3,4]. Thus it is conceivable that perturbation of antibody or anti-idiotypic antibody networks by immunoadsorption could result in disease reactivation.

Alternatively, reactivation of SLE has been reported in association with viral infection [5], and our patient did suffer such an infection prior to SLE relapse. This infection may have been a consequence of the immunosuppressive effects of immunoadsorption.

In conclusion, we suggest that patients with SLE and possibly also with other autoimmune diseases should be monitored carefully for reactivation of disease whilst receiving immunoadsorption.

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