Multiple Autoimmune Diseases after Autologous Stem-Cell Transplantation

To the Editor: Hematopoietic stem-cell transplantation can be an effective treatment in patients with refractory systemic sclerosis.1 We report on a 19-year-old woman with systemic sclerosis who underwent CD34+-selected autologous hematopoietic stem-cell transplantation in March 2001. Before the transplantation, the physical and laboratory findings showed no evidence of any other autoimmune diseases. After written consent was obtained from the patient, CD34+ hematopoietic stem cells were transplanted according to a method used for systemic sclerosis.1 The dermal sclerosis improved immediately after transplantation, but thrombocytopenia and Graves’ disease developed.

In June 2005, the patient was admitted to the
Modified Rodnan Score

Anti-Scl70, IgG-aCL, and Anti-Sm (U/ml)

TGF-β1 and Interleukin-6 (ng/ml)

Interleukin-17 (ng/ml)

Ratio of Th1 to Th2

FOXP3 mRNA
hospital because of fever and edema. Blood tests revealed proteinuria (11.4 g per day) and new autoantibodies in the serum (Fig. 1A). On the sixth hospital day, paralysis developed on the left side as the result of a right cerebral infarction. Systemic lupus erythematosus with membranous-type lupus nephritis (Fig. 2) and the antiphospholipid-antibody syndrome were diagnosed; the patient was treated with prednisolone, warfarin, and cyclosporine. She is currently in clinical remission and is back at work.

During the early phases of immune reconstitution, residual lymphocytes undergo proliferation and expansion, a process controlled by regulatory T cells.\textsuperscript{2,3} These cells, defined by the phenotype CD4+CD25+FOXP3+, are important in the prevention of autoimmunity. Interleukin-17–producing helper T (Th17) cells may play a role in the induction of autoimmunity.\textsuperscript{4,5} In our patient, the level of serum interleukin-17, released mainly by Th17 cells, was elevated at the onset of the systemic lupus (Fig. 1B). Levels of FOXP3 messenger RNA, a marker of regulatory T cells, were reduced, suggesting a deficiency of such cells (Fig. 1C). The findings in our patient suggest a role of both regulatory T cells and Th17 in the development of systemic lupus.

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