Dr. Vlachoyiannopoulos replies

To the Editor:

Dr. Korkmaz raises the following issues: (1) the fluctuation of the aCL antibody titers, either spontaneous or modified by prednisone treatment, might be responsible for the upregulation of aPL antibodies after CYC treatment and not CYC itself; (2) his personal experience from 4 patients with SLE treated either with CYC or AZA alone or with prednisone is opposite to our own; (3) the diagnosis of some patients with APS may not be accurate; and (4) renal biopsy findings of a patient with APS nephropathy are missing.

Fluctuation of aCL antibody titers was observed in both treatment groups. However, high antibody titers (absorbance higher than the 99th percentile of 100 normal individuals in ELISA) obtained on 2 occasions 12 weeks apart were commonly detected in the CYC-treated group and constituted the criterion for seroconversion. Patients with high aCL titers tended to be permanently positive for aCL. The patients from both groups were evaluated for aCL antibodies with the same frequency over time. The reports suggested by Korkmaz describe sequential measurements of aCL titers and lupus anticoagulant (LAC) in 54 patients with SLE; the design of these studies is not clear, but the non-distinct time intervals of blood sampling and the number of blood samples per patient give the impression that they were retrospective, thus not offering more information than our own study. Our work involved 320 consecutive patients with a mean disease duration of nearly 120 months. Nevertheless, the important knowledge offered by the studies mentioned was taken into account, as follows: first, during SLE remission IgG aCL are present and significantly associated with APS, as we have shown; second, aPL-positive patients with SLE cannot be classified based on only one measurement, as we also emphasize in the definition of seroconversion.

Contribution of Korkmaz’s personal experience from 4 patients is welcome. Interestingly, the patient who received only AZA continued to be negative for aCL, while the aCL titers of the patient who received CYC pulses were instituted. From 1994 to 1996 the patient remained 6.5 g protein. Kidney biopsy showed diffuse, proliferative lupus nephritis WHO class IVc (activity index 1, chronicity index 6), respectively. Fibrin microthrombi, fibrous intimal hyperplasia, and focal cortical atrophy, all characterizing the APS nephropathy, were also detected in the last biopsy. Therapy was decreased to prednisone 5 mg orally, AZA 150 mg orally, and aspirin 100 mg orally only. The patient later reached endstage renal disease and in December 2002 underwent renal transplantation. Based on high titers of aCL antibodies, as well as recently developed anti-IIg-glycoprotein I antibodies, warfarin was also initiated after transplantation, aiming to maintain an international normalized ratio between 2.9 and 3.5.

REFERENCES


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