CORRESPONDENCE

Circulating Immune Complexes in Thrombotic Thrombocytopenic Purpura (TTP)

To the Editor:

We read with interest the article of Bukowski et al. about the treatment of plasmapheresis in TTP. These results suggested for these authors the existence of circulating immune complexes. Neame and Hirsh in their letter described two patients suffering from TTP in whom they were unable to detect circulating immune complexes with the Raji cell technique.

Recently we studied a patient with a typical acute TTP. Clinical observations: hemolysis, purpura, paresthesias, and profound alteration in the state of consciousness; laboratory data: hemolytic microangiopathic anemia, Coombs' negative, thrombopenia, and proteinuria. The patient was treated with heparin without any clinical or laboratory improvement. The addition of antiplatelet drugs (dipiridamol and aspirin) was not more effective. Finally, with prednisone treatment (100 mg/day) the patient completely recovered. We were not able to detect circulating immune complexes using the $^{125}$I C1q binding test performed every 4 days.

On the other hand, the levels of total hemolytic C, C1q, C3, C3d (breakdown of C3), C4, and C3PA were within the normal range, and there were no antibodies detectable against native and denatured DNA. Finally, skin biopsy did not show any deposit of C, C1q, C3, or immunoglobulins in the vessels by immunofluorescence.

In this case and in the case reported by Morrison and McMillan, immune complexes were undetectable. On the other hand, Bayer et al. found immune complexes in patients with TTP, but their patients suffered concomitantly of infectious endocarditis, and immune complexes could be ascribed to the host response against the infectious agents. There is therefore no direct evidence that immune complexes play a major role in the pathogenesis of TTP or that treatment by plasmapheresis has a beneficial effect through the removal of immune complexes.

ANTONIO CELADA, M.D.
LUC H. PERRIN, M.D.
Department of Medicine
Cantonal Hospital
Geneva, Switzerland

REFERENCES

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A Celada and LH Perrin