Is Membranoproliferative Glomerulonephritis an Autoimmune Disease?

H.	Hiroyuki	Ohi
Y.	Yoshiaki	Tanuma
M.	Michinobu	Hatano

Department of Internal Medicine II, Nihon University School of Medicine, Tokyo, Japan

Hiroyuki Ohi, MD, Department of Internal Medicine II, Nihon University School of Medicine, 30-1, Oyaguchi-Kamimachi, Itabashi-ku, Tokyo (Japan)

Dear Sir,

Although the pathogenesis of membranoproliferative glomerulonephritis (MPGN) is totally unclear, it is different from other primary renal diseases, because it exhibits specific phenomena such as the presence of C3 nephritic factor (C3NeF) or partial lipodystrophy. We investigated the presence of C3NeF in the serum samples of MPGN cases at various stages obtained from several hospitals. Most of the cases were MPGN type 1. C3NeF was found in 21 cases out of 98 hypocomplementemia cases (C3 $\leq$ 40%). It was interesting to observe that C4 nephritic factor (C4NeF) was present in 19 cases of hypocomplementemia. Out of these 19 cases, both C3NeF and C4NeF were found in the same serum samples of 5 cases. Furthermore, for one C3NeF-positive case, after a history of 7 years, the patient had high titers of anti-DNA antibody and antinuclear antibody. Then, she suffered from typical SLE. Considering the appearance of multiple autoantibodies, such as two nephritic factors which are antibodies against one's own complements, and the case in which the patient showed a typical SLE, MPGN could be thought of as an autoimmune disease. Hence, for MPGN in which the pathogenesis is still unknown, it is considered worthwhile to approach certain cases as an autoimmune disease both clinically and research-wise.

Reference

1 Tanuma Y.; Ohi H.; Watanabe S.; Seki M.; Hatano M.: C3 nephritic factor and C4 nephritic factor in the serum of two patients with hypocomplementaemic membranoproliferative glomerulonephritis. Clin. exp. Immunol. 76:82–85 (1989).