Does Nephritic Factor Relate to the Disease Activity in Hypocomplementemic Membranoproliferative Glomerulonephritis?

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C3NeF was measured by EAC3bBb and C4NeF by EAC4b2a stabilizing assay. a C3NeF activity cannot be inhibited by antipropertdin (P). b C3NeF activity can be inhibited by antipropertdin (P).

Dear Sir,

The pathogenesis of membranoproliferative glomerulonephritis (MPGN) has not been elucidated yet. Hypocomplementemia and C3 nephritic factor (C3NeF) have been observed, and the relationship between complement system, disease activity and prognosis has been studied [1, 2]. However, most of the studies are of the opinion that there is no relationship between them.

100 cases of hypocomplementemic MPGN were investigated. NeF was found in 31 sera of 100. 3 types of NeF were found in the sera [3,4], and they were classified into 4 groups (A-D; table 1). The clinical findings of these groups showed distinct characteristics for groups A and C. Group A was found in partial lipodystrophy and dense deposit glomerulonephritis cases and showed a low level of C3, a normal level of C5 and either negative or mild renal symptoms. Immunofluorescent findings (IF) included only C3 deposits in the glomerulus. On the other hand, group C was usually found in mesangiocapillary glomerulonephritis cases and showed markedly low

levels of C3 and C5. Approximately 50% of cases with active renal symptoms turned into renal failure within 5 years. IF showed C3 and immunoglobulin deposits in the glomerulus. Since prior to this research only 1 type of C3NeF has been noticed, its relationship with MPGN has not been clarified previously.

Table 1. C3 splitting factor in MPGN

C3 splitting factor

However, our findings have proved the importance of studying the role of NeF in elucidating MPGN pathogenesis. Furthermore, to understand the mechanism of activity of low C3 cases, the important role of C5 in the membrane attack complex should be taken into consideration.

Complement component

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