CD16⁺ and CD56⁺ Cells as Markers of Necrotizing and Crescentic Glomerulonephritis and Active IgA Glomerulonephropathy

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Dear Sir,

Necrotizing and crescentic glomerulonephritis is currently regarded as a histological hallmark of renal vasculitis, including Wegener's granulomatosis, polyarteritis nodosa and idiopathic rapidly progressive glomerulonephritis (RPGN). In addition, mild necrotic and crescentic (NC) lesions are often seen in glomeruli of IgA nephropathy (IgAN), the most common nephropathy in the world. We suppose, therefore, that in some cases with IgAN, the underlying pathogenic mechanism is analogous to that of renal vasculitis, i.e. so-called 'smoldering necrotizing and crescentic glomerulonephritis' is implied.

As far as we know, no serological marker of necrotizing and crescentic glomerulonephritis has been demonstrated so far except for antineutrophil cytoplasm antibody (ANCA). ANCA is indeed a useful marker in patients with Wegener's granulomatosis or polyarteritis nodosa [1, 2]; however, in patients with idiopathic RPGN or IgAN, ANCA has been seldom detected [3]. It has been established that the majority of peripheral blood lymphocytes mediating non-major histocompatibility complex-restricted cytotoxicity express the CD16 and CD56 antigens [4, 5]. We investigated the numbers of CD16⁺ (Leu-11b, Becton Dickinson) cells and CD56⁺ (Leu-19, Becton Dickinson) cells in peripheral blood lymphocytes using flow-cytometry and found these cells may be markers of NC lesions.

As shown in table 1, the number of CD16⁺ cells and CD56⁺ cells was significantly high in patients with idiopathic RPGN. This significance was also observed in Table 1. Percentage of CD16⁺ cells and CD56⁺ cells in peripheral blood lymphocytes in IgAN and idiopathic NCGN

patients with IgAN with NC lesions, and the difference in percentage of these cells was also significant when compared with those without NC lesions. We also looked for ANCA in every patient with idiopathic RPGN and IgAN with NC lesions, but we failed to detect ANCA in those cases examined.

From these results, we assume that the population of CD16⁺ cells and CD56⁺ cells could reflect the severity of NC lesion and that the same pathogenic mechanism might be involved in idiopathic RPGN and IgAN with NC lesions. Furthermore, we can also speculate that these cells could be a useful diagnostic tool for vasculitic disorders.

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