Detection of Polymorphonuclear Cells, Superoxide Dismutase and Poly C9 in Glomeruli of Patients with IgA Nephropathy

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Dear Sir,

Detection of polymorphonuclear cells (PMN), super-oxide dismutase (SOD) and poly C9 (membrane attack complex; MAC) in glomeruli of patients with IgA nephropathy was examined by immunofluorescence. It is postulated that the progression and/or exacerbation of chronic glomerulonephritis might be correlated with the activities of reactive oxygen species. Renal tissues were obtained from 14 patients with IgA nephropathy. Renal cryostat sections were reacted with mouse monoclonal anti-human PMN antibody (Sigma, St. Louis, Mo., USA, Lot No. 67F-48161, 1:10 dilution) and then stained with FITC-labeled goat anti-mouse Ig antiserum (Cappel Laboratories, Cochranville, Pa., USA, 1:20 dilution) [1]. The sections were also stained with FITC-labeled polyclonal anti-human SOD antiserum (The Binding Site, Birmingham, UK, Batch No. G0404, F/P molar ratio 1.73, 1:10 dilution). The sections were reacted with mouse monoclonal anti-human poly C9 antibody (#MBM 10) and then stained with FITC-labeled goat anti-mouse Ig antiserum (Cappel Laboratories, Cochranville, Pa., USA, 1:32 dilution) as described previously [2]. One to five PMN per glomerulus was observed in all 7 patients with IgA nephropathy examined, while the PMN staining was essentially negative in the control renal tissues. Renal SOD staining was observed in 7 out of 14 patients (50%) with IgA nephropathy, but was not observed in the control tissues. SOD was deposited in the glomerular mesangial areas, with a variable intensity from 1(+) to 3(+). Trace amounts of SOD were observed in the tubular epithelial cells. Poly C9 (MAC) was observed in all patients with this disease, and was located in the glomerular mesangial areas, Bowman's capsule, and extraglomerular vascular walls with a variable intensity of 1(+) to 2(+). No poly C9 staining was observed in the control renal tissues. PMN, SOD and poly C9 (MAC) in the glomeruli were observed in patients with the 'advanced

stage' of IgA nephropathy, but there was no statistical significance. It appears that the infiltration of PMN in the glomeruli, which have a high potential for production of reactive oxygen species on immunological stimulation, may play an important role in glomerular injuries, and induce an abnormally high local SOD activity for the protection. Moreover, the renal presence of poly C9

(MAC), which is known as a stimulant of mesangial reactive oxygen species production [3], might contribute to the renal damages. Further investigations are needed to determine the roles of reactive oxygen species produced by PMN and the activity of SOD in the peripheral blood in patients with IgA nephropathy.

References

Tomino, Y.; Ozaki, T.; Koide, H.; Yagame, M.; Eguchi, K.; Nomoto, Y.; Sakai, H.: Glomerular T cell and monocyte populations in patients with IgA nephropathy. *Jap. J. Nephrol.* 31: 221–226 (1989).

Eguchi, K.; Tomino, Y.; Yagame, M.; Miyazaki, M.; Takiura, F.; Miura, M.; Suga, T.; Endoh, M.; Nomoto, Y.; Sakai, H.: Double immunofluorescence studies of IgA and poly C9 (MAC) in glomeruli from patients with IgA nephropathy. *Tokai J. Exp. Clin. Med.* 72:331–335 (1987).

Adler, S.; Baker, P.J.; Johnson, R.J.; Ochi, R.F.; Pritzl, P.; Couser, W. G.: Complement membrane attack complex stimulates production of reactive oxygen metabolites by cultured rat mesangial cells. *J. Clin. Invest.* 77:762–767 (1986).