Worse Urinary Findings after Stimulating Tonsils in Patients with IgA Nephropathy

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Dear Sir,

Although the pathogenesis of IgA nephropathy still remains uncertain, the IgA-type immune complex is presumed to be one of the causative factors of this disease [1]. The predominant deposition of IgA in the glomeruli makes us consider the role of mucosal immunity in the pathogenesis because of the importance of IgA in immune defense mechanism at the mucosal sites. It is well known that IgA nephropathy patients often show gross hematuria or deteriorated urinary findings after upper respiratory tract infection such as rhinopharyngitis or tonsillitis and it is supposed that the pathogen that causes the preceding infection may play a role as an antigen. It is also suggested that abnormalities of the secretory immune system may exist in IgA nephropathy patients. Several investigators reported the increase of tonsillar IgA-secreting cells [2], the increase of IgA concentration in pharyngeal washings [3] and elevated salivary IgA in IgA nephropathy patients [4, 5]. We speculated that chronic tonsillitis may be involved in the development of IgA nephropathy and tried to clarify the deleterious effect of mechanical tonsil stimulation on urinary findings.

The subjects were 62 patients with IgA nephropathy (35 male and 27 female; mean age 26.3 years, age range from 14 to 55 years) and 20 patients with other renal diseases (12 male and 8 female; mean age 29.6 years, age range from 15 to 54 years). Other renal diseases included 11 cases of mesangial proliferative glomerulonephritis without IgA deposition, 4 membranous nephropathy, 3 acute glomerulonephritis and 2 idiopathic renal bleeding. Each tonsil was stimulated for 5 min with the Tonsil Provocator (Nagashima Medical Instruments, Tokyo, Japan) producing an ultra shortwave to 40.68 MHz. The probe was put into the mouth until directly attached to the tonsil. A serial quantitative analysis of proteinuria and hematuria was evaluated before, 3 and 24 h after the stimulation. When

urinary protein increased more than 30 mg/dl or erythrocyte count in the sediment increased more than 10/HPF, it was regarded as a deleterious change.

Forty of 62 patients with IgA nephropathy (65%) showed a deterioration of urinary findings (group A) and 22 did not (group B), while only 6 of 20 patients with other renal diseases (30%) showed a deterioration. The deterioration was significantly more frequent in IgA nephropathy than in other renal diseases ($p < 0.005$). Previous episodes of gross hematuria following upper respiratory tract infections were present in 17 of 40 patients of group A (43%) versus 4 of 22 patients of group B (18%). They were significantly more frequent in group A than group B ($p < 0.05$).

In our study tonsil stimulation in IgA nephropathy patients deteriorated their urinary findings. This suggests that some local lesion of the tonsils such as chronic tonsillitis may be involved in the pathogenesis of IgA nephropathy. The cause for the deterioration of the urinary findings after stimulation of the tonsils is unknown. The pathogenesis of IgA

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nephropathy still remains undefined. It is supposed that IgA nephropathy is an immune complex-mediated glomerulonephritis and the antibody is IgA resulting from an immune response to the pathogen of the preceding infection. Different investigators advocated streptococcus [6], pneumococcus [7], cytomegalovirus [8] and a virus existing in the pharyngeal cells [9] as the antigen. It is further speculated that some humoral factors such as the immune complex might be released by the tonsils through mechanical stimulation and be trapped within the glomerulus, causing glomerular injury. There were more patients with previous episodes of gross hematuria following upper respiratory tract infection in group A than in group B. It is suggested that tonsil stimulation creates a condition similar to that induced by upper respiratory tract infection.

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