Dear Sir,

The cause and pathogenesis of the most common glomerular disease, IgA nephropathy, are not clearly understood. It had been assumed that IgA nephropathy develops randomly in individuals. However, a possible genetic influence in its pathogenesis has been recently suggested because of some clinical aspects; e.g., the association of specific HLA antigen with the development of the disease, regional and racial differences in its prevalence, and clustering of the disease in some families [1]. Therefore, it seems likely that the possible genetic influence in its pathogenesis may be responsible for the development of the disease in susceptible individuals.

If a gene or genes considered to be associated with genetic susceptibility to IgA nephropathy exist, the phenotype(s) derived from the gene(s) may possibly be in evidence in patients afflicted with IgA nephropathy who are products of consanguineous matings. Therefore, we are interested in the characteristic signs and findings found in these patients in this investigation to attempt to explore the hypothesized gene(s).

Of 162 patients with IgA nephropathy who were followed up at our outpatient clinic, we identified 6 patients (3.7%) who were products of consanguineous matings using a questionnaire. Interestingly, it was revealed that 4 of these 6 developed hypertension during the early course of the disease, by a retrospective review of patients’ records. Two of the 4 had malignant hypertension and had lost renal function at age 28 and 41, respectively. Neither proteinuria and/or hematuria of all 6 patients have been eliminated since the diagnosis of the disease. However, these patients could not be distinguished from the other 156 patients by histological or laboratory examinations.

The 6 inbred patients may offer some clues to investigate the hypothesized gene(s). First, these findings may indicate that the inbred IgA nephropathy patients are vulnerable to hypertension. It may be possible to speculate that our patients whose parents were consanguineous mates might have been homozygotes of a susceptible gene for hypertension, or that the hypothesized gene(s) associated with genetic susceptibility to IgA nephropathy might have also played a role in the development of hypertension.
Second, if it is possible to find the locus (loci) which shows very high homozygosity rate of DNA markers, such as microsatellites, among these inbred patients with IgA nephropathy, it may be likely that the hypothesized gene(s) can be located around the locus (loci).

Reference

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