Letters to the Editor

The cortical distribution pattern of diabetic glomerulopathy

Dear Sir,

It is with interest that we read the article by Hørlyck et al. [1] describing the cortical distribution of diabetic glomerulopathy, since we studied spatial distribution of glomeruli with diabetic nodular lesion. Our results were very similar. The study was presented in a workshop entitled "Mechanisms of progression of renal lesions in metabolic diseases" held at the 27th Annual Meeting of the Japanese Society of Nephrology in Tokyo on 15 November 1984 [2].

We examined serial sections of kidneys from 11 autopsy cases with distinct diabetic glomerulopathy who were on haemodialysis less than one year or who received no haemodialysis. Autopsy cases on haemodialysis more than one year were omitted, since too many glomeruli were occluded.

The glomeruli with nodular lesions were not randomly distributed, but showed clustering. They tended to be located in columns perpendicular to the kidney surface. Hørlyck et al. [1] did not distinguish nodular lesions from diffuse lesions. However, findings revealed by periodic acid-Schiff or periodic acid silver methenamin staining are different between these two lesions. We consider that nodular lesions are not advanced diffuse lesions but the result of organization of repeated mesangiolytic lesions [3] which may be related to fragility of mesangium affected by long-term diabetic state. For the formation of cystic (mesangiolytic) lesions, some haemodynamic force may be necessary in addition to fragility of mesangium. This may suggest that glomeruli with nodular lesions, are distributed in relation to areas of some vessel branches.

Yours sincerely,
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References

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Autoimmunity and the development of diabetes mellitus in relation to mumps infection

Dear Sir,

Numerous experimental findings in animals as well as epidemiological and clinical observations support the hypothesis that certain viruses play a role in the pathogenesis of Type 1 (insulin-dependent) diabetes mellitus [1, 3]. Directly or via immune and/or autoimmune processes viruses might induce B-cell damage. Among the proposed diabetogenic viral agents, the most likely candidates are the mumps and the Coxsackie viruses. Virus replication has been detected in cultured human B cells by the double-labelled antibody technique for mumps and Coxsackie viruses [4, 10]. Numerous publications have proven that there is a relationship between the length of time since mumps infection and the occurrence of Type 1 diabetes mellitus [5]. According to these observations, which are usually of an anecdotal case report nature, the interval between the two illnesses ranges from a few days to several months. They all involve case studies and individual observations within families whose members developed Type 1 diabetes mellitus. It is admittedly impossible to conclude without doubt from such case report observations that there is some causal relationship between the two illnesses.

Helmke et al. [2] recently studied islet cell antibodies (ICA) and islet cell surface antibodies (ICSA) in 127 non-diabetic children after mumps infection. The study group consisted of selected mumps patients characterized by complications of previous mumps infection (meningoencephalitis, pancreatitis). At the same time, we investigated prospectively the possible relationship of glucose tolerance, B-cell function, diabetes-associated HLA antigens, haptoglobin phenotype, ICA and ICSA in 125 randomly selected non-diabetic subjects with antecedent mumps infection with and without complications [7]. According to the study of Helmke et al. [2], all but one of the patients studied had normal glucose tolerance over the subsequent 3 to 5 years. In our study, impaired glucose tolerance was diagnosed in 3.2% ($n=3$), but onset of diabetes mellitus did not appear within 14 to 26 months after mumps infection. The proportion of complications (e.g. pancreatitis, meningitis, orchitis) during antecedent mumps infection was higher in patients older than 16 years of age than in children. No relationships, however, existed between complications of antecedent mumps and the subsequent result of the glucose tolerance test.

Helmke et al. [2] found ICSA in 42 out of 68 sera from children (62%) with mumps infection. According to our study, ICSA were detectable in about 36% of children and 62% in patients older than 16 years of age. ICSA persisted in more than 50% of subjects up to 26 months after mumps infection [7]. According to the study of Helmke et al. [2], the sera tested for ICSA were absorbed with rat liver cells as well as spleen cells. In contrast, no pre-absorption experiments were carried out in our laboratory. Therefore, non-organ specific antibodies cross-reacting with surface components of rat islet cells may also be expected in the heterogenous pool of ICSA. According to this method, we found a prevalence of ICSA of about 5% in healthy control subjects [9].