SIMILARITIES BETWEEN LAMINATED NODULAR LESIONS IN GLOMERULI IN CROW-FUKASE SYNDROME AND DIABETIC NEPHROPATHY

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On the morphogenesis of laminated diabetic nodular lesions, we previously pointed out the possibility that they were formed in the organizing process of the diabetic mesangiolysis (Yafumi Saito et al.1). In addition, we recently reported that they were not formed by the overreproduction of mesangial matrix, but by an increase in type VI collagen fibers in that process, verified with monoclonal antibodies to collagen fibers (Kenzo Ikeda et al.2,3). Incidentally, we found similar laminated nodular lesions in patients with Crow-Fukase syndrome who had no glucose intolerance. In this paper, the immunohistological similarities between nodular lesions observed in patients with Crow-Fukase syndrome and in those with diabetic nephropathy are described and the morphogenesis of the laminated nodule is discussed.

PATIENTS and METHODS: Fresh frozen and paraffin embedded kidney specimens obtained from three patients with Crow-Fukase syndrome who had no glucose intolerance (54 year old male, and 50 and 56 year old females) and 51 patients with diabetic nephropathy (25 males and 26 females, mean age: 53.6 years) were comparatively examined by means of serial sections treated for light (PAS and PAM stains) and immunofluorescence (respective monoclonal antibodies to type III, IV and VI collagen fibers were used) microscopy.

RESULTS and DISCUSSION: In two of the three patients with Crow-Fukase syndrome, typical laminated nodular lesions were observed (Fig.1). The core portion of the nodular lesion, being a sequela of the mesangial stalk, was strongly PAM-positive, but the surrounding laminated acellular area was weakly positive. In addition, various phases of mesangiolyses1 and microaneurysms were concomitantly observed. These findings closely resembled those of diabetic glomerulosclerosis except for focal mesangial proliferation and the absence of diffusely thickened capillary walls. By comparative observations of the nodular lesions with serial sections, type VI collagen was clearly seen in the weakly PAM-positive area (Fig.2), but type III was not detected in the lesions in either disease. These results suggested that the laminated nodular lesion was formed by an increase in type VI collagen fibers in the process of reconstruction of mesangiolytic microaneurysms in both diseases and was not a pathognomonic indication of diabetic nephropathy.

Laminated nodular lesions in glomeruli

**Figure 1** Laminated nodular lesions observed in a patient with Crow-Fukase syndrome. The laminated acellular area was weakly PAM-positive (X250).

**Figure 2** Type VI collagen was clearly seen (right) at the corresponding site of the weakly PAM-positive area of the nodular lesion (left) in serial sections (X250).