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Nodular Glomerulosclerosis (Kimmelstiel-Wilson) After Total Pancreatectomy

There is controversy about the frequency of nephropathy in pancreatogenic diabetes. Many reports have appeared on diabetic nephropathy in humans with chronic pancreatitis or hemochromatosis. To our knowledge, however, only two reports have been made on nephropathy after total pancreatectomy (1,2), one of which is devoid of histological documentation (2). We describe a case of histologically proved nodular glomerulosclerosis in a totally pancreatectomized patient.

In 1972, a Japanese man, 50 yr of age, underwent total pancreatectomy after diagnosis of gastric cancer invading the pancreas. There was no family history of diabetes or any past history of obesity. His fasting blood glucose was 94 mg/dl, and glycosuria had not been found before the operation. After total pancreatectomy, ketosis-prone insulin-dependent diabetes developed. Despite daily insulin injections of 12–18 U, his fasting blood glucose ranged from 43 to 714 mg/dl. Between 1976 and 1979, paresthesia of the lower extremities, intermittent proteinuria, pedal edema, and high blood pressure (170/96 mmHg) emerged one after another. He died of pulmonary edema in November 1982. One month before his death, his urine protein was 2.8–11.4 g/day, blood urea nitrogen was 45.2 mg/dl, serum creatinine was 1.8 mg/dl, serum growth hormone was 1.6 ng/ml, and no serum or urine C-peptide immunoreactivity was detected. The kidneys weighed 170 and 220 g. On microscopic examination, nodular and diffuse glomerulosclerosis, microaneurysms, hyalinization of arterioles, and capsular drops were noted (Fig. 1). A diligent search of a trypsin-digested specimen of the retina failed to show any microaneurysms. Thus, despite the nephropathy, there was no evidence of retinopathy.

This case provides another example of the development of diabetic nephropathy in a secondary type of diabetes, thus supporting the notion that diabetic nephropathy may result from some metabolic consequence of insulin deficiency. Note that three cases of diabetic nephropathy, including ours, could be followed for >10 yr (1,2), whereas the series without clinically evident diabetic nephropathy after total pancreatectomy survived shorter periods (3). Therefore, it may be because of the limited survival period that diabetic nephropathy has not been noted more often as a sequela to total

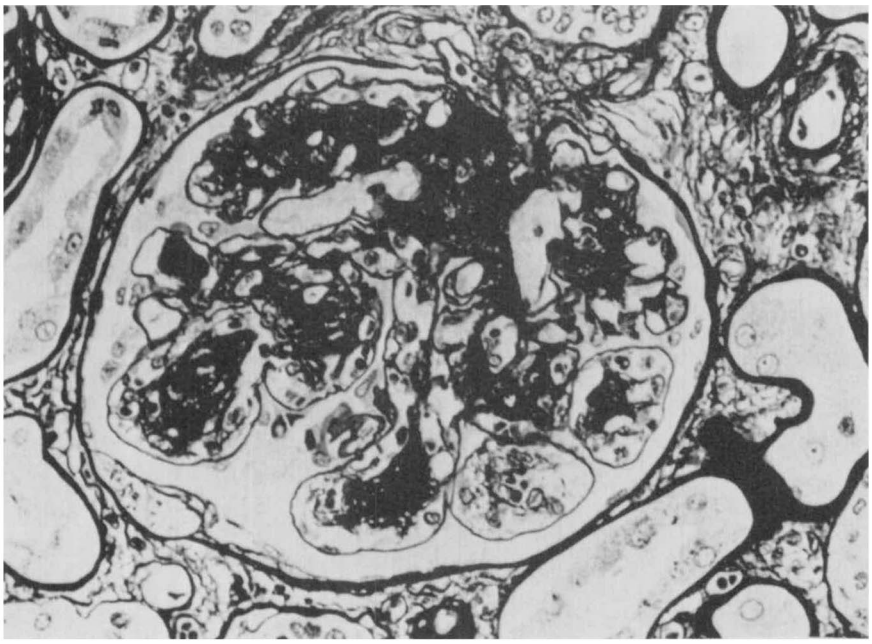

FIG. 1. Nodular glomerulosclerosis and microaneurysms (×100).

pancreatectomy. As total pancreatectomy becomes more prevalent and patients without a pancreas can expect to live longer (4), an increasing number of reports like the one described here will probably appear on microangiopathy in such patients.

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Psychological Adjustment to Diabetes: A Cautious Interpretation

The longitudinal study of psychological aspects of diabetes in a group of insulin-dependent diabetic children and their families by Jacobson et al. (1) is certainly an important one. The objectives of their 4-yr project—to examine the impact of diabetes on the adjustment of children and their families as well as the influences of psychosocial domains on various aspects of diabetes—if accomplished, should yield very needed answers indeed. However, their initial report has several limitations, many of which the authors freely admit could lead to serious misinterpretations if the data were not viewed with great caution.

First, it is not clear from the data whether the control group (acute illness) was comparable with the diabetic group in terms of the episode of illness. Were the days of hospitalization or the amount of pain similar? Did the acute-illness group have some intermediate or long-term disability, e.g., severe fractures or meningitis? Was there emotional upheaval as a result of some fractures, e.g., loss of a loved one in a motor vehicle accident? These data may not be overly important in the long term, but they should be particularly pertinent in the initial evaluation. Also, did the fact that almost half of the patients in the acute-illness group had fractures imply that these were accident-prone children (2)? This leads to the question, Should a truly healthy control group not be included? Both patients with diabetes and with other illnesses could fall into the illness-prone category and therefore be different from normal (3).

Second, as the authors themselves point out, the significant absence of representation in the group of diabetic patients in the lower social class (Hollingshead V) lends considerable doubt to their conclusion that social status is not a determinant of early adjustment to diabetes.

Third, their conclusions that diabetes in children does not lead to major psychological problems early in the course of their illness may be valid, but note that 42% of the responses were obtained from diabetic patients within 3 mo of diagnosis, i.e., in all likelihood before its chronic nature had "sunk in." Therefore, their findings cannot be interpreted as supporting "a growing body of research," suggesting, in long-term studies, that diabetes does not cause major psychological disruptions. Even their anecdotal case reports are testimony to the fact that serious adaptations must and do take place after the diagnosis of diabetes.

In following a group of patients on insulin-pump therapy (mean duration of diabetes 21 ± 13 yr), we are finding that psychological and social adaptation play significant roles in the incidence of pump-related complications encountered. Poorly adapted patients have had incidences of diabetic ketoacidosis and serious hypoglycemia 6.7 and 8.1 times more frequently, respectively, than well-adapted patients (both, $P < .05$) (4). I hope that the preliminary findings of Jacobson et al. will not be unjustifiably generalized, and I await their further reports with interest.

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