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Medical Memoranda

Childhood Nephrosis Followed by Acute Glomerulonephritis in Adulthood

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The causes of lipoid nephrosis and acute glomerulonephritis are still poorly understood, and it remains uncertain whether any relation exists between these entities. We have recently observed a patient in whom the sequential occurrence of the two diseases took place.

CASE REPORT

A 46-year-old man was admitted to hospital because of swelling of the ankles. He had been well until the onset of oedema of the face and legs three weeks previously. There was no history of pharyngitis, fever, shortness of breath, headache, oliguria, or haematuria. He had been in hospital 36 years previously after six months of anasarca. Records of that admission indicated marked oedema of the abdominal wall and lower extremities, ascites, and heavy proteinuria without haematuria; recovery was complete after six weeks in hospital. He passed Army physical examinations 24 and 19 years ago, and negative urinalyses were obtained by a private physician eight and seven years ago.

Physical examination on admission showed him to be well developed, well nourished, and in no apparent distress. Mild periorbital oedema was evident. His temperature was 98.6° F. (37° C.), pulse 52, respirations 20, and blood pressure 120/70 mm. Hg.

Photomicrograph of renal biopsy specimen showing a glomerulus with increased cellularity and prominent endothelial cells.

Cervical veins were not distended and the lungs were clear. The heart was not enlarged. Two-plus soft pitting peripheral oedema was present. The urine gave a two-plus test for protein; the sediment contained numerous red blood cells, 2–4 white cells per high-power field, and a few red cell casts. Antistreptolysin, anti-hyaluronidase, and antistreptodornase titres were normal. Streptococci could not be isolated from the nasopharyngeal passages. Blood urea nitrogen was 18 mg./100 ml. The 24-hour urine protein output was 249 mg.

The haematocrit was 34%; the white-cell count, differential, and E.S.R. were normal. X-ray examination of the chest showed the heart to be at the upper limits of normal in size. Intravenous urography was normal. An electrocardiogram showed sinus bradycardia. Renal biopsy performed on the fifth hospital day showed acute proliferative glomerulonephritis but gave no evidence of chronic renal disease (see Fig.).

Penicillin was given for 10 days. Diuresis of 14 lb. (6.4 kg.) occurred, with a rise in haematocrit from 34 to 42% and disappearance of oedema. At the time of discharge (two weeks after admission) the urine gave a negative reaction for protein and had a specific gravity of 1026; the sediment contained 2–4 red cells per high-power field. The blood urea nitrogen was 22 mg./100 ml., and the creatinine 1.5 mg./100 ml.

DISCUSSION

The clinical and histological features of this case are typical of acute glomerulonephritis (Jennings and Earle, 1961), though no proof of recent streptococcal infection could be obtained. The childhood episode appears characteristic of lipoid nephrosis. Though acute glomerulonephritis causing the nephrotic syndrome cannot be excluded with certainty, complete recovery after six months of anasarca and the absence of haematuria militate against this diagnosis (Lawrence *et al.*, 1963). The sequential occurrence of these two diseases in the same patient is apparently a rare phenomenon; we have found no report of a similar case. The fact that one kidney can show evidence of both conditions is of interest because it suggests that the causes of the two disease states are distinct or that the organism's pattern of response to a given stimulus may alter with age.

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