Membranous Glomerulonephritis Associated with Autosomal Dominant Polycystic Kidney Disease

S. Sanjiv Saxena
R.K. Hotchandani
U.N. Bhuyan
S.K. Agarwal
S.C. Tiwari
S.C. Dash

Departments of Nephrology and Pathology, All India Institute of Medical Sciences, New Delhi, India

Dr. Sanjiv Saxena, Assistant Professor, Dept. of Nephrology AIIMS, New Delhi-110029 (India)

Dear Sir,

Nephrotic range proteinuria is unusual in autosomal dominant polycystic kidney disease (APKD). In fact, APKD is never listed as a cause of nephrotic syndrome (NS) and neither is it described as a complication of this genetic disease [1]. In those rare patients who develop this complication, several histologic diagnoses have been seen. These include IgA nephropathy, rapidly progressive glomerulonephritis, focal glomerulosclerosis in rare cases and membranous glomerulonephritis (MGN) [2-4]. We report here a case of APKD with NS where the kidney biopsy showed MGN.

A 22-year-old male presented to the Nephrology services of our hospital for swelling over his face and feet for the last 2 months and a mild reduction in urine volume. There was no history of hematuria, cola-colored urine, dysuria, frequency or graveluria. He had been in perfect health prior to this illness. He was not addicted to any drugs. There was no preceding sore throat or pyoderma. He was neither a known diabetic nor hypertensive. Family history revealed that his father died of renal failure at the age of 32 years and was said to have had cysts in both kidneys. Examination of the patient revealed edema in both legs and facial puffiness; blood pressure was 130/80. The rest of the general and systemic examination was normal. Investigations showed hemoglobin to be 2.20 mmol/l (14 g/dl); ESR, 14 mm in the 1st hour; serum creatinine, 70.4 µmol/l (0.8 mg/dl); serum albumin, 25 g/l; serum cholesterol, 7.4 mmol/l (280 mg/dl); and fasting blood sugar, 3.9 mmol/l (70 mg/dl). Urine examination showed 4+ albumin with occasional RBCs and WBCs. The 24-hour urine showed albumin of 6 g, and creatinine clearance was 90 ml/min. Serum complements were normal. Hepatitis B surface antigen and antinuclear factor were negative. KUB x-ray showed bilateral normal-sized kidneys and no calculi. Ultrasonography showed multiple cysts in both kidneys though the liver and spleen were normal. In view of the nephrotic presentation, a kidney biopsy was done, revealing seven glomeruli showing diffuse thickening of

Fig. 1. Kidney biopsy showing diffuse thickening of basement membrane with epi-membranous spikes.

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glomerular basement membrane with normal mesangial cellularity. Silver methenamine staining showed epimembranous spikes (fig. 1). A diagnosis of MGN with APKD was made. APKD is a genetic disorder transmitted in an autosomal dominant manner. Proteinuria is commonly seen in APKD but is generally 1-2 g/day [5]. In Dalgaard’s [6] review of 284 patients with APKD, proteinuria was seen in 75% of patients. Only in 3 patients was proteinuria exceeding 5 g/l found. In a recent review by Montoyo et al. [7], only 1 out of 65 APKD had NS. Case reports of MGN as a complication of APKD do exist in the literature, but, to our knowledge, only 2 cases of MGN have been described in APKD to date. Our case report is the third such report.

In our patient, a meticulous search was made for any evidence of diabetes mellitus, systemic lupus, or any drug ingestion which could account for a similar histology, but none were found. Idiopathic MGN hence can occur with APKD.

References