Autosomal Dominant Polycystic Kidney Disease and Mesangioproliferative Glomerulonephritis

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Dear Sir,

Glomerulonephritis is rare in autosomal dominant polycystic kidney disease (APKD). In fact, moderate proteinuria is observed in many patients with APKD; however, the development of nephrotic-range proteinuria is considered to occur exceptionally in this disease. A few APKD patients who develop nephrotic-range proteinuria have been seen with several glomerular abnormalities such as IgA nephropathy [1], rapidly progressive glomerulonephritis [2], focal glomerulosclerosis [3], and membranous glomerulonephritis [4]. Here we report a case of glomerulonephritis in whom the kidney biopsy specimen showed mesangioproliferative glomerulonephritis which was later diagnosed as APKD.

In 1986, a 56-year-old woman presented to our hospital with hematuria and flank pain. Abdominal ultrasonography and 'kidney-ureter-bladder' (KUB) X-ray were normal, and urine cultures were negative. Urine examination showed numerous red and occasional white blood cells and granular casts. Proteinuria was 0.5 g/24 h. A kidney biopsy specimen revealed mesangial proliferative glomerulonephritis. The patient received prednisone 0.5 mg/kg/day. Hematuria and proteinuria improved after prednisone treatment. Blood pressure and renal function were normal. During the following years, urinary sediments occasionally showed microscopic hematuria. At this time, the renal function test results were within normal limits, the blood pressure was normal, and urine cultures were negative. In December 1987, KUB X-ray showed normal-sized kidneys, no calculi, and trabecular irregularities. In December 1991, the KUB X-ray was normal, but a newly designed ultrasonographic equipment showed multiple cysts in the right kidney and a normal-sized left kidney. Computerized tomography diagnosed APKD, showing two kidneys and pancreas occupied by numerous cysts of variable size. The family history revealed that her sister was a hemodialysis patient.

Because of adaptive hemodynamic and hypertrophic factors, focal glomerulosclerosis and proteinuria have been described in APKD [5]. According to the results of the series, the presence of nephrotic-range proteinuria is rare in APKD. In a study of 65 APKD patients, 60% had proteinuria < 1 g/day; 10.7% of the cases had proteinuria of 2-3.5 g/day.

In the literature, we did not find any case similar to ours. APKD and mesangioproliferative glomerulonephritis are a very rare coincidence. Although it may be postulated that our patient had acquired cystic disease due to renal changes with time rather than APKD, as described in similar case reports [6], because there were also pancreatic cysts and because of her sister's history, we believe that she suffered from pure APKD. Prior to this time we were not able to diagnose APKD accurately due to some technical deficiencies. To establish reliable diagnostic procedures, experimental models are needed to understand these findings in APKD.

References

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