Massive Proteinuria due to Renal Artery Stenosis

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Dear Sir,

Nephrotic-range proteinuria associated with renal artery stenosis has been reported by several authors [1–4]. Hyperreninemia secondary to renal artery stenosis was proposed as a possible cause of protein leak [2].

In this article, we present a case of renovascular hypertension exhibiting hyperreninemia with massive proteinuria. Renal biopsy showed no significant glomerular abnormalities on light microscopy, immunofluorescence study, and electron-microscopic examination. Following radical surgery, both proteinuria and hyperreninemia were dramatically improved.

Case Report

A 56-year-old man was referred to our hospital for evaluation of hypertension. His brachial blood pressure was 210/120 mm Hg. Peripheral pulses were symmetric and not weakened. Abdominal bruit was not audible. There was slight pitting edema in the legs. The electrocardiogram and chest X-ray were normal. Urinalysis showed massive proteinuria (4–7 g/day) with normal sediment. BUN was 26 mg/dl, serum creatinine 2.2 mg/dl, creatinine clearance 37 ml/min, and serum total protein 6.0 g/dl. Serum sodium was 142 mEq/l, potassium 4.0 mEq/l, and chloride 109 mEq/l. Serum cholesterol was 288 mg/dl. Antistreptolysin O titer and serum level of C3, C4, and CH50 were within the normal range. Antinuclear antibodies were absent. Plasma renin activity (PRA) was over 100 ng/ml/6 h (normal range 5–30), and plasma aldosterone concentration 15.6 ng/ml (normal range 2–12). Renal scintigram and renogram exhibited marked reduction in the size and perfusion of the left kidney. Abdominal arteriogram showed diffuse atherosclerosis of the aorta with severe stenosis of the left renal artery (approximately 75% stenosis). The right renal artery was almost intact.

From these clinical data, he was diagnosed as having renovascular hypertension due to left renal artery stenosis, and underwent a bypass surgery that reconstructed the left renal artery by auto-vein grafting (the femoral vein was used). Following surgery, the blood pressure and PRA returned to normal, and proteinuria decreased to 0.5 g/day.

Renal biopsy, performed at the operation, revealed no significant abnormalities on light microscopy and immunofluorescence

study. On electron microscopy, there were no electron-dense deposits or basement membrane changes, and epithelial foot process were intact (fig. 1, 2).

Fig. 1. Light micrograph of the kidney biopsy specimen showing an essentially normal appearance. There is no cellular proliferation, segmental sclerosis, or basement membrane thickening. HE. $\times 250$.

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Fig. 2. Electron micrograph of the glomerulus illustrates absence of effacement of the epithelial cell foot processes. No electron dense deposits or basement membrane changes are seen. $\times 6,000$.
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In our case, the right kidney was not histologically examined. Therefore, a kind of glomerulopathy affecting only the right kidney cannot be ruled out as the cause of massive proteinuria. However, the right kidney was not manipulated at all during surgery. Dramatic improvement of proteinuria following bypass surgery seems to indicate that proteinuria was induced by renal artery stenosis itself. Namely, glomerular hyperfiltration secondary to hyperreninemia is thought to have caused significant proteinuria just as Montoliu et al. [2] speculated.

Following the reports by Montoliu et al. [2] and Eiser et al. [3], this letter is, to our knowledge, the third report of case in whom the ultrastructure of the kidney was examined, enabling one to exclude a concomitant complication of primary glomerular diseases in the nephrotic syndrome associated with renal artery stenosis.

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