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Case Report

Renal artery stenosis presenting with nephrotic-range proteinuria: a case report

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Abstract

Renal artery stenosis (RAS) is commonly presented with hypertension and chronic kidney disease. We report a rare case of RAS occurring in a 78-year-old man who presented with nephrotic-range proteinuria. Renal biopsy on the left side was performed, and results showed mesangiopathic glomerulonephritis, which was not compatible with the cause of nephrotic-range proteinuria. Proteinuria was decreased by angiotensin receptor blocker, but azotemia was aggravated. Therefore, angiotensin receptor blocker was discontinued inevitably and thorough evaluation for the possibility of RAS was performed. Computed tomography angiography revealed significant RAS on the left side and a renal artery stent was inserted. After stenting, aortic dissection developed and progressed despite tight control of blood pressure. After inserting another stent graft through the true lumen of the left renal artery, the patient's renal function and proteinuria improved markedly.

Introduction

Renal artery stenosis (RAS) is frequently associated with hypertension and renal insufficiency [1]. Nephrotic-range proteinuria is usually caused by primary or secondary glomerular diseases associated with diabetes, drugs, malignancy, infectious disease, or autoimmune disease [2]. Although proteinuria caused by RAS has been reported in some cases, it is uncommon to cause significant heavy proteinuria [3,4]. During recent decades, ongoing research has pursued treatment options for renovascular disease, focusing on the effectiveness of medical therapy and endovascular intervention [5–9]. Previous clinical studies showed that renal artery stenting did not confer significant benefit over medical therapy with respect to preserving kidney function and preventing adverse cardiovascular events [5–8]. However, revascularization still plays a substantial role in the treatment of RAS, depending on each patient's clinical characteristics.

We herein describe a 78-year-old man with severe RAS who presented with heavy proteinuria and renal insufficiency and was treated successfully by angioplasty and repeated stenting.

Case report

A 78-year-old man known to have had hypertension for 5 years was admitted because of 2-month history of pitting edema in lower extremities. He had taken diuretics (furosemide) for a month, but still complained of edematous legs. He had been in good health until 2 months ago and had not taken any

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medication except for antihypertensive drug. The blood pressure (BP) was 125/70 mmHg on admission with amlodipine 5 mg orally once a day after switching from telmisartan 40 mg orally once a day about 2 weeks before admission. He did not show abdominal bruits and other physical signs suggesting infection. Laboratory evaluation showed serum creatinine (Cr) of 1.58 mg/dL, serum sodium of 142 mmol/L, serum potassium of 4.2 mmol/L, serum albumin of 3.8 g/dL, and total cholesterol of 192 mg/dL. Liver function profiles, uric acid, and serum electrolytes were within normal range. The number of white blood cells and the levels of C-reactive protein and erythrocyte sedimentation rate were also within normal range. His spot urine protein-to-Cr ratio (PCR) and spot urine albumin-to-Cr ratio (ACR) were 4.51 mg/mg and 3,943.8 μg/mg, respectively. Tests for hepatitis B surface antigen, antibody to hepatitis C virus, antinuclear antibodies, and antineutrophilic cytoplasmic antibodies were negative.

Renal ultrasonography showed right and left kidneys measuring 9.8 and 8.3 cm in length, respectively, with slightly increased cortical echogenicity. Percutaneous biopsy of the left kidney revealed mesangiopathic glomerulonephritis (Fig. 1A), with 30% of glomeruli showing global sclerosis. Immunofluorescence was negative, and electron microscopy was unremarkable (Fig. 1B). Heavy proteinuria could not be explained by the results of kidney biopsy. Small restrictions of the gamma globulin region were found at the serum electrophoresis, and an abnormal band was observed against anti-IgG and anti-kappa from serum immunofixation. However, bone marrow biopsy showed no definite evidence of clonality in plasma cells.

A week after resuming angiotensin receptor blocker (ARB) (losartan 50 mg orally once a day), spot urine PCR and ACR decreased from 4.51 to 2.30 mg/mg and from 3,943.8 to 1,887.1 μg/mg, respectively. However, renal function significantly deteriorated (increase in serum Cr from 1.73 to 2.75 mg/dL). ARB was discontinued because of rapidly progressive azotemia. Considering the size discrepancy of both kidneys and progressive azotemia by ARB, RAS was suspected. Therefore, renal artery computed tomography (CT) angiography and angiogram of renal artery were performed. The results showed severe stenosis of left main renal artery origin site (90%; Figs. 2A–B) and mild luminal narrowing of proximal right main renal artery (less than 30%). A stent was successfully inserted into the left renal artery (Fig. 2C). However, focal aortic dissection developed right after the intervention.

Three days after renal artery stenting, the spot urine PCR and ACR decreased from 2.30 to 0.55 mg/mg and from 1,887.1 to 77.8 μg/mg, respectively. However, serum Cr level increased to 2.84 mg/dL. Aorta noncontrast CT showed acute intramural hematoma at descending and abdominal aorta and localized dissection at the distal segment of abdominal aorta. Conservative management with tight control of BP was continued. Thoracoabdominal CT angiography taken 5 days later showed progression of the intramural hematoma of aorta. The diameter of the aorta was increased, and the aortic dissection extended from the origin of superior mesentery artery to right common iliac artery. The progressed aortic dissection partially blocked the entry of the stent originally inserted into left renal artery, but the left kidney still received blood flow from the true lumen of aorta (Fig. 3). Three days later, angiography was performed again because of persistent azotemia showing serum Cr higher than 2.0 mg/dL. Angiography showed that the entire orifice of left renal artery stent was in the false lumen because of progressed aortic dissection, so the left kidney was not receiving any blood flow from the true lumen (Fig. 4A). Another stent graft insertion into the original stent and balloon dilatation were therefore performed on left renal artery, restoring blood flow from the true lumen of the aorta (Fig. 4B). After intervention, serum Cr level decreased to 1.76 mg/dL, and the patient was discharged on aspirin and a β-blocker (atenolol). Spot urine PCR and ACR at discharge were 0.27 mg/mg and 156.6 μg/mg, respectively.

He has had stable renal function with serum Cr around 1.50 mg/dL and minimal microalbuminuria, and well-controlled BP was observed for more than 2 years after discharge.

Discussion

RAS is a common cause of curable hypertension and renal insufficiency [1], but has not been mentioned as a major cause of heavy proteinuria in previously reported reviews [2]. However, a few cases of nephrotic-range proteinuria in patients with renovascular disease have been reported, usually resulting from atherosclerosis, especially in the elderly [3,4,10,11]. In some cases, massive proteinuria has been successfully treated with angiotensin-converting enzyme inhibitors (ACE-i) or ARB or by revascularization or removal of the affected kidney. In our case, an elderly patient presented with nephrotic-range proteinuria without typical signs of RAS, such as uncontrolled

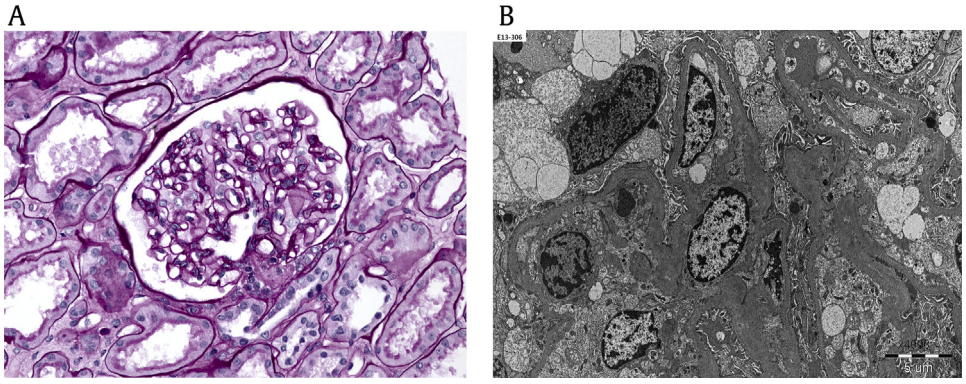

Figure 1. Features of the renal biopsy. (A) The glomeruli are mildly hypercellular and show focal mesangial proliferation in PAS stain. Mesangial matrix is mildly increased (PAS, ×400). (B) The glomerular basement membrane is slightly irregular in contour with mild effacement of epithelial foot processes; mesangial matrix is slightly increased (transmission electron microscopy, ×4,000).
PAS, periodic acid-Schiff.