Case Report

Successfully Treated “Accelerated” Renovascular Hypertension with Intravascular Stenting

Kent DOI, Hidekazu MORIYA*, and Shuzo KOBAYASHI*

A 76-year-old man developed progressive renal dysfunction with refractory hypertension. Bilateral renal artery stenosis due to atherosclerosis was revealed. Both the hypertension and renal dysfunction were improved by percutaneous transluminal renal angioplasty with stenting. Based on the rapidly progressive elevation of plasma renin activity and the improvement of both renal dysfunction and hypertension after stenting, this was considered a case of “accelerated” renovascular hypertension. There have been an increasing number of patients with bilateral renal artery stenosis due to atherosclerosis. The present case reminds us that a rapid progression of renal dysfunction suggests, in addition to besides rapidly progressive glomerulonephritis with crescent formation, bilateral renal artery stenosis, the incidence of which is on the rise. In the present case, angioplasty with stenting was effective for blood pressure control and preservation of renal function. (Hypertens Res 2002; 25: 945–948)

Key Words: atherosclerosis, percutaneous transluminal renal angioplasty, progressive renal dysfunction, renal artery stenosis, renovascular hypertension

Introduction

There are two major causes of renal artery stenosis: fibromuscular dysplasia and atherosclerosis. The number of patients with renal artery stenosis due to atherosclerosis as well as atherosclerotic cardiovascular disorders is increasing. Atherosclerosis accounts for 90% of the cases with renovascular hypertension and is frequently associated not only with hypertension but also renal dysfunction, especially in the elderly. Therefore, those patients who show rapidly progressing renal dysfunction require further evaluation for not only crescentic glomerulonephritis but also the co-existence of renal artery stenosis.

Three therapeutic alternatives are available: medical therapy with renin angiotensin blockers, percutaneous angioplasty, and surgery. Conventional balloon angioplasty for atherosclerotic lesions has a lower initial success rate and higher restenosis rate as compared with that for fibromuscular dysplasia. However, percutaneous angioplasty with stenting has recently achieved dramatic improvements in a large number of patients (1).

We here will report a case of bilateral renal artery stenosis with refractory hypertension and progressive renal dysfunction. Percutaneous angioplasty with stenting in both the atherosclerotic lesions was effective for blood pressure control and preservation of renal function.

Based on the clearly defined clinical course of the serum creatinine levels, plasma renin activity and changes in the renogram pattern, the present case was considered a case of “accelerated” renovascular hypertension with similarity to angina pectoris.

Case Report

A 76-year-old man was admitted to our hospital because of dyspnea and pitting edema of the lower extremities in June 2000. Proteinuria had first been observed in this patient in

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1998, but his blood pressure was normal at that time. In 1999 his blood pressure began to elevate and treatment for hypertension was started. He was admitted to the hospital twice—once in March 2000 and once in May 2000—with severe hypertension and overhydration. On each occasion, diuretics and antihypertensives improved his symptoms. Renal biopsy was performed on the first admission in March 2000 because of proteinuria. Renal biopsy obtained from his right kidney showed mild mesangial proliferative glomerulonephritis with 15% global sclerosis. Immunofluorescence revealed that there was a positive staining of immunoglobulin G (IgG) in the mesangium associated with electron dense deposits. At that time he showed mild hypertension with renal dysfunction (creatinine 2.3 mg/dl) and normal plasma renin activity (0.9 ng/ml/h).

Upon admission in June 2000, physical examination revealed that his blood pressure was 140/61 mmHg, his pulse rate was at 47 beats/min, and his body temperature was 36.3ºC. His body weight had increased by 3 kg over the previous 3 months. Results of the examination of the head, eyes, ears, nose, and throat were unremarkable except for dilatation of the jugular veins. Cardiac examination results were normal, but inspiratory crackles were audible at both lung bases. There was neither abdominal organomegaly nor bruit. The patient had bilateral pitting edema in the lower extremities.

Arterial blood gas analysis performed in room air showed $pO_2$ 67.7 mmHg, $pCO_2$ 33.2 mmHg, HCO$_3$ 24.1 mEq/l, and pH 7.478. The serum chemistry was as follows: sodium 136 mEq/l, potassium 4.2 mEq/l, chloride 99 mEq/l, blood urea nitrogen 75 mg/dl, and creatinine 3.7 mg/dl. Albumin was 4.5 g/dl, urate 4.9 mg/dl, calcium 8.5 mg/dl, and phosphorus 3.9 mg/dl. Hematology studies showed hemoglobin of 9.5 g/dl, a white blood cell count of 5,600 cells/mm$^3$ with a normal differential, and platelet of 173,000 cells/mm$^3$. Urinalysis revealed a pH of 5.0, protein $\uparrow$, and occult blood $\uparrow$, and microscopic urinalysis showed no cellular casts, and white and red blood cell counts of only one-to-four per high-powered field. A 24-h urine collection contained 0.3 g of protein, and the creatinine clearance was 16.1 ml/min (15.4 ml/min/1.73 m$^2$). The ultrasound with color Doppler demonstrated an atrophic left kidney with a higher resistive index than the right kidney (left kidney: 74 mm $\div$ 42 mm, resistive index 0.767; right kidney: 93 mm $\div$ 44 mm, resistive index 0.688). The resistive index was defined as follows: [1-($\text{end-diastolic velocity} \div \text{maximal systolic velocity}$)] $\times$ 100. Echocardiography showed a good contraction of the left ventricle, moderate pericardial effusion and dilatation of the inferior vena cava.

Despite the fact that the patient had been treated with large amounts of diuretics and antihypertensives in the outpatient department—i.e., furosemide 120 mg, amlodipine 10 mg, doxazosin 4 mg, carvedilol 10 mg, and methyldopa 500 mg—he was readmitted because of overhydration. A rapid increase in plasma renin activity and serum aldosterone level within several months suggested that renal artery stenosis had progressed to the verge of a total occlusion; that is, he had so-called “unstable” renovascular hypertension (Table 1). Angiography of the renal artery with renal vein renin measurement demonstrated that there was 90% stenosis at the left renal artery and 99% stenosis at the right renal artery (Fig. 1). Percutaneous transluminal renal angioplasty (PTRA) with stenting was performed to the bilateral renal arteries.

Table 1. Plasma Renin Activity and Serum Aldosterone Level Increased during a Course of Several Months

<table>
<thead>
<tr>
<th></th>
<th>Plasma renin activity (ng/ml/h)</th>
<th>Aldosterone (pg/ml)</th>
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<tbody>
<tr>
<td>2000/3/3</td>
<td>0.9</td>
<td>20</td>
</tr>
<tr>
<td>2000/5/24</td>
<td>4.2</td>
<td>230</td>
</tr>
<tr>
<td>2000/6/7</td>
<td>13.0</td>
<td>370</td>
</tr>
</tbody>
</table>

Plasma renin activity was highest in the right renal vein (right renal vein, 36 ng/ml/h; left renal vein, 14 ng/ml/h; inferior vena cava, 13 ng/ml/h). The systemic plasma renin ac-
Activity decreased from 10.0 ng/ml/h before PTRA to 4.8 ng/ml/h on the day after PTRA, and continued to decrease to 1.0 ng/ml/h after 2 weeks. The serum creatinine also decreased from 3.5 mg/dl to 1.8 mg/dl, and the large amounts of diuretics and antihypertensives were discontinued. Twenty-four-hour urinary protein increased transiently after PTRA and then decreased to the baseline level (Fig. 2). The pleural effusion and cardiomegaly on the chest X-ray improved. The comparison of renal scan with 99mTc-DTPA before and after PTRA showed that the glomerular filtration rate (GFR) of the left kidney did not change at all, while that of the right kidney increased, suggesting that PTRA improved only the right kidney (Fig. 3).

Discussion

Atherosclerotic disease in the elderly and patients with diabetes, hypertension or hyperlipidemia often involves the bilateral renal arteries and is rapidly progressive. Scoble and Hamilton (2) reported that 10 to 15% of patients over the age of 50 with an advanced renal failure had bilateral renal artery stenosis caused by atherosclerosis.

In the past, medical therapy with angiotensin converting enzyme inhibitors has generally been the preferred treatment for older patients with renal artery stenosis, since conventional balloon angioplasty for atherosclerotic lesions has shown a lower initial success rate and higher restenosis rate as compared with fibromuscular dysplasia (1, 3). However, the introduction of stenting has brought about a dramatic improvement in PTRA (4-6). In the present case, angioplasty with stenting at the bilateral renal artery stenosis of atherosclerotic lesions was effective for blood pressure control and preservation of renal function.

We performed stenting at the bilateral renal arteries, but only the right kidney was responsive. The left kidney was found to be small by ultrasonography and contributed to decreased GFR, as revealed by the renal scan with 99mTc-DTPA. Radermacher et al. (7) reported that the value of the resistive index at the segmental artery in the kidney could predict that whether the kidney would be responsive to angioplasty. In their report, a resistive index of more than 0.8 indicated that angioplasty would not improve renal function, blood pressure, or kidney survival. In the present case, the resistive index was less than 0.8 at both kidneys, but was higher at the atrophic left kidney than the non-atrophic right kidney. Doppler ultrasonography indicated that the right kidney might be more responsive to angioplasty than the left kidney. The results of renal vein renin measurement and the comparison of renal scan with 99mTc-DTPA before and after PTRA demonstrated that the right kidney was responsive and the left kidney was not.

It was difficult to determine whether the stenosis has a significant hemodynamic effect on renin secretion. There was no consensus about which kidney should undergo revascularization. In the present case the stenosis of the right renal artery seemed to have progressed so rapidly that the right kidney did not become atrophic. Furthermore, the rapid increase of systemic plasma renin activity in only 3 months and the finding that the renin activity was higher on the right side than the left confirmed that the right kidney was much more responsive to refractory hypertension. The clinical course in terms of the progressive nature of refractory hyper-
tension indicated that a non-atrophic kidney with renal artery stenosis could be rescued by revascularization.

Thadhani et al. (8) and Fukushima et al. (9) observed that focal segmental glomerulosclerosis in cases with renovascular hypertension. In our case, the patient had chronic glomerulonephritis with histologically proven mild mesangial proliferative glomerulonephritis. However, renal biopsy did not demonstrate any pathologic findings, such as atrophic glomeruli, wrinkling of capillary walls or interstitial fibrosis, which are usually seen in the clipped-kidney in 2-kidney 1-clip Goldblatt hypertensive rats (10). These findings suggest that the stenosis at the right renal artery was mild at the time of the March 2000 biopsy. Indeed, at this time, the patient’s hypertension was mild and his plasma renin activity was not elevated.

In conclusion, the present patient had bilateral renal artery stenosis accompanied by renal dysfunction. We performed percutaneous transluminal renal angioplasty with stenting, and his blood pressure and renal function subsequently improved.

The patient’s course of rapid progression of renal dysfunction, refractory hypertension, and elevation of the systemic plasma renin activity implied that the progression of renal artery stenosis was very rapid. Importantly, these results suggest that not only rapidly progressive glomerulonephritis with crescent formation but also renal artery stenosis should be considered in patients with rapidly progressive glomerulonephritis.

We performed angioplasty at both renal arteries because of the bilateral stenosis, despite the fact that the left kidney was atrophic. After angioplasty, however, the non-atrophic right kidney alone responded to the revascularization. It was difficult to predict which side of the renal artery played the larger role in the hypertension and thus would contribute more to improvement. The patient’s clinical course of rapidly progressive refractory hypertension indicated that PTRA could rescue his kidney.

References