Renal Artery Thrombosis in a Patient with Membranous Glomerulonephritis

Toshiaki Shibasaki, Fumio Ishimoto, Kazuya Kodama, Iwao Ohno and Osamu Sakai

A 64-year-old man with renal artery thrombosis (RAT) associated with nephrotic syndrome (NS) is reported. Although this patient was diagnosed as NS due to membranous glomerulonephritis (MGN) and treated with prednisolone, RAT occurred as a result of unknown mechanisms and caused mild renal dysfunction. Creatinine clearance has been about 70 ml/min for 9 years since the onset of RAT. The renal scintigraphic image has not changed since the onset. NS responded to prednisolone therapy initially and at the time of the relapse. Recent data have shown proteinuria levels of less than 0.2 g/day.

Key words: nephrotic syndrome, renal scintigram

Introduction

It is well known that nephrotic syndrome (NS) may be accompanied by thromboembolic complications in various arteries and veins (1). These complications may involve life-threatening episodes or may be responsible for intractability to glucocorticoid treatment. Renal vein thrombosis (RVT) has frequently been observed, especially in membranous glomerulonephritis (MGN) (2) and in certain steroid-resistant cases. Anticoagulants such as heparin or warfarin, have frequently been used in combination with glucocorticoids in the treatment of RVT, and favorable effects in the improvement of renal function and reduction of proteinuria have been observed (3, 4). Renal artery thrombosis (RAT), on the other hand, is rarely noted in NS patients (5–7). RAT may cause transient hypertension as a result of hyperreninism, and concomitant acute renal failure when bilateral renal arteries are involved (5, 8). As a result, the prognosis of NS following RAT is believed to be poor. We report a long-term follow-up case of RAT associated with MGN.

Case Report

A 64-year-old man came to the Second Department of Internal Medicine of the Jikei Hospital because of facial and pedal edema and was admitted in June 24, 1980. Daily urinary protein excretion was greater than 5.0 g, and serum total protein and albumin were 5.4 g/dl and 2.1 g/dl, respectively. Renal biopsy revealed MGN (Fig. 1), and creatinine clearance was 120 ml/min. No abnormal findings were found on intravenous pyelography (IVP), renogram, or renoscintgram. The patient was treated with prednisolone (30 mg/day), and proteinuria gradually decreased. After discharge from hospital, he complained of right lower abdominal pain and was surgically treated for appendicitis in September 1980. Pathological appendicitis was in the mild catalytic stage. IVP revealed a deformity in the right kidney contour. Aortography (Fig. 2) and a renal scintigram (Fig. 3) also showed a defect in the right upper pole of the kidney. Serum lactate dehydrogenase and plasma renin activity were 675 mg/dl and 5.8 ng/ml/h, respectively. Creatinine clearance was 60–80 ml/min. Based on these findings, this patient was thought to have RAT associated with NS, and was treated with urokinase at 60,000–120,000 unit/day for 12 days. However, no improvement of RAT was achieved when it was evaluated by renal scintigraphy. Subsequently proteinuria was reduced, and he was followed in the outpatient clinic. On January 8, 1982, prednisolone was discontinued under the condition of incomplete remission. The patient’s remission stage persisted for a further 5 years without any deterioration in renal function or RAT. From April 1986 urinary protein began to increase gradually and in February 1987, daily proteinuria and serum total protein...
Discussion

We report a rare case of RAT associated with MGN. The onset of RAT is clearly and easily recognized because it is more acute than in RVT. The symptoms of RAT consist of transient elevation of blood pressure, abdominal or flank pain, high fever, hematuria, and increased daily excretion of protein. These symptoms resemble RVT which does not give rise to an absolutely life-threatening state, such as acute renal failure or the hypertensive crises (9, 10). The present case was diagnosed as appendicitis, and the patient was treated surgically without treatment of RAT. Table 1 lists a few reports of RAT associated with NS including the present case. These cases are in moderately aged patients, and the renal histology is MGN in 2 cases, a minor glomerular lesion in one, and one case is unknown. All of these cases, excluding the present case, were resistant to a variety of drugs, and their serum albumin was less than 2.5 g/dl. The outcome of the nephrotic syndrome was incomplete remission in 2 cases, one patient developed massive proteinuria, and one patient died.
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Definite diagnosis of RAT depends on angiography, CT X-ray, or magnetic resonance scanning (16, 17), and sometimes renal scintigraphy. Characteristic clinical symptoms, such as abdominal or flank pain, oliguria, hematuria and an elevation in blood pressure due to hyperreninism also aid in the diagnostic decision (18). It is well known that iodine contrast media eventually causes nephrotoxicity in cases of renal dysfunction (15). Thereby, it is difficult to use contrast media in patients with RAT. Scintigraphy is ordinarily available to diagnose RAT despite renal dysfunction and can be used repeatedly for long-term follow-up. In the present case, scintigraphy yielded a great deal of information concerning the patient’s RAT, especially with regard to the long-term follow-up, and allowed us to determine the function and size.

Initial treatment of RAT associated with NS consists of high doses of urokinase to resolve the thrombosis and anticoagulants such as heparin or warfarin to prevent recurrent thrombosis (14). In general, when these agents are used too late, it is difficult to resolve the thrombosis.

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Table 1. Reported Cases of Renal Artery Thrombosis Associated with Nephrotic Syndrome

<table>
<thead>
<tr>
<th>Reports</th>
<th>Year</th>
<th>Age/Sex</th>
<th>Renal Histo.</th>
<th>Initial</th>
<th>Recent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kendall AG et al</td>
<td>1971</td>
<td>56/M</td>
<td>MGN</td>
<td>2.1</td>
<td>N.D.</td>
</tr>
<tr>
<td>et al (7)</td>
<td></td>
<td></td>
<td></td>
<td>1.2</td>
<td>N.D.</td>
</tr>
<tr>
<td>Temes XL et al</td>
<td>1979</td>
<td>55/M</td>
<td>MGL</td>
<td>1.0</td>
<td>2.2</td>
</tr>
<tr>
<td>et al (5)</td>
<td></td>
<td></td>
<td></td>
<td>2.4</td>
<td>N.D.</td>
</tr>
<tr>
<td>Pochet JM et al</td>
<td>1988</td>
<td>36/F</td>
<td>N.D</td>
<td>1.0</td>
<td>2.2</td>
</tr>
<tr>
<td>et al (6)</td>
<td></td>
<td></td>
<td></td>
<td>2.4</td>
<td>2.8</td>
</tr>
<tr>
<td>Present case</td>
<td>1990</td>
<td>64/M</td>
<td>MGN</td>
<td>0.9</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.1</td>
<td>3.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5.0-8.0</td>
<td>1.0</td>
</tr>
</tbody>
</table>


RAT is usually caused by trauma or angiography, and is observed rarely following aerobic exercise and hypercoagulation syndromes, such as in syphilis and other inflammatory diseases of various etiologies, and NS (11, 12). The pathogenesis of RAT in patients with NS has not been precisely defined, but it is well known that NS is accompanied by hypercoagulability consisting of low levels of antithrombin III (13), elevations of serum factor V and VIII (14), and an increased platelet count (7). The precise mechanism of thrombosis in NS is unknown. Since almost all cases of NS are associated with a hypercoagulable state and hyperlipidemia in the nephrotic stage, atherosclerosis associated with hyperlipidemia may have a powerful thrombosis-inducing effect in patients with NS. Thrombosis in NS is suggested to particularly occur in aged patients (15).

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The prognosis of RAT associated with NS depends on the degree of thrombosis, i.e., it is life-threatening in the case of bilateral renal occlusion due to thrombosis. RAT, however, frequently appears at a limited area in an unilateral kidney. In the present case, renal function must be compensated by a contralateral kidney at the onset of RAT.

References


