We experienced two cases of renal infarction with atrial fibrillation who presented with acute abdominal pain. On initial urinalysis, both patients showed no hematuria, but the plasma lactate dehydrogenase level was markedly elevated with little or no rise in plasma transaminases. Their diagnosis was confirmed by contrast-enhanced CT of the abdomen on the second and third days of the crisis. We immediately initiated anticoagulant therapy, resulting in successful prevention of new embolism. Contrast-enhanced CT should be considered if abdominal symptoms develop in patients with atrial fibrillation. Renal infarction could be diagnosed in the early course, even in cases with incomplete occlusion of the renal arteries and normal renal function. (Hypertens Res 2004; 27: 523–526)

Key Words: renal infarction, embolism, atrial fibrillation, hypertension

Case Report

Two Cases with Renal Infarction Diagnosed in the Early Course Using Contrast-Enhanced CT

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Introduction

Renal infarction is one of the causes of acute abdominal pain, but can be difficult to diagnose. Laboratory data and clinical features are often not helpful. Medical therapy is expected to improve renal function if occlusion of the renal arteries is incomplete or if effective thrombolysis is initiated within 90 to 180 min, which represents the ischemic tolerance of normal renal tissue (I). Therefore, diagnosis should be established much earlier. We here report two cases of renal infarction with atrial fibrillation, whose diagnosis was confirmed on the second and third days of the crisis by contrast-enhanced CT of the abdomen, and discuss the procedures for diagnosis of renal infarction.

Case Presentation

Case 1

A 70-year-old man visited Nagoya City University Hospital with the chief complaint of right lateral abdominal pain, and was admitted there. The day before admission, he was given a non-steroidal anti-inflammatory drug by his referring doctor to reduce his pain. He had been followed-up for atrial fibrillation and hypertension without any medication for 11 years.

Physical examination on admission revealed fever elevation (38°C) and atrial fibrillation (heart rate, approximately 86 bpm) in addition to right abdominal tenderness. His blood pressure was 156/93 mmHg. Laboratory examination of blood showed a marked rise in lactate dehydrogenase (LDH: 2,161 IU/l) and slight rises in two transaminases, aspartate aminotransferase (AST: 340 IU/l) and alanine aminotransferase (ALT: 275 IU/l), while alkaline phosphatase (AL-Pase) was not elevated (Table 1). The serum creatinine concentration was normal (1.0 mg/dl). White blood cell counts (10,000/µl), C reactive protein (1.70 mg/dl) and D-D dimer (1.8 µg/ml) were slightly elevated, while eosinophilia (0.0%) was not recognized. Plasma renin activity (2.5 ng/ml/h) was slightly elevated, while plasma aldosterone concentration (67 pg/ml) remained normal. Initial urinalysis with a dipstick showed no hematuria, but proteinuria (2+) was present. Microscopic examination of urine sediment showed no casts.
or epithelial cells. Abdominal ultrasonography indicated no abnormality of the gall bladder, common bile duct or kidneys. After admission, the patient’s abdominal pain was shifted to the right lateral side of the back.

On the second day of admission, we started treatment with antibiotics because white blood cell (12,300/µl) and C reactive protein (11.31 mg/dl) levels were further elevated. The plasma LDH and transaminases levels were gradually decreased, while C reactive protein remained high (upper panel of Fig. 1). Contrast-enhanced CT of the abdomen, performed on the second day, revealed segmental infarction of the ventral half of the right kidney, which appeared as a hypodense area (upper panel of Fig. 2). We made a diagnosis of renal infarction likely due to embolism from the heart, and then initiated continuous intravenous drip infusion of heparin (10,000 U/day), while stopping the antibiotic regimen.

On the 5th day of admission, the patient’s symptoms almost disappeared, and his diet was restarted. Oral warfarin was also started to target an international normalization ratio of 2–3. On the 8th day, hematuria became positive (3⁺) and proteinuria was still recognized (2⁺). Because a follow-up CT performed on the 11th day showed no change in the kidneys, heparinization was terminated. Echocardiography revealed left atrial dilatation and a slight degree of mitral stenosis. Transesophageal echocardiography showed no thrombus in the left atrium.

During his stay in our hospital, the patient’s blood pressure was approximately 138/86 mmHg without antihypertensive agents, and his serum creatinine concentration and Al-Pase level remained within the normal limits. There have been no clinical features of new embolism anywhere since the renal infarction. The patient was discharged on the 30th day of admission. CT on the 44th day revealed that renal perfusion was improved with a reduced hypodense area and slight atrophy of the right kidney.

**Case 2**

A 67-year old man visited a hospital with a chief complaint of abdominal pain and right flank pain, and was admitted there on suspicion of urolithiasis. He had been given medication to treat hypertension and paroxysmal atrial fibrillation for 6 years.

Physical examination on admission revealed high blood pressure (190/108 mmHg), atrial fibrillation (heart rate, approximately 78 bpm) and right flank pain. Laboratory data showed a rise in LDH (608 IU/l) as well as a slight rise in both AST (28 IU/l) and ALT (44 IU/l), while his Al-Pase level was within the normal limits (Table 1 and lower panel

**Table 1. Laboratory Findings on Admission in Two Cases**

<table>
<thead>
<tr>
<th>Normal range</th>
<th>Case 1 (70 years, male)</th>
<th>Case 2 (67 years, male)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDH (U/l)</td>
<td>119–229</td>
<td>2,161</td>
</tr>
<tr>
<td>AST (U/l)</td>
<td>10–33</td>
<td>340</td>
</tr>
<tr>
<td>ALT (U/l)</td>
<td>6–37</td>
<td>275</td>
</tr>
<tr>
<td>Al-Pase (U/l)</td>
<td>115–359</td>
<td>237</td>
</tr>
<tr>
<td>WBC</td>
<td>3,600–9,600</td>
<td>10,000</td>
</tr>
<tr>
<td>CRP (mg/dl)</td>
<td>&lt;0.3</td>
<td>1.70</td>
</tr>
<tr>
<td>D-D dimer (µg/ml)</td>
<td>&lt;0.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>negative</td>
<td>2⁺</td>
</tr>
<tr>
<td>Hematuria</td>
<td>negative</td>
<td>negative</td>
</tr>
</tbody>
</table>

LDH, lactate dehydrogenase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; Al-Pase, alkaline phosphatase; WBC, white blood cell count; CRP, C reactive protein.

**Fig. 1.** Clinical courses of two patients with renal infarction. The serum concentrations of lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) as well as C-reactive protein (CRP) are shown as functions of time after the onset of pain. The upper and lower panels indicate cases 1 and 2, respectively.
of Fig. 1). White blood cell counts (10,100/µl) and C reactive protein (1.40 mg/dl) were both slightly elevated without anemia or eosinophilia (1.0%). His serum creatinine concentration was 1.2 mg/dl. Urinalysis showed no hematuria, but proteinuria was evident (2+). There were no abnormalities in the microscopic examination of urinary sediment. On the second day of admission, contrast-enhanced CT of the abdomen revealed segmental infarction of the right kidney, which appeared as a wedge-shaped area (lower panel of Fig. 2). White blood cell (22,500/µl), C reactive protein (25.9 mg/dl) and LDH (1,635 IU/l) were markedly increased in addition to the appearance of hematuria (3+). He was diagnosed with renal infarction caused by embolism from the heart, and continuous drip infusion of heparin (10,000 U/day) was started. On the 5th day, he was transferred to Nagoya City University Hospital, although his symptoms had almost disappeared.

On the 8th day, there were improvements in white blood cell (7,600/µl), C reactive protein (14.09 mg/dl) and LDH (932 IU/l) levels, while his Al-Pase level was elevated to 1,216 IU/l. Since his D-D dimer level was 3.6 µg/ml, oral warfarin was added to his therapy. Echocardiography showed left ventricular hypertrophy and a mild degree of mitral regurgitation, while no thrombus was recognized in the left atrium. Heparinization was terminated on the 12th day because a follow-up CT revealed no change in the infarction area. His blood pressure was controlled to approximately 140/90 mmHg with combined use of angiotensin receptor antagonist, candesartan cilexetil, and a β-blocker, bisoprolol fumarate. The serum creatinine concentration remained within the normal limits during hospitalization, and hematuria became negative on the 21st day. In addition, the Al-Pase level was gradually decreased to within the normal limits by the 22nd day. He left our hospital on the 29th day because of the good clinical course.

**Discussion**

We here reported two cases of renal infarction that were considered-based on the presence of atrial fibrillation to be due to embolism from the heart; this diagnosis was confirmed by contrast-enhanced CT on the second day of admission, or the second (case 2) or third (case 1) days of the crisis. Both cases involved segmental infarction of the right kidney without renal dysfunction. After diagnosis in the early course of renal infarction, we immediately started anticoagulant therapy with intravenous heparinization followed by oral warfarin, and this regimen successfully prevented new embolism thereafter.

It is well known that thromboemboli usually originates from the heart, particularly in patients with arterial fibrillation (2, 3). Therefore, we thought that the renal infarction was probably caused by embolism from the heart, although thrombus was not found in the left atrium by echocardiography in either case. However, compared to cerebral circulation, the risk of isolated renal artery thromboembolism without involving cerebral circulation is relatively low (3). Despite the lack of significant clinical findings suggesting thromboembolism in other than the renal circulation, multiple old cerebral infarction was found by CT in the left basal ganglia and left radiate crown in case 2. Renal infarction has proven difficult to diagnose in the early stages, despite the fact that it often causes acute abdominal pain. Routine laboratory tests are often not helpful in the emergent setting. Urinalysis for hematuria is a simple diagnostic procedure, and it has previously been considered that hematuria is always present in cases of renal infarction (4). However, based on the present cases, in which hematuria was not apparent upon initial urinalysis, it would appear that hematuria is less common than previously thought. Its absence in the present cases probably reflects the marked reduction in blood flow to the infarcted area, which resulted in the local cessation of glomerular filtration and urine flow (5). There is, however, one finding that, in the appropriate clinical setting, is strong-
ly suggestive of renal infarction: a markedly elevated plasma LDH level, often more than 5 times the upper limit of normal, with little or no rise in plasma transaminases and Al-Pase (6). Such an elevation of plasma LDH was seen in both of the present cases. This pattern can also be seen in other conditions which are usually easily distinguishable from renal infarction: acute myocardial infarction, hemolysis, and renal transplant rejection. An elevation in blood pressure is also considered to characteristically develop during the first week from the onset, and is usually sustained for 2 to 3 weeks. Increased renin release plays an important role in elevating blood pressure, and therapy with inhibitors of the renin-angiotensin system is often effective. In case 1, plasma renin activity was only slightly elevated. In both cases, the level of hypertension was not markedly higher than that measured before the onset of renal infarction. In case 2, combination antihypertensive therapy with an angiotensin II receptor antagonist and β-blocker was not very effective. Thus, in our cases, the renin-angiotensin system did not seem to play an important role in hypertension, which might had been kept since before the onset of their crisis, independently of renal infarction, probably because ischemic area was small.

Although it has been reported that ultrasonography was useful to detect renal infarction as a hypo- and/or hyperechoic lesion in dogs with renal infarction (7), the abdominal ultrasonograms were normal in both of the present cases. And although recent developments in power Doppler ultrasonography allow us to clearly visualize the tertiary vessels (8), this technique is of limited value due to the difficulty in imaging the entire renal arteries (9). In our cases, contrast-enhanced CT was useful for making a diagnosis. Although renal arteriography remains the gold-standard method for obtaining a definitive diagnosis, contrast-enhanced CT could be an alternative method, especially if conservative management is preferred without acute intervention by thrombolysis or surgery. The choice of methods may be largely dependent on their availability in individual hospitals. There are other diseases that cause segmental hypoperfusion in the renal circulation, such as polyarteritis nodosa and renal artery aneurysm. The former could be excluded in our cases, because the inflammatory findings disappeared without the use of anti-inflammatory agents. As for renal artery aneurysm, this is a rare disorder, and hardly cause renal infarction.

Thrombolytic therapy with streptokinase or tissue-type plasminogen activator has been shown to be effective at lysing the occluding clot in most cases (10). However, it is possible that this modality is useful only when the ischemic renal tissue is still viable (1), which in most cases is within 12 h from the onset of renal ischemia. Our two cases were diagnosed on the 2nd and 3rd days, and their renal function remained almost normal. Therefore, such therapy was not indicated in either case, and they were treated conservatively with the anticoagulants heparin and warfarin. Unfortunately, the diagnosis of renal infarction is often difficult, and is frequently delayed because the significance of the characteristic findings is not recognized. In one study of 17 patients, for example, the time to diagnosis was usually 3 to 6 days, with only 5 patients being diagnosed on the first day (5). Classical studies by Hoxie and Cogan with 14,000 postmortem examinations found a 1.4% incidence of embolic renal infarction, but only 2 of these 205 cases had been recognized clinically (9). In our cases, both patients presented unspecified symptoms, but their laboratory findings, which were characteristic of renal infarction, led us to strongly suspect renal embolism. We were finally able to diagnose both cases with renal infarction on the second day of admission (the second and third days of crisis) by contrast-enhanced CT of the abdomen, despite a segmental infarction of the right kidney that did not cause renal dysfunction.

We conclude that renal infarction should be considered when abdominal symptoms develop in patients with atrial fibrillation, even if there is no hematuria. In addition, we emphasize that a markedly increased plasma LDH value and contrast-enhanced CT of the abdomen were essential for diagnosing renal infarction in the present cases.

References