Problems in the Initial Diagnosis of Renal Infarction

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We retrospectively analyzed 20 cases of renal infarction to identify the problems in tentatively diagnosing renal infarction. The subjects consisted of 12 outpatients and 8 inpatients whose diagnosis was confirmed by renal scintigram and/or contrast computed tomography. Renal infarction was tentatively diagnosed in only 4 of the 12 outpatients. Causes of hospitalization were cerebral emboli in 5 cases, peripheral emboli in the extremities in 2 cases and one case involved percutaneous transesophageal commissurotomy. On initial urinalysis, 11 cases (55%) showed less than 2+ hematuria using dipsticks to test for occult blood. The mean lactic dehydrogenase value was as high as 2,096 IU while the mean aspartate aminotransferase and mean alanine aminotransferase were 83.1 IU and 78.6 IU. Abdominal ultrasonography revealed abnormalities in only one of 18 cases. In conclusion, since only a moderate degree of hematuria was seen in about half the cases and it was difficult to detect renal abnormalities by ultrasonography, a tentative diagnosis of renal infarction may be difficult in some cases.

Key words: tentative diagnosis, enzymes, hematuria

Introduction

Renal infarction is one of the causes of acute abdominal pain and can be difficult to diagnose (1). Increased alanine aminotransferase (ALT) and total bilirubin are suggestive of hepatobiliary disease, increased creatine phosphokinase with epigastralgia suggests myocardial infarction, and hematuria with flank pain suggests ureteral stone. In addition to simple blood test, abdominal ultrasonography provides valuable information in patients with abdominal pain. The purpose of the present study was to retrospectively analyze clinical manifestations in 20 cases of renal infarction to elucidate the difficulty in making a tentative diagnosis.

Subjects and Methods

The subjects consisted of 10 males and 10 females with a mean patient age of 65 years. Twelve cases came to the outpatient department while renal infarction developed during hospitalization in the other 8 cases. Diagnosis of renal infarction was made by the presence of multiple or single perfusion defects either by renal scintigram and/or contrast computed tomography (2). We did not use abdominal ultrasonography with a color mode. Nineteen cases showed atrial fibrillation and one was in sinus rhythm. Seven cases were associated with left ventricular systolic dysfunction, 7 cases showed mitral stenosis, and 6 cases showed lone atrial fibrillation. Treatment included intravenous administration of heparin and/or urokinase; none of the patients received intraarterial thrombolytic agents.

Results

Tentative diagnosis in the outpatient group was acute abdomen in 5 cases, renal infarction in 4 cases, and renal stone, sepsis and fever of unknown origin in one each. Only one case developed recurrent arterial emboli to other organs in the succeeding month.

Among the 8 inpatients, reasons for hospitalization were cerebral infarction in 5, arterial emboli in the extremities in 2 and one case involved percutaneous transesophageal commissurotomy. Renal infarction developed an average of 7 days (ranging from 4 to 11 days) after admission in 5 cases of cerebral infarction when hyperosmotic agents had been administered to ameliorate cerebral edema. In the other 2 cases of peripheral emboli in the extremities, renal infarction developed 3 and 7 days after admission.

Abdominal or flank pain was one of the chief complaints in 10 of 14 patients whose mental status was not disturbed. General fatigue was also a major complaint in 3 cases. Asympt-
Clinical Manifestation of Renal Infarction

Symptomatic macro-hematuria with increased lactic dehydrogenase (LDH) were clues to the diagnosis in patients with disturbed mental status due to cerebrovascular disease.

On initial urinalysis using dipsticks to test for occult blood, 8 cases showed no hematuria, + in 2 cases, ++ in 3 cases, +++ in 7 cases (Table 1). Mean LDH was markedly and diffusely increased (2,096 IU: normal range 210-470) regardless of mild elevated both aspartate aminotransferase (AST) (mean 83 IU: normal range 11-32) and ALT (mean 79 IU: normal range 3-30) (Fig. 1). Peak creatinine was 2.1 mg/dl on average with a maximum of 8.7 mg/dl; it was less than 1.5 mg/dl in 8 cases. Among the remaining 12 cases, the creatinine level decreased in the following year except in one patient who died during the acute phase. Anticoagulation agents were administered just before renal infarction occurred in 12 patients with a mean prothrombin time of 14.4 seconds (Fig. 2). None of the patients showed renal failure necessitating hemodialysis. At the initial presentation, there were only 3 patients whose systolic blood pressure was more than 180 mmHg; we did not measure plasma renin activity.

Two patients died; one died within one month after admission because of concomitant superior mesenteric artery thrombosis and multiple organ failure, the other death was due to multiple cerebral infarction one year after the renal infarction.

Abdominal ultrasonography revealed low echoic area in the affected kidney in only one of 18 cases. Transthoracic echocardiography revealed left atrial thrombus in only one of the 20 cases while transesophageal echocardiography, performed within one week after renal infarction, revealed left atrial thrombus in 6 of 9 cases.

Quality of life after the insult in the present study depended on the extent of cerebral infarction and concomitant peripheral emboli.

### Discussion

Regarding patient selection in this study, a markedly increased LDH value was an important indication for renal scintigram or contrast computed tomography. Therefore, the cause of renal infarction in these patients may have been cardiac

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<th>Table 1. Presence of Occult Hematuria by Dipstick Test</th>
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![Figure 1. Histogram of the peak LDH, AST and ALT in the 20 cases (expressed as mean±SD).](image-url)
Renal infarction can be diagnosed as a hypoechoic lesion on ultrasonography (4). However, we were able to diagnose renal infarction by abdominal ultrasonography in only one case. Therefore, abdominal ultrasonography, a useful tool for the diagnosis of acute abdominal pain in an emergent setting, did not commit the diagnosis. Recent developments of power Doppler ultrasonography have enabled us to visualize tertiary vessels clearly (5). If we had used this type of ultrasonogram, more cases may have been diagnosed as renal infarction.

Only four cases were initially tentatively diagnosed as renal infarction among the outpatient group, presumably because of the lack of specific findings and poor recognition of renal infarction by internists. Extraordinarily increased LDH value with a diffuse nature without increased AST and ALT were characteristic laboratory signs of renal infarction (6). However, increased LDH only is considered nonspecific for some diseases and LDH measurement is not included as an emergency night-time examination in the majority of hospitals. In addition to the absence of significant hematuria in 50% of the cases and negative ultrasonography, we think that tentatively diagnosing renal infarction is difficult without measuring LDH especially during the night-time.

Renal infarction developed shortly after admission in 7 of the 8 inpatients, excluding the post-percutaneous transluminal commissurotomy patient; it occurred an average of 7 days after hospitalization in 5 patients with cerebral infarction when hyperosmotic agents were used to ameliorate cerebral edema. In one patient with peripheral emboli in the extremities whose renal infarction was diagnosed on the third day, renal infarction might have developed at the same time as the peripheral emboli. As the treatment for renal infarction in the 12 outpatients was to correct dehydration, only one case developed recurrent emboli in the succeeding month. Anticoagulation was performed in 12 cases just before renal infarction and a mean prothrombin time of 14.4 sec was noted. However, this was not sufficient to prevent renal infarction. Dehydration due to hyperosmotic agents in addition to the presence of atrial fibrillation may precipitate additional thromboembolism or recurrent emboli may develop shortly after the initial episode.

Renal function at presentation was not markedly disturbed in 40% of the patients and it normalized in the remaining 60% except for the one patient who died during the acute phase. This fact contrasted with presentation of renal infarction due to cholesterol emboli which progresses more insidiously and sometimes requires hemodialysis (7).

We conclude that renal infarction should be considered when abdominal symptoms develop in atrial fibrillation patients. Adequate anticoagulant therapy cannot completely prevent renal infarction. Precipitating dehydration is most dangerous especially after cerebral infarction.

References