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Renal and Splenic Infarction in a Patient with Familial Hypercholesterolemia and Previous Cerebral Infarction

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 47
Final Diagnosis: Acute renal and splenic infarction
Symptoms: Flank pain • low-grade fever
Medication: —
Clinical Procedure: CT scan
Specialty: Nephrology

Objective: Challenging differential diagnosis

Background: This is a case report of a male patient who presented with a history of right flank pain based on renal infarction. Initially the symptoms were misdiagnosed as acute pyelonephritis.

Case Report: A 47-year-old male with a history of familial hypercholesterolemia and cerebral infarction presented at the Emergency Department with a 3-day history of acute right-sided flank pain. Physical examination revealed hypertension, subfebrile temperature, and costovertebral angle tenderness. Blood tests were unremarkable except for renal impairment, a high C-reactive protein level of 215 mg/L (normal <8 mg/dL) and an elevated lactate dehydrogenase (LDH) of 1289 U/L (normal <248 U/L). Renal ultrasonography was normal. He was admitted with a presumed diagnosis of acute pyelonephritis and treated accordingly. However, 2 days later, we rejected this diagnosis as the urine culture was sterile. Based on the acute onset of symptoms and the initial high LDH, renal infarction was suspected. A computed tomography scan confirmed right-sided partial renal and splenic infarctions likely due to spreading emboli from atherosclerosis of the descending aorta.

Conclusions: Acute renal infarction is often missed or delayed as a diagnosis because patients often present with flank pain that can resemble more frequently encountered conditions such as pyelonephritis and nephrolithiasis. Renal infarction should be considered in cases with acute flank pain accompanied by (low-grade) fever, high LDH level, increased C-reactive protein level, hypertension, and renal impairment, especially in those patients with an increased risk of thromboembolism.

MeSH Keywords: Acute Kidney Injury • Flank Pain • Infarction • Splenic Infarction

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/911990

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Background

This is a case report of a male patient who presented at the Emergency Department (ED) with a history of right flank pain based on renal infarction. Initially, however, the patient's symptoms were misdiagnosed as acute pyelonephritis. This case review should be of interest to clinicians as acute renal infarction is often a missed or delayed diagnosis because patients usually present with flank pain that can resemble more frequently encountered acute conditions, such as pyelonephritis and nephrolithiasis, as in our case.

Case Report

A 47-year-old male, Caucasian, with weight of 72 kilograms (158 pounds) and height of 175 centimeters (69 inches), and a history of familial hypercholesterolemia, presented to the ED with a 3-day history of acute right-sided flank pain and general malaise. At the age of 42 years, 5 years before his presentation to our ED, he had a cerebral infarction in the area of the brain supplied by the left arteria cerebri media. He suffered from confusion and aphasia. However, he recovered from these symptoms. He was treated by a neurologist with aspirin, dipyridamole, and simvastatin. Additional workup at that time did not reveal a cardiologic disorder (echocardiogram and 24-hour Holter monitoring were normal). During follow-up he decided to discontinue his medication because he no longer considered it necessary as he was completely recovered from the symptoms of the cerebral infarction. Thus, at the time of presentation, he was not using any medication.

Physical examination revealed a blood pressure of 152/110 mmHg, a heart rate of 100 regular beats per minute, subfebrile temperature (37.7°C) and costovertebral angle tenderness. Blood tests were unremarkable except for a creatinine level of 185 µmol/L (normal <115 µmol/L), estimated glomerular filtration rate 34 mL/min/1.73 m^2, a C-reactive protein level of 215 mg/L (normal <8 mg/L) and an elevated lactate dehydrogenase (LDH) of 1289 U/L (normal <248 U/L). Urinalysis revealed hematuria (0–5 erythrocytes per high power field), pyuria (10–20 leucocytes per high power field) and proteinuria (1.2 gr/L). Renal ultrasonography showed normal kidneys without signs of a urinary obstruction or nephrolithiasis. He was admitted with a presumed diagnosis of acute pyelonephritis and treated accordingly. However, 2 days later, we rejected this diagnosis as the urine culture was sterile. Based on the acute onset of symptoms and the initial high LDH, renal infarction was suspected. A computed tomography scan confirmed right-sided partial renal and splenic infarctions (Figure 1) likely due to spreading emboli from atherosclerosis of the descending aorta (Figure 2). Additional workup did not reveal a cardiac disorder (i.e., normal echocardiogram and 24-hour Holter monitoring),

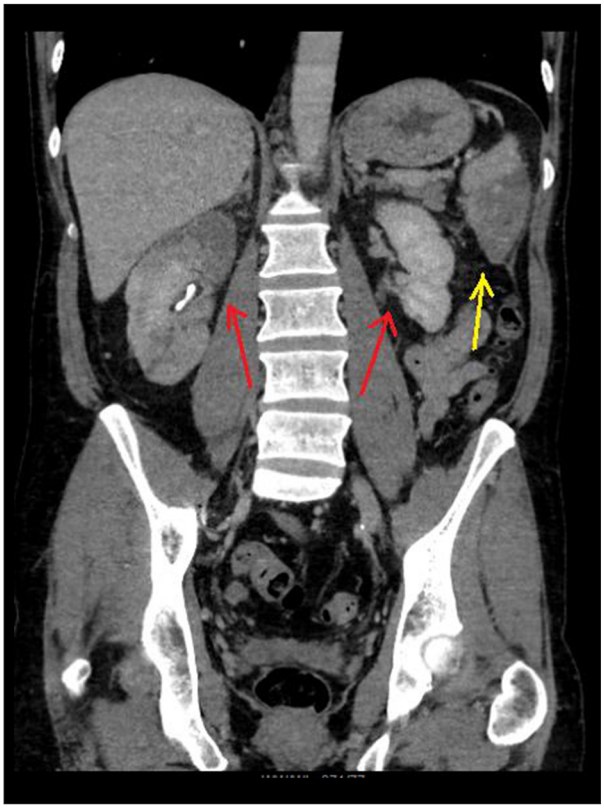

Figure 1. Computed tomography of a right-sided partial renal infarction. In addition, a partial splenic infarction can be seen. The red arrows indicate the kidneys and the yellow arrow the spleen.

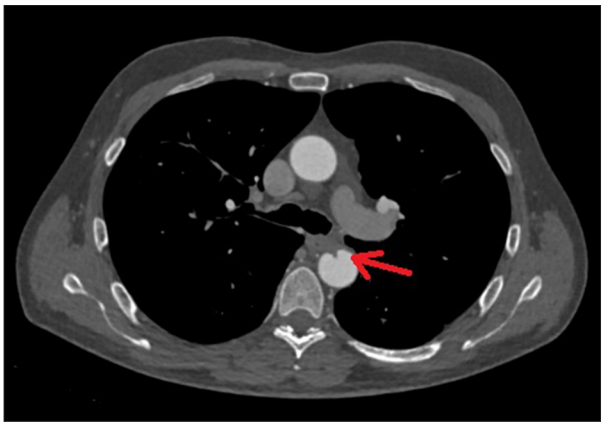

Figure 2. Atherosclerosis of the descending aorta, the most likely source of spreading emboli.

and no pro-coagulant disorders such as antiphospholipid syndrome. Therefore, the most likely cause of the renal infarction was spreading emboli from atherosclerosis of the descending aorta. We treated our patient with oral warfarin. His flank pain gradually resolved and 1.5 years after initial presentation, his kidney function was improved (estimated glomerular filtration

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rate 52 mL/min/1.73 m^2, as compared to an estimated glomerular filtration rate of 34 mL/min/1.73 m^2 at presentation).

Discussion

Diagnosis of acute renal infarction is often missed or delayed because the patient usually presents with flank pain that can resemble more frequently encountered acute conditions such as pyelonephritis and nephrolithiasis [1]. The mean duration from the onset of symptoms to the diagnosis of renal infarction is often more than 2 days [2], as in our case.

In a series of 438 patients with renal infarction diagnosed between 1993 and 2013, flank pain was seen in 50% of cases, abdominal pain in 53% of cases, nausea in 17% of cases, vomiting in 13%of cases, and fever in 10% of cases; 32% of cases had hematuria and the mean LDH was 656 U/L (normal <248 U/L) [3]. In another case series of 94 patients, abdominal and/or flank pain was seen in 97% of cases, fever in 20% of cases, hypertension in 48% of cases, elevated LDH in 91% of cases, elevated C-reactive protein level in 78% of cases, and renal impairment in 40% of cases; LDH concentrations remained above the upper limit of the normal range until the 15th day of first clinical presentation [4]. In another case series involving 20 patients, all the patients had either abdominal or flank pain and tenderness, and 95% had an elevated serum LDH level with a mean of 812 U/L [5].

LDH seems to be a useful marker in considering renal infarction in the differential diagnosis as neither nephrolithiasis nor pyelonephritis is associated with an elevation in LDH [1]. Urinalysis in acute pyelonephritis typically reveals pyuria and/or bacteriuria, while in renal infarction, urinalysis typically shows hematuria [1,3,6]. In contrast to pyelonephritis, renal infarction is also usually accompanied by hypertension, assumed to be renin-mediated [1,4].

The major cause of renal infarction includes cardioembolic disease (especially atrial fibrillation) or a hypercoagulable state [3,7,8]. Clinicians should be suspicious when patients with risk factors for emboli, such as atrial fibrillation, present with sudden onset flank pain, especially when an initial diagnosis of nephrolithiasis or pyelonephritis is rejected during follow-up [7].

Because in the majority of cases a thromboembolic phenomenon is the cause of the renal infarction, a cardiac workup is indicated to test for any thromboembolic phenomenon (e.g., echocardiography and Holter monitoring to assess for arrhythmias). Furthermore, a hypercoagulability screen should be considered (e.g., antiphospholipid antibodies, factor deficiencies like Factor V Leiden, antithrombin III activity, proteins C and S, homocysteine levels, diagnostic screen for a malignancy or polycythemia vera).

Our patient had an acute kidney injury at presentation. Although his kidney function improved after 1.5 years of follow-up, it still remained diminished. In one published case series, acute kidney injury due to acute renal infarction occurred in 76% of patients [8]. The estimated glomerular filtration rate decreased to ~70% and recovered to ~80% of the original value after 1 year. Another study found that while most patients recovered from acute kidney injury, 7% of the patients developed persistent renal impairment (chronic kidney disease progression), which was closely correlated to the magnitude of the infarct size [9]. Similarly, another study observed that although acute kidney injury was present in 40.7% of the study patients, long-term renal outcome was relatively good [10]. The volume of the infarction has been suggested to be a factor in the degree of renal function decline in acute renal infarction [11].

The optimal treatment of renal infarction is uncertain, and comparative studies are lacking. Reported strategies include anticoagulation, endovascular therapy, and open surgery [1,3,4]. Oral warfarin is regarded as the standard anticoagulation regimen with the rationale to prevent future events, but there is no literature comparing outcomes to those of untreated patients [1]. Guidelines for use of direct oral anticoagulants (DOACs) need to be determined. Percutaneous endovascular therapy might be of benefit for patients with acute renal artery occlusion involving the main renal artery or segmental branch, that is diagnosed early in the course of renal infarction [1]. Based on the current literature, there seems to be no indication for surgical therapy for renal infarction with a possible exception in the case of a trauma patient, where surgery has to be performed for other reasons [1].

Conclusions

In summary, acute renal infarction is often a missed or delayed diagnosis because patients usually present with flank pain that can resemble more frequently encountered conditions such as pyelonephritis and nephrolithiasis. Renal infarction should be considered in those patients with acute flank pain accompanied by (low grade) fever, high LDH level, increased C-reactive protein level, hypertension, and renal impairment, especially in those patients with an increased risk of thromboembolism.

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