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Imaging Teaching Case

Renal Infarction Versus Pyelonephritis in a Woman Presenting With Fever and Flank Pain

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Patients with fever, flank pain, and dysuria frequently are encountered in the emergency department. Acute pyelonephritis is the most likely diagnosis; however, its clinical and radiologic presentation consistently overlap with that of acute renal infarction. Ultrasound is unable to distinguish early infarction from nonabscessed acute pyelonephritis. Hence, computed tomography or magnetic resonance imaging are needed. We report the case of a 68-year-old woman who presented with fever, flank pain, and dysuria, along with respiratory distress and tachycardia. Elevated values for inflammatory indexes suggested a diagnosis of acute pyelonephritis, and subsequent contrast-enhanced computed tomography showed hypodense wedge-shaped areas in both kidneys. However, the presence of a thin rim of capsular enhancement (cortical rim sign), the absence of perirenal inflammatory changes, and the location of the lesions apart from defined calyces suggested the alternative diagnosis of renal infarction. The underlying cause was not identified until an episode of acute dyspnea revealed paroxysmal arrhythmia. Our case demonstrates that a thorough knowledge of the imaging findings of renal infarction and acute pyelonephritis is essential to correctly making the diagnosis.
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INDEX WORDS: Multifocal kidney infarction; acute renal infarction; acute pyelonephritis; upper urinary tract infection; cardiac-embolic disease, transient arrhythmia; computed tomography; renal imaging; renal atheroembolic disease.

INTRODUCTION

Renal infarction is an uncommon occurrence and an example of clinical mimicry, especially in severe cases. It is characterized by acute and excruciating flank pain, fever, and increased values of inflammatory indexes.[1,2] To date, few studies assessed its frequency of presentation in the emergency department, reporting a prevalence of 0.004%-0.007%.[1,3,4] Acute pyelonephritis is a much more common cause of acute flank pain with fever and systemic signs of inflammation.[5-7] However, other different, severe, and even rare causes must be excluded, including aortic dissection, renal cancer, retroperitoneal fibrosis, and pleuritis.[5-7]

Diagnostic imaging is crucial for obtaining the correct diagnosis. Ultrasound examination is not able to detect an early infarction or acute pyelonephritis until necrotic or abscessed areas occur.[5,6,8] Even after abscess formation, the distinction between renal infarction and acute pyelonephritis may be not possible because the ultrasound appearance of necrotic tissue often is indistinguishable from an abscess. Therefore, computed tomography (CT) or magnetic resonance imaging usually are employed, although the differential diagnosis may still be challenging.[9-11] Furthermore, the correct diagnosis of renal infarction is important because treatment planning and prevention of recurrences are based on identification of the underlying cause. The causes are numerous because renal infarction occurs in a variety of clinical settings. However, the cause remains unknown for 20% to >50% of cases.[1,12,13]

We present the case of a 68-year-old woman referred to the emergency department for fever, flank pain, and increased inflammatory index values, whose clinical presentation demonstrated the difficulties diagnosing renal infarction and distinguishing it from acute pyelonephritis.

CASE REPORT

Clinical History and Initial Laboratory Data

A 68-year-old woman presented with a 4-day history of severe left flank pain, nausea, malaise, and fever unresponsive to oral antibiotics to our emergency department. She also reported dysuria and dark urine during the previous days, together with respiratory distress and tachycardia. The patient's medical history was otherwise unremarkable, except for a 15-year history of hypertension treated with diuretics, angiotensin-converting enzyme inhibitors and β-blockers. On physical examination, her temperature was 38°C, pulse was regular at 100 beats/min, and blood pressure was 120/80 mm Hg. Laboratory studies showed white blood cell count of $11.5 \times 10^3/\mu L$, hemoglobin level of 13.9 g/dL, C-reactive protein level of 15.5 mg/dL, lactate dehydrogenase (LDH)

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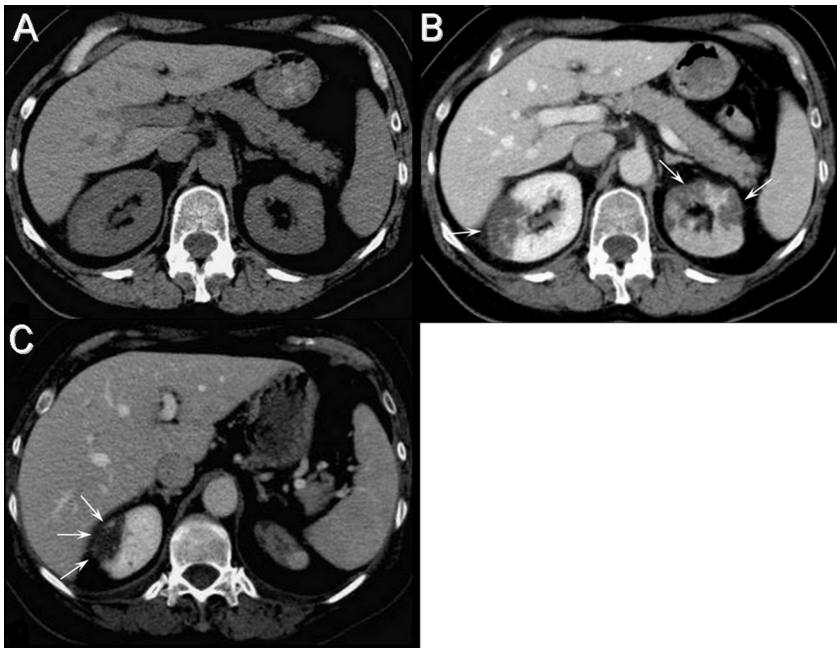

Figure 1. Unenhanced and multiphase enhanced helical computed tomographic (CT) scans obtained 4 days into hospitalization. (A) Precontrast CT scan shows homogeneous attenuation of kidneys and no calcifications. (B) Nephrographic contrast-enhanced phase demonstrates multiple focal wedge-shaped areas of decreased enhancement in both mid-kidneys with loss of corticomedullary differentiation (arrows). The left kidney demonstrates global decreased uptake of contrast material. Note the normal and homogeneous aspect of fat around the kidneys, with no stranding or fluid collections throughout the perinephric space. (C) Nephrographic enhanced scan shows a thin rim of capsular enhancement (the cortical rim sign; arrows), a finding consisting with a focal infarct.

level of 1,283 U/L, and serum creatinine level of 1.07 mg/dL, corresponding to estimated glomerular filtration rate of 53.5 mL/min/1.73 m^2 as calculated by the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation. Urinalysis showed the following: leukocytes (++), erythrocytes (+), and proteinuria with protein excretion of 10 mg/dL. She was hospitalized with a presumptive diagnosis of acute pyelonephritis and treated with ceftriaxone and amikacin. Four days later, the patient was afebrile, although analgesic treatment was continued for the severe back pain.

Echocardiography performed because of the patient's known history of rheumatic fever showed mild ventricular hypertrophy with normal wall motion (ejection fraction, 60%) and moderate mitral insufficiency and stenosis (valvular area according to pressure half-time, 2.44 cm^2; median transvalvular gradient, 4.4 mm Hg).

Imaging Studies

Upon admission, renal ultrasound showed a normal right kidney and a left kidney of normal size with irregular margins and an inferior scar. Four days after hospitalization, the patient underwent multiphase contrast-enhanced CT (Fig 1) that showed several hypodense wedge-shaped areas in both kidneys (Fig 1B and C) bound by a thin rim of capsular enhancement (Fig 1C).

In addition, no signs of perirenal inflammation were observed (with no stranding or fluid collections throughout the perinephric space; Figs 1 and 2), and no abnormalities of the abdominal aorta and renal arteries were demonstrated (not shown).

Diagnosis

The diagnosis suggested by imaging findings was multiple bilateral renal infarctions.

Clinical Follow-up

Subsequently, the patient underwent further evaluation to identify the underlying cause of renal infarction. No signs of collagen vascular disease or vasculitis were found, with negative antinuclear antibodies, anti-DNA, rheumatoid factor, lupus-like anticoagulant, anti-cardiolipin antibodies, and antineutrophil

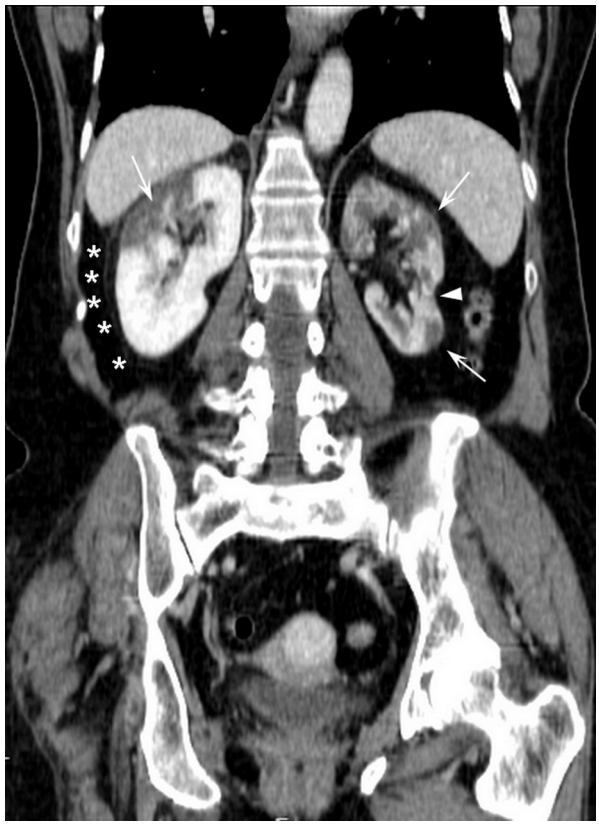

Figure 2. Coronal reformatting of the nephrographic phase obtained at the same time as Fig 1 shows multiple round and wedge-shaped areas of hypoattenuation in both kidneys with poor corticomedullary differentiation (arrows). A preexistent cortical scar with parenchymal thinning is seen in the lower left kidney (arrowhead). No obliteration of the perirenal fat planes or thickening of the perirenal fascia is observed (asterisks).