Acute Renal Infarction: An Underdiagnosed Disorder

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Abstract

Acute renal infarction is usually diagnosed when the triad of flank pain, hematuria and elevated lactate dehydrogenase is observed. Since the symptoms are non specific, diagnosis requires high degree of suspicion or may be missed or confused with renal colic. The incidence of ARI in emergency department visits is 0.007 % whereas in autopsy series is reported 1.4 % indicating the condition goes undiagnosed frequently. With the increase use of contrast enhanced computed tomography and magnetic resonance angiography the accuracy might be improving at the present time.

Keywords: Acute renal infarction; Flank pain; Hematuria; Lactate dehydrogenase; Contrast-enhanced computed tomography

Introduction

The incidence of acute renal infarction (ARI) in the emergency department (ED) visits has been reported as 7 in 100,000 \cite{1}. It was found in 1.4\% of cases in an autopsy study, whereas clinical diagnosis was made in 0.014\% of those patients \cite{2} suggesting that the condition is frequently missed and that a high degree of suspicion is needed to arrive at the diagnosis.

Case Report

Case 1

A 47-year-old man presented with right lower abdominal and back pain for three days associated with vomiting. The pain was throbbing in nature, had no radiation, and had an intensity of 10/10. He had smoked at least 5 cigarettes daily for the past 25 years, used heroin in the past, and currently received methadone 120 mg daily. Physical examination was unremarkable except for severe right flank pain. His vital signs showed a blood pressure was 150/80 mmHg, pulse 60 beats per minute and regular, respirations 16 per minute, and temperature 36.5 °C.

Laboratory tests revealed hemoglobin of 14 g/dl, leucocyte count 7600 per mm\textsuperscript{3}, neutrophils 80.5\%, platelets 241000 per mm\textsuperscript{3}, serum creatinine 1.3 mg/dl, aspartate aminotransferase 76 units/liter (normal 10 - 42), alanine aminotransferase was 65 units/liter (normal 10 - 40), lactate dehydrogenase (LD) 657 units/liter (normal 91 - 180). Urinalysis was strongly positive for blood, and urine protein was 300 mg/dl. Total cholesterol was 196 mg/dl (normal 140 - 200), low density lipoprotein (LDL) 143 mg/dl (normal < 130), triglyceride 68 mg/dl (normal 35 - 160) and homocysteine 6.9 micromoles per liter (normal < 11.4).

The electrocardiogram showed sinus bradycardia at 52 beats per minute and inverted T waves in the inferior wall and chest leads. Echocardiogram revealed normal ejection fraction and no evidence of thrombus or vegetations. Impaired relaxation pattern of the left ventricular diastolic filling was observed.

Contrast-enhanced computed tomography (CECT) of
the abdomen and pelvis showed right renal infarct with occlusion of the posterior branch of the right renal artery (Fig. 1). Gadolinium-enhanced magnetic resonance angiography (MRA) of the abdominal aorta and renal arteries confirmed right renal infarction involving the upper and posterior cortex.

He was anticoagulated with heparin and warfarin. His symptom improved significantly, and was discharged.

Case 2

An 89-year-old woman presented with right lower quadrant pain that started suddenly in the early morning hours, sharp in quality, 5/10 in severity, non radiating, and associated with nausea but no vomiting.

Her past medical history included atrial fibrillation, hypertension, congestive heart failure, and osteoarthritis. Medications on presentation were amlodipine, metolazone, enalapril, and aspirin. She had been taking warfarin in the past but had discontinued after an episode of toxicity. She never smoked or abused alcohol or illicit drugs. Her blood pressure was 160/60 mmHg, pulse 67 beats per minute, irregular, respirations 16 per minute, and temperature 36.2 °C. She had right lower quadrant tenderness with no guarding or rebound. Bowel sounds were present.

Laboratory tests yielded a serum creatinine of 0.7 mg/dl, hemoglobin13.1 g/dl, leukocyte count 6400 per mm³, platelets 213000 per liter, lactate dehydrogenase 1957 units per liter. Urinalysis was strongly positive for blood, and urine protein was 100 mg/dl.

Electrocardiogram showed atrial fibrillation. Abdominal sonogram did not reveal abnormalities.

CECT scan of the abdomen and pelvis revealed poor enhancement in the lower two thirds of the right kidney and a focal filling defect in the distal right renal artery, suggestive of thromboembolism causing the infarct of the right lower two thirds of the kidney. Echocardiogram showed no evidence of thrombus or vegetations and the ejection fraction was 55%. She was treated with heparin drip and later discharged on warfarin.

Case 3

A 41-year-old male was admitted to the hospital after suffering a gunshot wound, and had penetrating and blunt injuries. Past medical history and social history were unobtainable. On presentation blood pressure was 59/45 mmHg, pulse 98 beats per minute, respirations 40 per minute, and temperature 34.2 °C. He underwent splenectomy with repair of mesenteric injuries as well serosal tears of his colon.

Initial laboratory data showed a serum creatinine of 1 mg/dl, hemoglobin12.4 g/dl, leukocyte count 7500 per mm³, platelets 153000/L. No urinalysis was available. Computed tomography angiography (CTA) revealed hypoperfusion of the left kidney with multiple areas of wedge-shaped hypodensities representing possible infarcts. The left renal artery only demonstrated contrast enhancement on some of its branches suggesting renal artery injury.

Anticoagulation was initiated with heparin drip. Later he developed acute kidney injury and underwent multiple sessions of hemodialysis until renal function recovered. A follow-up CECT was done and revealed normal kidneys, at which time the heparin infusion was discontinued.

Discussion

The clinical presentation of ARI is nonspecific and may be confused with that of a renal colic. Patients commonly present to the ED with flank or abdominal pain associated with fever, nausea or vomiting, or may only present with acute onset of hypertension [4]. The prevalence of spontaneous kidney infarction in patients with new onset hypertension is 3% and the incidence is 0.18% per year [5]. The classic triad consists of persisting abdominal or flank pain; elevated serum LD and/or microscopic hematuria; and high risk of thromboembolism.

Laboratory studies reveal hematuria and proteinuria in most instances, and the blood levels of LD and the amino-transferases are frequently raised. Absence of hematuria indicates more serious loss of renal function [6]. Measurement of urinary LD may also be helpful.

The diagnosis is confirmed with CECT, angiography, or isotope scan. The sensitivity of computed tomography (CT) is 80%, renal isotope 97% and angiography is 100% [6]. CT characteristically shows a perfusion defect with a mass effect representing the zone of infarct, sometimes surrounded by a cortical rim sign indicative of the zone of ischemia (functioning nephrons supplied blood via capsular collaterals). The isotope scan demonstrates absent or reduced blood flows to the affected area. Angiography can clearly delineate...
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3. Bolderman R et al [13] identified sixteen patients (59%) over 2.5 years who developed ARI without any structural or arterial urokinase infusion, but no improvement was observed. The patient had a serum creatinine of 3.5 with a contralateral contracted kidney, required maintenance dialysis. Angioplasty and stent placement had been tried in some cases. The success of the intervention is limited by the duration of the ischemia.

The prognosis of ARI is determined by the etiology and the size of the infarct. In most series of unilateral disease there was not a significant loss of kidney function most likely due to compensatory hypertrophy of the remaining kidney tissue, but in most instances of bilateral infarct complete loss of renal function follows. Some patients develop persistent hypertension following ARI [5].

Conclusion

A high degree of suspicion remains the key for early diagnosis of ARI. Patients presenting with flank pain, hematuria and proteinuria as well as elevated level of LD should undergo CECT scan to obtain confirmation. Early anticoagulation may reduce symptoms and help preserve kidney function by preventing recurrence.

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Conflict of Interest

None.

References


