

case report

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Renal Artery Thromboembolism: A Rare Cause of Flank Pain

Introduction

Due to infrequent and nonspecific presentation, renal artery embolism is an uncommon cause of flank pain and its diagnosis is often mistaken as renal or ureteral colic. Consequently, better knowledge of this rare clinical entity and its potential complications is vital to proper diagnosis and management. We describe below a case of acute renal artery thromboembolism in a patient with newly diagnosed atrial fibrillation.

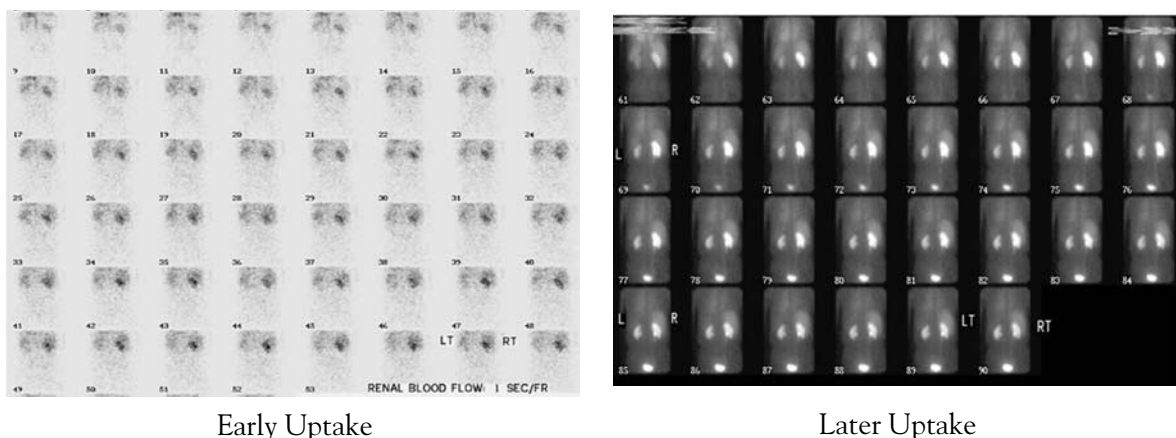
Case Report

A 76-year-old male presented to the Emergency Department (ED) with complaints of left-sided flank pain of one day's duration, in addition to chronic dyspnea upon exertion. The pain was radiating down to the left lower quadrant and associated with nausea. The patient was documented to have a regular pulse and only mild tenderness in the left lower abdomen on examination. He was found to have an elevated creatinine level of 1.6 mg/dL from his baseline value of 1.1 mg/dL. Urine analysis showed mild hematuria, pyuria and negative leukocyte esterase. Computed tomography (CT) of the abdomen and pelvis was negative for any stone but revealed moderate thickening of the bladder wall. The patient was discharged with ciprofloxacin for five days.

Within 10 days of this initial visit, the patient returned to the ED with complaints of worsening dyspnea and intermittent abdominal pain. The patient also had orthopnea and substernal chest pressure. Upon examination, he was found to have hypertension, irregularly irregular heartbeat, bilateral lung crackles and pitting pedal edema. Prior to this presentation, his hypertension had been effectively managed with metoprolol. Chest radiograph revealed features of congestive heart failure and an electrocardiogram disclosed new-onset atrial fibrillation. The patient's brain natriuretic peptide was high (1260 pg/mL); he had mild left ventricular (LV) hypertrophy, mild left atrial enlargement, and preserved LV systolic function on echocardiogram. He was admitted to the hospital for management of the congestive heart failure and treated with diuretics. Additionally, he was seen by a cardiologist and started on Coumadin. Due to his persistently elevated creatinine, the renal service was consulted.

After two days of hospitalization the patient's dyspnea and pedal edema improved. CBC was within normal limits, but blood urea nitrogen and creatinine levels were 32 and 1.8 mg/dL respectively. Given the patient's clinical history, our initial differential diagnosis

Figure 1. Greatly prolonged peak activity time and elimination half time on left side



included acute kidney injury secondary to worsening congestive heart failure, or renal artery embolus. He was noted to have an elevated level of serum lactate dehydrogenase of 235 IU/L and normal serum uric acid level. A renal scan (Figure 1) was performed which disclosed markedly abnormally diminished left renal function.

Due to an abnormal renal scan, a CT angiogram of the kidneys (Figure 2) was performed. This indicated a complete occlusion of the left renal artery secondary to a roughly 2 cm long acute thrombus with significant atherosclerotic stenosis in the left renal artery. There was probable collateral flow from the left diaphragmatic or splenic artery causing delayed excretion from the left kidney. In addition, there was mild diffuse atherosclerotic disease of the aorta and right renal artery. Both

kidneys were normal in size on initial noncontrast CT. Due to the presence of collateral circulation, normal size kidneys and the short duration of clinical course, the plan of action for this patient was revascularization. The left renal artery was injected with 100 mcg of nitroglycerin, and 3000 units of intravenous heparin. Subsequently, two express stents of 1.5 cm length were successfully placed across the stenosis in the left renal artery. A completion arteriogram confirmed a widely patent left renal artery.

The patient's renal failure remained stable after revascularization and a transesophageal echocardiogram (Figure 3) revealed a thrombus in the left atrial appendage. It is difficult to say if the renal artery thrombus originated from the left atrium as this patient had significant atherosclerotic disease of the

Figure 2. A complete occlusion of left renal artery

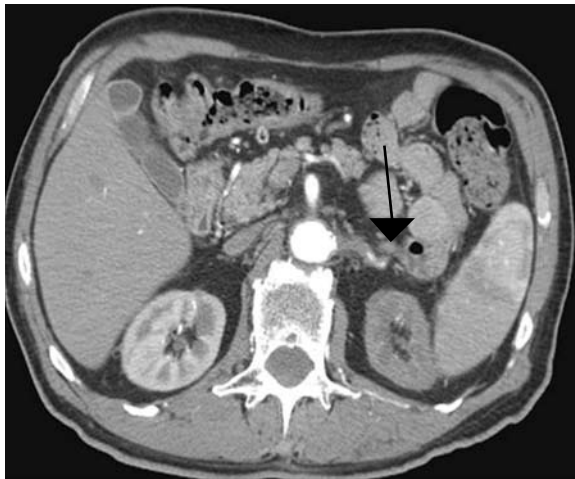


Figure 3. Thrombus in left atrial appendage.

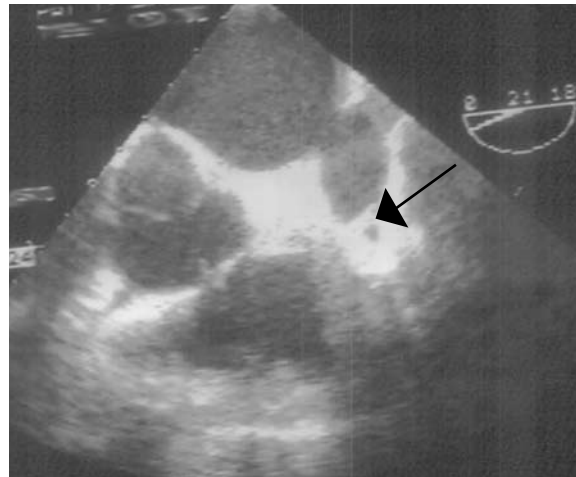
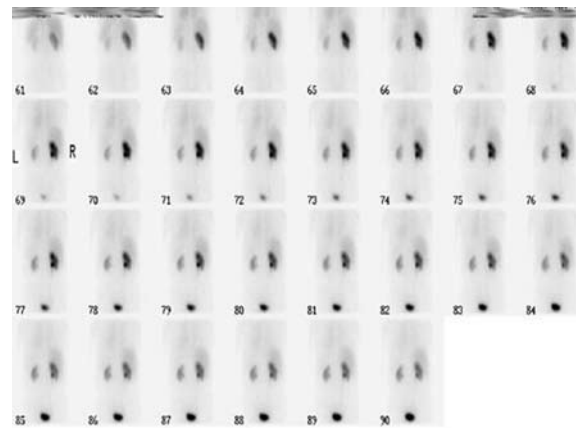
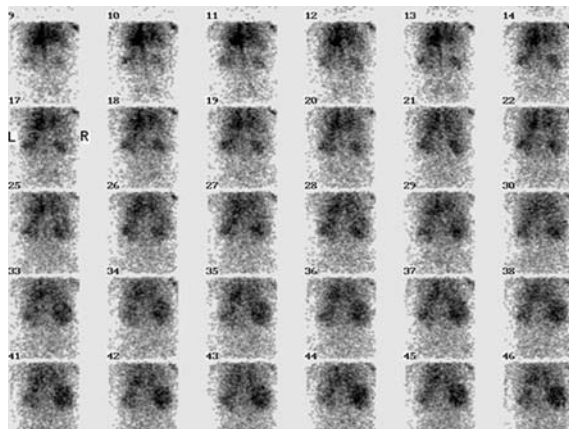


Figure 4. Mild improvement in left renal function



aorta and renal arteries. The patient was discharged and put on a regimen of anticoagulation. Over the next four months, his creatinine decreased to 1.6 mg/dL and his glomerular filtration rate improved from 39 to 45 mL/min/kg. A repeat renal scan (Figure 4) two months post discharge showed minimal improvement in the left renal function. Split renal uptake demonstrated 80% of activity within the right kidney and 20% in the left kidney this time in comparison to 15% activity on the left side before revascularization.

Discussion

Acute radiating flank pain is a common clinical feature of urinary calculus disease, but may also occur due to other more rare and serious intra-abdominal pathologies.¹ Acute renal artery thromboembolism, a rare disease, may manifest as flank pain similar to renal colic. The patient described in this case presented to the emergency room with unilateral flank pain which was mistaken for renal colic.

Acute renal artery thromboembolism is a serious problem. Rapid diagnosis and treatment is necessary, as ischemia can cause irreversible kidney damage within minutes.² In most cases, the source of embolus is the heart. Common contributing cardiac risk factors include atrial fibrillation, infarction history or potential, mitral stenosis, infective endocarditis, septal defect, hypertension and ischemic heart diseases.² Occasionally, peripheral atherosclerotic disease and renal artery stenosis may increase the risk of a sudden occlusion of the renal artery due to thrombus formation. In the present case, we assume that the source of renal artery thromboembolism could be from the heart or from aortic atherosclerosis. Our patient had new onset atrial fibrillation and intra-atrial thrombus on his echocardiogram which highly increases suspicion for a cardiac source.

Laboratory findings are typically nonspecific. Elevated serum lactate dehydrogenase (LDH I, II) are considered good biological markers of screening in patients suffering from thromboembolism.³ Serum creatinine can be abnormal in cases of bilateral embolism or unilateral embolism in the one functional kidney. A commonly used diagnostic modality in patients presenting with unilateral flank pain and hematuria is noncontrast CT. The accuracy of this technique in diagnosing urolithiasis has been determined to be as high as 97%, with a sensitivity of 95% and specificity of 98%; however, this has no role in diagnosing renal artery disease.⁴ A noninvasive imaging study, such as renal scintigraphy, ultrasonography with color Doppler, should be the initial study of choice.⁵

Despite technological advances in noninvasive methods for evaluating the renal arteries, definitive diagnosis of renal artery pathology continues to depend on visualization of renal artery anatomy with techniques such as MRI, renal CT, magnetic resonance angiogram, and transfemoral arteriography. Occasionally, contrast-induced nephropathy following angiography may itself lead to acute renal failure.²

Conclusion

Treatment of renal artery thromboembolism includes embolus extraction, thrombolysis and percutaneous transluminal renal angioplasty.⁶ Presence of collateral circulation can prolong the survival time of the kidney so that revascularization can be successful even after long durations. Consequently the duration of arterial occlusion compatible with recovery of renal function is patchy. Despite successful revascularization, kidney function is rarely restored to the level prior to the renal artery thromboembolism. This was certainly the finding in our case. In conclusion, renal artery thromboembolism must be considered in the differential diagnosis of acute flank pain in patients with certain risk factors such as atrial fibrillation, ischemic heart disease, and atherosclerotic vascular disease.

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Answers to the CME Questions

1a. d
1b. e
1c. b
1d. a

2a. b
2b. b
2c. b
2d. b
2e. b

3a. d
3b. a
3c. b
3d. e

4a. d
4b. a
4c. a
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5a. b
5b. c
5c. d
5d. a

6a. c
6b. a
6c. a
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