

Idiopathic Renal Infarction: Suspicion is the Key

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Renal infarction is relatively a rare condition that is often linked to the presence of underlying cardiac disease such as atrial fibrillation and valvular heart disease. While the classic presentation of renal infarction includes persistent abdominal or loin pain with high serum levels of lactate dehydrogenase (LDH), microscopic hematuria and high C-reactive protein (CRP), patients can present without these features. It is important to have high index of suspicion for this condition in patients who present with unexplained back or abdominal pain, even if there are no known traditional risk factors. Herein, we describe the case of a 42-year-old man who presented with abdominal pain and found to have acute right and subacute bilateral renal infarcts. No cause was identified despite exhaustive work up. Interestingly, his urinalysis, LDH and CRP were within normal limits.

Key words: *anticoagulation, idiopathic, renal infarction*

42-year-old Caucasian man with a history of hypertension has presented to our institution with nausea and abdominal pain for 2 days. The pain was bilateral in the upper abdominal quadrants radiating to the back, right more than the left. There was no history of kidney stones. Approximately 6 months prior to presentation, he was diagnosed with deep venous thrombosis of the right leg and was treated with warfarin for 3 months. Notably, he had similar pain at that time but of lesser intensity and no abdominal imaging was done. His home medications include Lisinopril 40 mg per day and simvastatin 20 mg per day.

On examination, he was afebrile, blood pressure was 144/86 and pulse rate 98 bpm. There was tenderness over bilateral costo-vertebral angles and paraspinal regions without peritoneal signs. Urinalysis was negative for blood or pyuria. Total leucocyte was 8 thou/cu.mm, hemoglobin 13 g/dL, platelet count 350 thou/cu.mm and serum creatinine 0.7 mg/dL. CT scan of the abdomen without contrast was done primarily to rule out urolithiasis and it showed possible bilateral renal infarcts. Magnetic resonance imaging

(MRI) and angiography (MRA) with contrast of the abdomen was performed to further characterize the lesions and look for any vascular pathology. Subacute bilateral focal infarcts were seen in both the kidneys with a new wedge-shaped infarct in the right kidney (Fig. 1, arrow). Aorta and branch vessels demonstrated normal vascular enhancement without evidence for wall thickness, aneurysm or stenosis (Fig. 2). EKG showed sinus rhythm and telemetry monitoring during his inpatient stay did not show any arrhythmia. ANA, ANCA, viral hepatitis panel, HIV test were negative. CRP and LDH were normal. Hypercoagulability work up was essentially negative. Etiology of his renal infarction could not be identified in spite of extensive work up and was discharged on oral anti-coagulation.

Discussion

Renal infarction is a rare condition that was reported as early as 1856. Most common causes of renal infarction are cardiac conditions such as atrial fibrillation, ischemic or valvular heart disease followed by

Received: September 19, 2016; Revised: October 4, 2016; Accepted: October 17, 2016.

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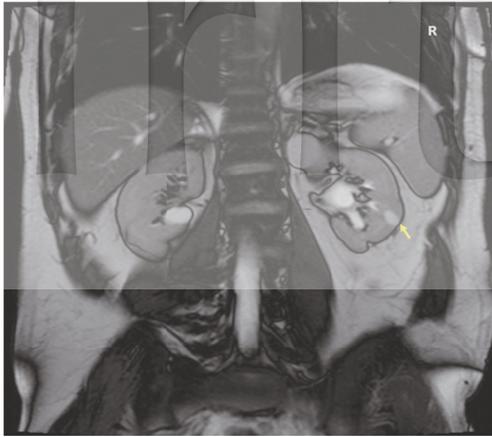


Fig. 1. Magnetic resonance imaging (MRI) with contrast of the abdomen showing the wedge-shaped infarct in the right kidney.



Fig. 2. Magnetic resonance angiography (MRA) with contrast of the abdomen showing normal vascular enhancement of aorta and branch vessels without any structural abnormalities.

other etiologies including hypercoagulable states and renal artery dissection.¹ Interestingly, in a retrospective series with 94 patients with renal infarction, no cause could be identified in 28.7% patients.²

Classic presentation of renal infarction includes persistent abdominal or loin pain with high serum levels of lactate dehydrogenase (LDH) and/or microscopic hematuria, high C-reactive protein (CRP) and high risk of thromboembolic phenomena. However, it can occur without these features and just present with unexplained abdominal pain simulating renal colic. In a case-series, presenting signs and symptoms were comparable in the idiopathic and cardiogenic renal infarction, except that nausea and lumbar tenderness

were more frequent in the idiopathic group.³ Our case emphasizes the fact that high index of suspicion is required to diagnose renal infarction in such patients with the help of appropriate imaging. Helical CT scan without contrast is generally the preferred initial test for suspected renal colic as it is the gold standard for the diagnosis of renal and ureteral stones, which are much more common than renal infarction. If there is no evidence of stone disease on non-contrast CT, a contrast-enhanced CT scan or Magnetic resonance imaging (MRI) should be performed to assess for renal infarction. Pertinent work up to exclude cardiac arrhythmias and vasculitis should be considered. Early recognition is important because it may have long-term implications on kidney health. For example, in a retrospective study of 89 patients with renal infarction, about one-third showed acute kidney injury on initial presentation, and a quarter subsequently developed chronic kidney disease.⁴

Though anticoagulation is indicated when warranted by the underlying disease such as atrial fibrillation, the role of prophylactic anticoagulation in the absence of a hypercoagulable state or an embolic source is less certain. It is prudent to anticoagulate such patients for 3-6 months with low molecular weight heparin or warfarin, in addition to lifelong aspirin. In patients with evidence of occlusion of the main renal artery or a segmental branch by thrombus or embolus, percutaneous endovascular therapy such as thrombolysis or thrombectomy should be considered. Though studies showed successful reperfusion in most patients with these techniques, the improvement in renal function was only modest in those who had acute kidney injury (AKI) at presentation.^{5,6} Angioplasty with or without stent placement is another option in cases where renal infarction is caused by an intrinsic abnormality of renal vessels, such as dissection. Surgical therapy is usually reserved for traumatic cases.

References

1. Eickhoff C, Mei JM, Martinez J, Little D. Idiopathic renal infarction in a previously healthy active duty soldier. *Mil Med.* 2014;179:e259-e262.
2. Bourgault M, Grimbert P, Verret C, et al. Acute renal infarction: a case series. *Clin J Am Soc Nephrol.* 2013;8:392-398.
3. Bolderman R, Oyen R, Verrijcken A, Knockaert D, Vanderschueren S. Idiopathic renal infarction. *Am J Med.*

2006;119:356.e9-12.

4. Yang J, Lee JY, Na YJ, et al. Risk factors and outcomes of acute renal infarction. *Kidney Res Clin Pract.* 2016;35:90-95.
5. Salam TA, Lumsden AB, Martin LG. Local infusion of

fibrinolytic agents for acute renal artery thromboembolism: report of ten cases. *Ann Vasc Surg.* 1993;7:21-26.

6. Blum U, Billmann P, Krause T, et al. Effect of local low-dose thrombolysis on clinical outcome in acute embolic renal artery occlusion. *Radiology* 1993;189:549-554.