

Renal Infarction: The Forgotten Cause of Abdominal Pain

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Abstract- Renal infarction is a rare under-reported condition and is rarely diagnosed at presentation. As it can mimic other conditions, such as nephrolithiasis and pyelonephritis, the diagnosis is often delayed or missed. Unfortunately, this leads to late treatment initiation and therefore may result in irreversible kidney damage. The most common causes are thromboembolism and atheromatous disease.

We present three case reports of renal infarction with similar clinical presentation although distinctive etiologies and evolution. The first one presenting in a context of pulmonary embolism, the second one is reported in a patient with Ehlers-Danlos syndrome, and the third one after surgical valve repair

Index terms- Renal infarction, Ehlers-Danlos syndrome, pulmonary embolism, valve repair

I. INTRODUCTION

Renal infarction is a pathological condition which is caused by renal artery occlusion that may lead to permanent renal damage. It is a rare under-reported condition and is rarely diagnosed at presentation. The nonspecific clinical presentation, frequently like other renal manifestations such as nephrolithiasis and pyelonephritis, is the main factor responsible for delay in diagnosis. Unfortunately, this leads to late treatment initiation and therefore may result in prolonged ischemia and development of irreversible kidney damage. Thromboembolism and atheromatous diseases are the usual causes of renal infarction (1). Nevertheless, other etiologies are described in the literature such as autoimmune diseases and hypercoagulable states. The authors present three cases of renal infarcts presenting initially with acute abdominal pain but with distinct causes, which underlines the importance of medical thoroughness in both patient history and warning clinical signs.

II. CASE REPORT

A. Case 1

A 62-year-old female patient with a known history of myeloproliferative syndrome presenting at the emergency department with severe respiratory distress. The patient had no other comorbidities such as hypertension, diabetes mellitus,

coagulation disorders, renal or heart disease. On physical examination, the patient was in a poor general state, the temperature was 99.1°F, blood pressure was 110/75 mmHg, heart rate was 62 bpm and respiratory rate was 36/min. She presented severe chest pain and dyspnea. CT angiography revealed pulmonary embolism. The patient was immediately admitted for surveillance and received

anticoagulation therapy. Twenty-four hours later, the patient reported acute left flank pain. Blood analysis revealed leukocytosis ($18.41 \times 10^3/\mu\text{L}$ with 81.8% neutrophils), modest elevation of transaminases (aspartate transaminase 134 U/L, alanine transaminase 142 U/L) and frank elevation of lactate dehydrogenase (LDH) - 885 U/L (reference value 135 - 225 U/L). Cytobacteriological testing of urine was normal. Abdominal ultrasound revealed liver no renal nor spleen abnormalities. An abdominal CT was performed revealing a hypodense peripheral triangle-shaped area in the left kidney (**Fig1**). According to Vascular Surgery's opinion, there was no indication for interventional therapy at that time, nor for additional drugs as the patient was already receiving anticoagulation therapy. She was discharged after two weeks with no kidney damage. Presently the patient is on chronic coumarin therapy and under surveillance on Cardiology and Hematology consultation. Biological tests showed renal normal function.



Figure 1: Contrast-enhanced axial CT image demonstrate a wedge-shaped non-enhancing lesion in the left kidney

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B. Case 2

A 40 years old male patient with a known history of a mutation in COL3A1 gene associated with vascular Ehlers–Danlos syndrome, referred to the emergency department for sudden onset of abdominal pain with 16 hours of evolution, located in the upper quadrant and right flank, concomitant with nausea, vomiting and fever. On examination, the patient was agitated, his temperature was 101.3 °F, heart rate was 112 bpm, and blood pressure was 98/71mmHg. Abdominal examination was negative for tenderness or any other abnormality. White blood cell count was elevated at 14 600 cells per cubic millimeter. Urinalysis showed 3 white blood cells per field and no hematuria. Non-contrast abdominal computed tomography showed a triangular hypodense area in the right kidney, initially interpreted as “severe pyelonephritis”. The patient received antibiotics without clinical improvement. In the absence of an evident sign of a urinary tract infection, a CT angiogram was pursued as a renal infarction was highly suspected. It revealed a dissection of the right renal artery with subsequent infarction in the inferior pole of the right renal parenchyma (**Fig 2**). Antibiotic therapy was withheld and the patient received beta-blockers and heparin with symptom resolution after three days of anticoagulation. An etiological study was conducted, including electrocardiogram (ECG) and echocardiography in search for a cardiopathy, as well as a thrombophilia assay. No abnormality was noted. At 3 months follow-up, the patient presented normal renal function and no pertinent symptoms.

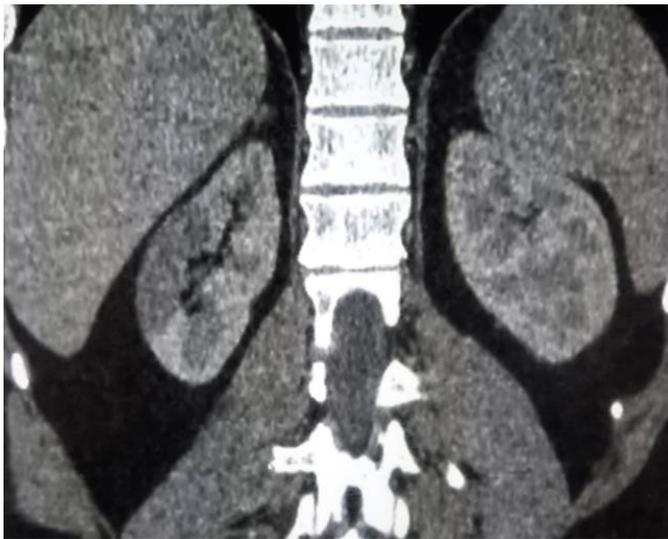


Figure 2: Contrast-enhanced coronal CT image demonstrates a wedge-shaped non-enhancing lesion in the right kidney with no perinephric inflammatory stranding

C. Case 3

A 51-year-old female underwent surgical valve repair for a severe mitral insufficiency (using cardiopulmonary bypass). There were no immediate postoperative complications. Five days following the intervention, the patient presented with acute abdominal pain and oliguria. Her vital signs were unremarkable. Physical examination was significant for tenderness in the right flank and abdominal distention.

There was no clinical sign of a distended bladder. Lab workup demonstrated leukocytosis at 16,200, elevated C-reactive protein at 236mg/l, acute kidney injury (creatinine: 3.1 mg/dl from a baseline of 0.7 mg/dl), and elevation of lactate dehydrogenase (LDH) - 827 U/L (reference value 135 - 225 U/L). Urinalysis was positive for hematuria, whereas urine culture was negative for any infection. Echocardiography estimated left systolic ventricular ejection at 30%. Acute kidney injury was initially considered as a complication of the cardiac surgery. Given the course of evolution, additional paraclinical investigations were requested by the nephrology team. Computed tomography (CT) scan of the abdomen and pelvis without contrast showed paralytic ileus but with no sign of a frank intestinal occlusion. There was no evidence of nephrolithiasis as well.

A renal doppler ultrasound was performed as an abdominal contrast CT was contraindicated. It revealed multiple triangle-shaped hypoechogenic areas in both kidneys with reduced arterial flow (**Fig 3**). The diagnosis of bilateral renal infarction was retained, although according to the vascular surgery team’s opinion, there was no indication for an interventional therapy. Kidney failure continued its aggravation with need for emergency renal replacement therapy. The patient was discharged three weeks after the intervention and continued hemodialysis in a hospital near her domicile. At 6 months follow-up, the patient had irreversible kidney damage with need for chronic hemodialysis treatment.

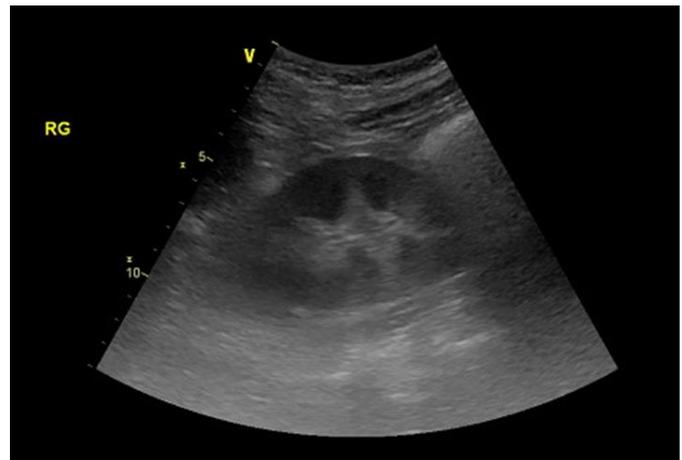


Figure 3: Doppler ultrasound with longitudinal grey-scale image showing renal ischemia as hypoechoic areas of the renal cortex

III. DISCUSSION

In nephrology practice, renal infarction is a very rare entity. This fact may be explained by its nonspecific clinical presentation, as it usually overlaps with more common conditions such as infectious or lithiasic kidney disease (2,3). Lab work often shows leukocytosis, elevated LDH (up to four times higher than normal) and mild elevation of transaminases and C-reactive protein (4,5,6). Microscopic hematuria is frequently present, although gross hematuria is rare (7). On the other hand, acute kidney injury is a common feature at presentation or in the days following diagnosis, particularly in the case of bilateral infarction (8,9). Because of all these unspecific features, it is preferable to always suspect renal infarction in the presence of acute flank pain and increased LDH

levels, especially of the patient has thromboembolic risk factors. Renal angiography is considered the gold standard exam, although the use of non-enhanced computed tomography (CT) is more common in the emergency room.

However, contrast-enhanced CT is of great importance as it contributes to the establishment of the diagnosis. The CT study may show different presentations of renal infarction: the common finding is a wedge-shaped perfusion defect, but it can also present as multifocal or global renal ischemia (10). The presence of a cortical rim sign can be noted, which is an enhanced thin layer of renal parenchyma at the periphery, due to preserved capsular circulation (7,10).

Risk factors for renal infarction involve atrial fibrillation, previous, pulmonary embolism, and heart disease, whether it is ischemic or valvular (11). Therefore, etiological investigation is a vital step in the evaluation of renal infarction and must include the following exams: ECG, echocardiography, CT angiography and evaluation for prothrombotic and autoantibodies assay (12).

Because there is a lack of comparative studies, the optimal treatment for renal infarction is undetermined. Nevertheless, based on the consensus of the literature, anticoagulation with intravenous heparin followed by oral warfarin is the standard therapy protocol in this situation.

The prognosis of renal infarction usually depends more on the underlying comorbidities than on the renal infarction itself. Its most common repercussions include persistent hypertension and renal failure. Some patients will even need to be placed on a dialysis program, such as the case 3 (7, 13).

IV. CONCLUSION

In summary, early diagnosis is of vital importance to prevent permanent loss of renal function. Clinical suspicion of renal infarction should be raised in the presence of sudden onset of abdominal pain and high lactate dehydrogenase in a patient with risk factors. Early anticoagulation is paramount to a rapid recovery.

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