CLINICAL CASE OF THE MONTH

A 56-Year-Old Man with Sudden Onset Abdominal Pain

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Acute abdominal pain is one of the most common presentations encountered in the emergency department (ED). The differential diagnosis of acute abdominal pain is extensive and identifying the underlying etiology can be challenging. Spontaneous renal artery thrombosis is a rare cause of acute abdominal pain. We review a case of acute presentation of renal artery thrombosis in a patient without risk factors for thromboembolism, and highlight the importance of considering this rare cause of abdominal pain.

CASE REPORT

A 56-year-old man with no significant past medical history presented to the emergency department (ED) with sudden onset of abdominal pain. The pain started one hour prior to presentation; was located in the epigastrium and across the anterior abdomen, was sharp and constant with no radiation. He denied any associated symptoms or history of trauma. He was not taking any medications and had no known drug allergies. He denied use of tobacco or illicit drugs and reported less than three alcoholic drinks per week. His family history was significant for hypertension.

Upon arrival, his vital signs revealed a temperature of 98.3° F, pulse of 66 beats per minute, blood pressure of 125/85 mmHg, respiratory rate of 22 breaths per minute, and oxygen saturation of 98% on room air. His physical examination was unremarkable except for the right lower abdominal quadrant tenderness.

An electrocardiogram revealed normal sinus rhythm with no ST or T wave abnormalities. Complete blood count (CBC), kidney function tests, liver function tests, cardiac enzymes, urinalysis, lipase and amylase, and urine toxicology screen, were all within normal limits. A lactate dehydrogenase (LDH) level was mildly elevated at 225 units per liter (Normal range 80 - 180 units per liter). Abdominal radiographs (plain films) revealed no...
abnormalities. Due to lack of clarity about the etiology of his symptoms, computed tomography angiogram (CTA) with IV contrast of the abdomen was performed and revealed a thrombus in the anterior right renal artery branch with associated areas of ischemia/infarct in the right mid and lower kidney (Figure 1).

It was unclear whether the renal artery occlusion was due to thromboembolism phenomenon or thrombosis in situ. Additional testing was conducted to determine a possible underlying cause. These tests included a hypercoagulability workup including prothrombin time, partial thromboplastin time, antiphospholipid antibodies, protein C and S activity, antithrombin III activity, homocysteine level, and prothrombin F2 G20210A mutation. All test results were within normal limits. Transthoracic echocardiography and subsequently transesophageal echocardiography (Figure 2) revealed no evidence of right-to-left intracardiac shunts, intracardiac thrombus or cardiac tumor(s). His aorta was smooth without any suggestion of an atheroembolic source.

Anticoagulation with unfractionated heparin infusion was initiated. Invasive angiography confirmed occlusion of a branch of the right renal artery (Figure 3). Despite multiple attempts at revascularization, the occlusion could not be successfully crossed. The patient was managed medically with anticoagulation, pain control and hydration.

His abdominal pain resolved and kidney function remained normal. The unfractionated heparin infusion was switched to rivaroxaban and he was discharged home in a stable condition. Post discharge, a 30-day event monitor showed sinus rhythm with no atrial or ventricular arrhythmias, and anticoagulation was continued for six months.

**DISCUSSION**

Acute renal infarction (ARI) due to renal artery occlusion is rare. Its presenting symptoms of abdominal pain, flank pain, nausea and/or vomiting are nonspecific. Suspicion for this diagnosis is warranted in patients with a high risk of thromboembolism. In their analysis of medical records of 17 patients with established diagnosis of ARI, Domanovits et al. reported that pain was the main presenting symptom, involving the flank, abdomen and lower back in decreasing frequency. Eighty-two percent of these patients had at least one risk factor for thromboembolism including atrial fibrillation, hypertension, or history of embolism, ischemic heart disease and mitral valve disease.

Acute renal infarction usually occurs after a thromboembolic event from sources such as a left atrial thrombus in atrial fibrillation or atheromatous plaque in the aorta. In spite of the fact that thromboembolism is the main cause of ARI, renal arteries are considered an unusual target. In their retrospective study evaluating patients with peripheral arterial embolic thromboembolism, Frost et al. reported that the renal artery was the least common site (2%) compared to extremities (61%), mesenteric (29%), pelvic arteries (9%), and aorta (7%).

ARI can also be attributed to less common causes such as trauma, idiopathic dissection of the renal artery, paradoxical embolism, or underlying hypercoagulable states such as antiphospholipid antibody syndrome.

The diagnosis of ARI is challenging not only due to the vague
clinical symptoms at presentation, but also the non-specific laboratory findings noted in these patients. Urinalysis reveals hematuria and proteinuria in about half of patients with ARI, and kidney function is usually only slightly impaired. LDH, a marker for tissue damage, is classically elevated. An imaging modality is necessary to establish the diagnosis of ARI in most cases. In one study, the renal isotope scan was the most sensitive test for detection (97%), followed by contrast-enhanced computed tomography scan (80%) and ultrasound (11%). Angiography is the gold standard with a sensitivity of 100%. Once the diagnosis of ARI is established, a work-up to identify underlying predispositions to hypercoagulability and arterial thrombosis or embolus is indicated.

Management of patients with ARI usually focuses on restoring patency to the affected artery and treating the underlying cause. Surgical intervention is sometimes needed for highly symptomatic patients, especially those with renal artery dissection after trauma. In a majority of patients, anticoagulation with unfractionated heparin or low molecular heparin followed by oral anticoagulation with warfarin is the mainstay of management. The role of newer anticoagulation agents in such patients remains unclear but was chosen for our patient based on extrapolated data from the treatment of other thromboembolic events such as pulmonary embolus. Reperfusion therapy (thrombolysis or thrombectomy) has also been reported as a successful modality of treatment, but is usually only effective when pursued early in the course of the ischemic insult. Determining the ideal duration of anticoagulation, especially in patients without an identified underlying cause is quite challenging, since there are no recommendations to guide in this regard. In our patient, we chose to treat for six months, mirroring the recommendations for treatment of pulmonary emboli without hemodynamic compromise.

CONCLUSION

Spontaneous renal artery thrombosis without any obvious underlying cause in an otherwise healthy patient is rare. Therefore, a high level of suspicion especially in patients with risk factors is needed to establish a prompt diagnosis and improved outcome.

REFERENCES


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