Renal infarction mimicking cholecystitis: a case report of a patient presenting with stroke

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Abstract
Objective: Renal infarction which is a rare phenomenon can be missed on the patient’s workup making its incidence low.

Case Presentation: We report a 68-year-old lady with known history of hypertension presented with epigastric pain and vomiting for 4 days. During her stay in the emergency room, she developed sudden onset of left-sided weakness. Electrocardiogram was normal sinus rhythm. Computed tomography scan of the abdomen showed acute pancreatitis with partial impingement of superior mesenteric vein.

Conclusion: Imaging showed cholelithiasis without evidence of cholecystitis and lower pole right renal infarct likely secondary to obstruction of the right lower renal artery due to calcified plaques at their origin from the aorta. Subsequently, the patient was kept on antiplatelets and planned for discharge uneventfully.

Keywords: Renal infarction, Cholecystitis, Abdominal pain

Introduction
Our case was challenging as she was presented with acute abdominal pain and sudden onset of stroke. Electrocardiogram was unremarkable. Percutaneous angioplasty with stenting showed promise in the presence of renal artery dissection. It has to be suspected and managed appropriately hence this case will help emergency and internal medicine physicians to be aware of this rare possibility.

Case Presentation
A 68-year-old lady with a prior history of hypertension was presented with epigastric pain and vomiting for 4 days. Epigastric pain was dull aching, radiated to the right upper quadrant, 6/10 on the pain scale, continuous with no clear aggravation with food intake. The patient had associated nausea with two episodes of vomiting which were non-bilious, non-projectile and contained food particles. The patient did not complain of altered bowel habits. There was no associated fever, cough, chest pain or urinary complaints. The patient was brought to the emergency room due to non-settling nature of pain. Her medication list included calcium channel blockers twice daily. She had no known allergies or addictions. On arrival, the patient vital signs were: blood pressure 200/110 mm Hg, Heart rate 94 bpm, respiratory rate 23/minutes, oxygen saturation of 96% on room air and temperature 37°C. General physical examination was unremarkable. Chest examination showed bilateral equal breath sounds and was clear to auscultation. The abdomen was soft on palpation with tenderness in the right upper quadrant; however, murphy sign was negative and rebound tenderness was negative with negative costovertebral tenderness.

During the emergency room stay, the patient developed left-sided weakness which was of sudden onset. Neurological examination revealed left-sided upper motor neuron facial weakness and power was 2/5 on the left upper and lower limb proximally and distally. The left planter was up-going. Power on the right side was 5/5 with down-going planters.

ECG revealed normal sinus rhythm with no ischemic changes. Troponins were negative. Initial lab investigation revealed total leucocyte count 33 cells per cubic millimeter, C-reactive protein 194 mg/L, creatinine 1.6 mg/L and lipase 71 U/L. Ultrasound abdomen revealed cholelithiasis; however, there was no evidence of cholecystitis. Small right renal cyst with no significant abnormality was identified in the left kidney. Magnetic resonance imaging brain revealed acute right strio-capsular infarct.

Management of the patient included glyceryl trinitrate...
infusion to lower the blood pressure, aspirin 75 mg and atorvastatin 40 mg. Neurological consultation was taken. Analgesia was administered for abdominal pain and general surgery input was taken. The patient was kept nil per oral. Blood pressures were gradually controlled due to the recent nature of the ischemic stroke.

With the background of unresolving abdominal pain, computed tomography (CT) abdomen with contrast was planned after acute kidney injury was resolved with gentle hydration. Serum creatinine went down to 0.7 mg/L. CT abdomen revealed findings suggestive of acute pancreatitis with partial impingement of superior mesenteric vein. Cholelithiasis without evidence of cholecystitis and lower pole right renal infarct were noted due to obstruction of the right lower renal artery owing to calcified plaques at their origin from the aorta.

On stroke workup, an electrocardiogram was performed and showed an ejection fraction of 60% with normal cardiac chambers, mild left ventricular hypertrophy, normal left ventricular systolic function and grade I left ventricular diastolic dysfunction. Ultrasound carotid doppler showed mild diffuse atheromatous changes seen in both common carotid arteries. Thrombus plaque in the left bifurcation was seen causing focal morphological lumen narrowing. Proximal left internal carotid artery was measured about 47.1%. No other high-grade stenosis was identified either side. The patient was discharged to home on antiplatelets and antihypertensive medications with marked clinical improvement.

Discussion
Renal infarction is a rare cause of abdominal pain and often a missed diagnosis. Delay in diagnosis and lack of the physician and radiologist awareness could be a contributing factor for missed cases. It is most commonly due to thromboembolic phenomena with emboli coming from the heart or the aorta. Most common causes are atrial fibrillation, infective endocarditis, thrombi from the aorta, renal artery dissection, hypercoagulation, prior endovascular intervention, cocaine use, sickle cell disease, trauma or idiopathic (1). Upper pole predilection for segmental renal infarction is noted in the literature (1).

Patients with unilateral flank pain with increased risk of thromboembolism are at high risk of developing this condition. Hematuria, leukocytosis and elevated lactate dehydrogenase are supportive of the diagnosis. An observational study in the emergency room aiming to review the CT scan of abdomen showed an incidence of 0.007% (1). As CT renal is commonly performed for the acute flank pain evaluation, it is important to add contrast for its timely diagnosis.

The challenging aspect of this case was its misleading clinical manifestation. It was thought initially that the patient might be suffering from cholecystitis. Most common associated symptoms were nausea (63%), vomiting (33%), fever (41%), urinary symptoms (15%), flank pain (63%) and abdominal pain (74%) (1,2).

Renal infarction is renin mediated hence acute elevation of blood pressure was noted in our case. Lab investigations which led us to suspect renal infarction included elevated serum lactate dehydrogenase, C-reactive protein, leukocytosis, microscopic hematuria, proteinuria, elevated serum creatinine, and creatinine kinase (2,3).

Exploring the root cause analysis also included electrocardiogram, transthoracic echocardiography, Holter monitoring, thrombophilia panel, homocysteinemia measurement, and magnetic resonance abdominal angiography. CT angiography with renal angiography is the standard modality to establish diagnosis showing a wedge-shaped region of reduced density (3,4). Global or segmental renal infarction is demonstrated by the absence of blood flow in the Doppler evaluation. Conventional ultrasound and CT scan could also miss the diagnosis which is worth noting (3-5).

Literature supports the use of intra-arterial thrombolytic therapies and thrombectomy with successful reperfusion. Angioplasty with stent placement is used for patients with intrinsic renal vessels abnormality such as renal artery dissection (4,5).

Conclusion
The present investigation shed light on the rare causes of abdominal pain to create awareness for the emergency physicians to consider it in their differential diagnosis. It is a serious pathology and requires great deal of suspicion to avoid missed diagnosis. The gold standard method of diagnosis is renal angiography and requires anticoagulation.

Authors’ contributions
SS and UJ designed, performed the literature search, and wrote the manuscript. All authors provided feedback and reviewed the entire article.

Ethical issues
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for the clinical information to be reported in the journal. The patient understand that his name and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

References
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