

## Acute Abdomen as an Unusual Presentation of Myocardial Infarction

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### Abstract

A 29 years old male patient, chronic smoker with no history of diabetes, hypertension, ischemic heart disease or renal dysfunction was admitted in our hospital with complaints of acute onset pain in the right iliac fossa radiating to the umbilical region associated with multiple episodes of vomiting without hematemesis. Patient was clinically diagnosed to have acute appendicitis and treated for the same.

Routine electrocardiogram done in the surgical intensive care unit suggested an acute anterior wall ST segment elevation myocardial infarction for which the patient was thrombolysed. Patients symptoms immediately subsided. Coronary and renal angiogram showed a recanalized left anterior descending artery with right renal artery thrombus. The acute renal infarction was responsible for his acute abdomen. A transient left ventricular clot produced by the stunned myocardium was held responsible for the above events.

### Introduction

Acute renal infarction is an uncommon and underdiagnosed disease [1]. Its clinical presentation is nonspecific and often mimics other more common disease entities. The diagnosis is usually missed or delayed, which frequently results in irreversible renal parenchyma damage. High index of suspicion is required for early diagnosis, as timely intervention may prevent loss of kidney function [1-4]. We report a case of acute renal infarction following an acute myocardial infarction in a patient who presented to us with acute abdominal pain mimicking appendicitis.

### Case

A 29 years old male patient, chronic smoker with no history of diabetes, hypertension, ischemic heart disease or renal dysfunction was admitted in our hospital with complaints of acute onset pain in the right iliac fossa radiating to the umbilical region associated with multiple episodes of vomiting without hematemesis since around 7 hrs duration. On examination patient had a heart rate of 100 beats/min with a blood pressure of 100/70 mm/hg with profuse sweating. Abdominal examination revealed tenderness in the right iliac fossa with some rebound tenderness.

Patient was thought to be suffering from either

1. Appendicitis
2. Ureteric colic
3. Enteritis

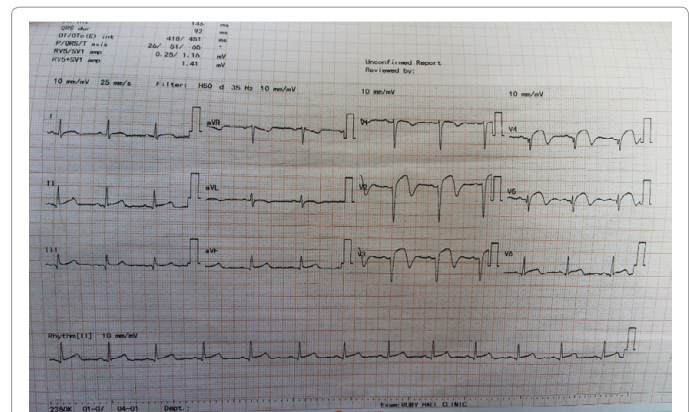
Patient was immediately admitted in surgical ICU as a case of acute abdomen and managed accordingly. Laboratory investigations done were as follows:

- Hemogram revealed hemoglobin of 18 g/dl with a hematocrit of 54.2% with leukocytosis WBC-21000 with neutrophilic dominance.
- Serum sodium-116 mmol/l, serum osmolality 240 mosmol/kg-patient was conscious oriented without any signs of hyponatremia.
- Liver function tests were normal with LDH levels of 3139 u/l.
- Lipase, amylase was normal with a serum creatinine of 1.7 mg/dl.
- Urine routine showed mild amounts of proteinuria with no hematuria.

- Abdominal and chest x-rays were normal. Ultrasonography of abdomen and pelvis was normal.

- A routine 12 lead Electrocardiogram was done which showed frank anterior wall ST segment elevation myocardial infarction (Figure 1).

Immediately a cardiologist was involved. Cardiac colour Doppler was done which showed distal septal and apical hypokinesia with an ejection fraction of 40% without any left ventricular clot or any other mechanical complications of myocardial infarction. Abdominal pain, raised LDH and proteinuria prompted us to think about the possibility of a right renal artery infarction.



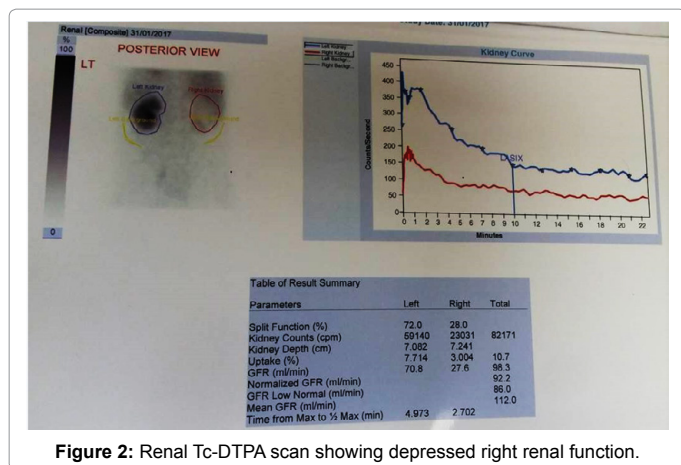
**Figure 1:** 12 Lead electrocardiogram showing acute anterior wall ST segment elevation myocardial infarction.

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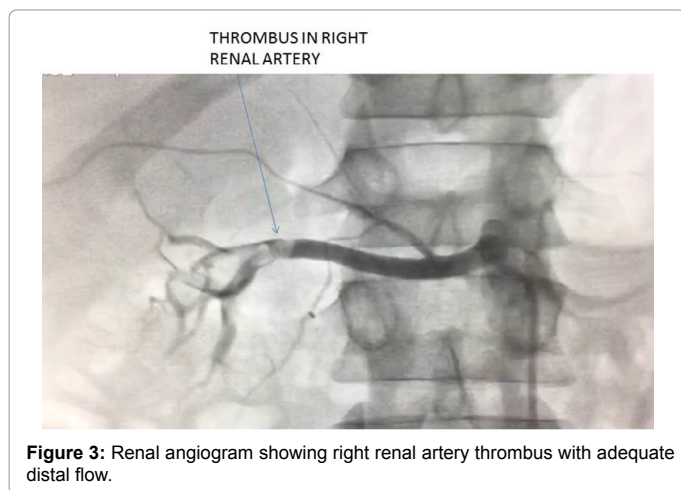
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**Figure 2:** Renal Tc-DTPA scan showing depressed right renal function.



**Figure 3:** Renal angiogram showing right renal artery thrombus with adequate distal flow.

So, a decision for thrombolysis was taken. Patient was loaded with 4 tablets of 75 mg Clopidogrel and 325 mg of Aspirin with 80 mg Atorvastatin. Patient was thrombolysed with Streptokinase with the time since symptom onset to thrombolysis 10 hrs. Relatives were not willing for a primary angioplasty. Post-thrombolysis abdominal pain and electrocardiogram changes subsided completely. Patient was put on dual anti-platelets, statin, beta blocker, diuretics, low molecular weight heparin and an ACE inhibitor. 4-5 hrs after thrombolysis patient developed altered sensorium. Neurological examination showed altered sensorium with no other focal neurological deficit.

Magnetic resonance imaging of brain was done which showed multiple ischemic lacunar infarcts with no bleed.

The stroke was managed conservatively after neurology consultation. Patient improved significantly in the next 24 hours to near normal sensorium. After the patient was stabilized neurologically, coronary angiography was done which showed a recanalized left anterior descending artery and renal angiography showed right renal artery thrombus with adequate distal flow. Serum Homocysteine levels were found to be 90 umol/l with serum vitamin B12 levels of 150 pg/ml. Warfarin was added to the treatment and it was decided to give triple therapy (dual anti-platelets with Warfarin) for six months followed by Clopidogrel and aspirin for 6 months and then Aspirin to be continued lifelong. A pre-discharge renal Tc-DTPA scan was done which showed poorly functioning right kidney with moderate parenchymal dysfunction. Patient was also given intravenous vitamin

B12 supplements and then put on oral vitamins on discharge (Figures 2 and 3).

## Discussion

Acute renal infarction is a rare and under diagnosed clinical condition. In one study, renal infarction was found in 205 of 14,411 autopsies, but clinical diagnosis was made in only two patients during life [5]. More than 95% of patients have a history of one or more risk factors for thromboembolism. Atrial fibrillation, valvular heart disease, ischemic heart disease, inherited or acquired coagulopathies, and a previous history of thromboembolic events are the major predisposing factors [1-4,6] Less commonly, idiopathic cases of acute renal infarction in patients with no preexisting disorders have also been reported [1,2].

In a review of forty-four cases of acute renal embolism, Hazanov et al. [6] found that 68% of patients presented with generalized abdominal pain, 32% had lumbar pain, and 7% had right upper quadrant pain. Nausea and vomiting occurred in 43% of patients and 41% of patients had fever. Domanovits et al. [2] reported that 94% of patients with acute renal infarction had elevated serum lactate dehydrogenase upon admission, and all patients showed an elevated serum lactate dehydrogenase after 24 hours. Hematuria was found in 80% of patients, and 67% of patients had proteinuria.

Leucocytosis can also be found in majority of the patients [1-3]. Mildly elevated serum creatinine is common, but severe oliguric or anuric renal failure occurs only in patients with bilateral disease or infarction of a solitary kidney [3,6].

Huang et al. [1] found that 80% of patients with acute renal infarction present with a triad of flank or abdominal pain, elevated serum lactate dehydrogenase, and proteinuria. These features, however, are nonspecific and not diagnostic.

Contrast-enhanced CT is a non-invasive widely available modality that can establish the diagnosis of renal infarction in suspected cases [1,2,6]. In our case since the patient also had an acute anterior wall myocardial infarction we could not waste more time in doing a computerized tomography of the abdomen as the patient was well within the window period of 12 hours for thrombolysis.

Due to its rarity, optimal therapy for renal infarction has not been established. Medical management with local low-dose, intra-arterial thrombolysis or systemic anticoagulation is generally preferred over surgical embolectomy, which is usually reserved for bilateral disease or involvement of a solitary kidney [2,4,6,7]. However, recovery of renal function only usually occurs if these interventions are employed within 90-180 minutes of symptom onset, which represents the ischemic tolerance of normal kidney [4]. Thus, prompt diagnosis is imperative if irreversible renal damage is to be avoided. However, in patients with a history of long-standing renovascular disease, viability of renal parenchyma can sometimes be maintained after the complete occlusion of renal artery through well-developed collateral circulation, although perfusion might be inadequate to produce urine. Restoration of renal blood flow in such selected cases may lead to improvement in renal function even after prolonged occlusion [8]. In our patient, the diagnosis of renal artery embolism was made after more than twenty-four hours after the onset of symptoms, and so the right renal function remained relatively depressed.

So, finally the conclusion was that the patient had an acute anterior wall ST segment elevation myocardial infarction whose symptoms were maybe masked by the acute renal infarction that could have been caused by a transient left ventricular clot produced by the stunned myocardium which also led to multiple cerebral lacunar infarcts.

## Conclusion

Acute renal infarction can present with abdominal pain mimicking appendicitis. High clinical suspicion is needed for prompt diagnosis. Urgent radiological confirmation using contrast enhanced CT should always be considered in patients presenting with atypical flank or abdominal pain, especially in patients with an increased risk for thromboembolism. The presence of haematuria, proteinuria, leucocytosis, and an elevated serum lactate dehydrogenase level further support the diagnosis.

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