

Epigastric Pain in Acute Renal Infarction: the Forgotten Presentation

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ABSTRACT

Epigastric pain is a manifestation of several medical and surgical conditions. However, when persistent epigastric pain is associated with microscopic or frank haematuria and elevated lactate dehydrogenase (LDH), especially in patients with increased risk of thromboembolic events, acute renal infarction (ARI) should be considered. We report the case of a 77-year-old male patient who presented with sudden persistent epigastric pain and elevated LDH who was found to have atrial fibrillation. The patient was diagnosed with ARI. ARI is not usually a typical differential diagnosis in patients with persistent epigastric pain and elevated LDH in whom the risk of thromboembolic events is high. Thus, physicians should perform a contrast-enhanced CT scan as early as possible to rule out or confirm renal infarction.

LEARNING POINTS

- In acute kidney infarction, epigastric pain can be one of the clinical manifestations.
- The combination of unexplained epigastric pain and elevated lactate dehydrogenase and haematuria in a patient with an increased risk of thromboembolic events should raise the suspicion of acute kidney infarction.
- The physician should perform a contrast-enhanced CT scan as early as possible to rule out or confirm kidney infarction.

KEYWORDS

Epigastric pain, atrial fibrillation, lactate dehydrogenase

INTRODUCTION

The clinical manifestations of acute renal infarction (ARI) usually mimic those of other, more common, entities. As the symptoms and laboratory findings are nonspecific, the diagnosis of ARI requires a high degree of clinical suspicion and a low threshold for performing early contrast-enhanced CT scans (Fig. 1), especially in patients with an increased risk of thromboembolic events.

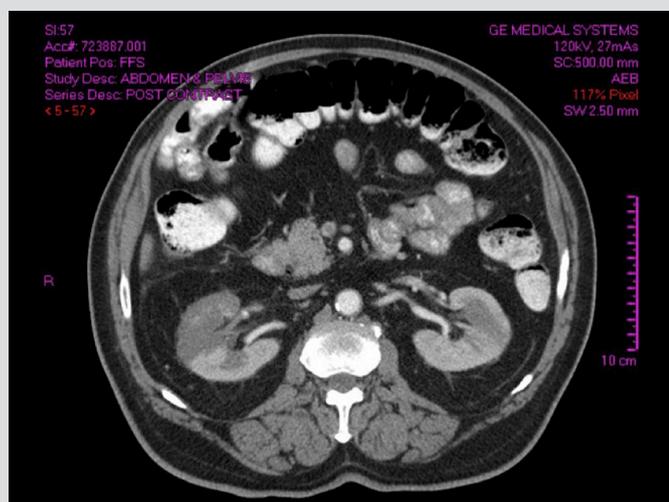


Figure 1: Contrast-enhanced computed tomography of the abdomen and pelvis showing right renal infarction.

CASE HISTORY

A 77-year-old man presented with sharp epigastric pain that had started suddenly a few hours prior and moderate vomiting. The patient had a significant medical history for diabetes mellitus type II, hypertension and prostate enlargement. He never smoked or abused alcohol. The patient's blood pressure was 160/90 mmHg and had an irregular pulse of 95 beats per minute, respiration rate of 20 breaths per minute and a temperature of 37.8°C. Physical examination revealed an irregular heartbeat and tenderness in an epigastric area. The rest of the exam was unremarkable.

Laboratory tests revealed a haemoglobin value of 15 g/dl, leukocyte count of 13,000/mm³, neutrophils 80.5%, platelets 260,000/mm³, serum creatinine 105 µmol/l (normal 80–100 µmol/l), lactate dehydrogenase (LDH) 833 units/l (normal 90–180; this increased to 1700 units/h on the next day), aspartate aminotransferase 168 units/l (normal 10–42) and alanine aminotransferase 100 units/l (10–40). Urinalysis was positive for blood, and urine protein was 250 mg/dl. Three sets of cardiac enzymes were normal.

The patient's electrocardiography showed atrial fibrillation. Echocardiogram revealed a dilated left atrium, normal ejection fraction and no evidence of a thrombus or vegetation. The abdominal ultrasound showed no evidence of gallbladder disease.

Contrast-enhanced computed tomography (CECT) of the abdomen and pelvis showed a right renal infarction.

The patient was anti-coagulated with heparin and warfarin. His symptoms were significantly improved, and he was discharged.

DISCUSSION

ARI can be a serious medical emergency. The exact incidence of ARI is not known because the diagnosis is usually missed. In one study, the incidence of kidney infarction in patients with atrial fibrillation was approximately 2%^[1].

The most common source of renal emboli is a diseased heart^[2]. Atrial enlargement secondary to valvular heart disease or a dyskinetic left ventricle after myocardial infarction can provide the source of mural thrombi that may dislodge and become renal artery emboli^[2].

Hazanov et al. reported 44 cases of renal infarction secondary to atrial fibrillation^[3]. In a study containing 27 patients diagnosed with ARI, cardiac disease was found in 11 patients (41%), 10 of whom had documented atrial fibrillation. In the other 16 patients, the aetiology of ARI was considered to be idiopathic. Different thrombophilic abnormalities were found between the patients in the idiopathic group.

Hyperhomocysteinaemia was detected in five patients; one had protein S deficiency, one had a factor V Leiden mutation and one had antithrombin deficiency^[4].

The clinical presentations of ARI are non-specific and may be confused with other entities. ARI patients commonly present to an emergency department with flank pain associated with fever, nausea, vomiting and/or acute onset hypertension^[5].

An abrupt onset of abdominal or flank pain, along with nausea and vomiting, was found in approximately 50% of patients in a study conducted by Chu and colleagues. In the same study, 30% of patients reported fever. Elevated white blood cell count and LDH were noticed in a blood analysis of the patient with ARI. Urinary analysis revealed haematuria and proteinuria^[6].

Imaging is the key to diagnosing renal infarction. The diagnosis can be confirmed by CECT, angiography or isotope scan. The sensitivity of CECT is 80%, that of renal isotope is 97% and that of angiography is 100%^[3]. The absence of contrast in the affected renal tissue is best demonstrated by CT^[2]. Smaller infarcts are observed as wedge-shaped, low-density areas within an otherwise normal appearing kidney (partial kidney infarction). Complete kidney infarction is identified by a non-enhanced kidney with a cortical rim sign, i.e., the outer 2–4 mm are enhanced due to perfusion by the capsular branches^[2].

The differential diagnosis of ARI includes renal colic, pyelonephritis, biliary colic, acute cholecystitis and other thromboembolic events, such as mesenteric ischaemia and splenic infarction^[7].

Optimal treatment modalities for ARF have not been evaluated in large prospective studies because of the rarity of this condition^[8]. In 1973, a comparison between surgical and medical management for unilateral renal embolism showed favour towards conservative therapy.

Anticoagulation with or without thrombolysis is considered to be the primary treatment strategy for ARI^[10].

Timing is important when considering thrombolytic therapy as a treatment option for ARI. ARI should be diagnosed within the first 4 h after symptom onset to ensure the feasibility of using thrombolysis with intra-arterial urokinase^[11].

Ideally, ARI should be diagnosed within the first 90 min of symptom onset^[12], a situation that rarely occurs^[13,14]. Many studies have reported favourable results using anticoagulants (unfractionated heparin, low-molecular-weight heparin and warfarin) in the treatment of ARI^[3,13].

For patients with total parenchymal involvement due to bilateral emboli or for those with a unilateral main renal artery embolus in a solitary kidney, surgical embolectomy remains the only treatment option, although it is rarely performed^[13,14].

In our case, the patient presented with unexplained persistent epigastric pain, which could have been due to a number of reasons. However, the combination of elevated LDH, which doubled the next day, mild elevation of aminotransferase, blood in the urine and the presence of atrial fibrillation as a marker of increased risk of thromboembolic events raised the suspicion of a thromboembolic manifestation, which was confirmed by a CECT scan.

CONCLUSION

Persistent epigastric pain can be a manifestation of ARI. In combination with elevated LDH, especially in patients with an increased risk of thromboembolic events, a CECT scan should be performed as early as possible to rule out or confirm renal infarction.

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