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CASE REPORT

Acute Renal Infarction: An Unusual Cause of Abdominal Pain

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Acute renal infarction is an uncommon and underdiagnosed disease. 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. We report a case of acute renal infarction following coronary angiography in a patient with paroxysmal atrial fibrillation who initially presented with acute abdominal pain mimicking appendicitis.

Keywords acute renal infarction, atrial fibrillation, renal artery occlusion, thromboembolism

INTRODUCTION

Acute renal infarction is an uncommon disease with an estimated incidence of 0.004–0.007% in patients visiting emergency departments.[1,2] Its clinical features are non-specific and often mimic other more commonly encountered diseases, such as nephrolithiasis, cholecystitis, appendicitis, pyelonephritis, acute mesenteric infarction, and even myocardial infarction.[3,4] Thus, the diagnosis is often missed[2,5] or delayed, which frequently results in irreversible renal parenchymal damage. A high index of suspicion is required for early diagnosis, as timely intervention may prevent loss of kidney function.[4]

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CASE REPORT

A 78-year-old female presented to the emergency department with a history of sudden onset right iliac fossa pain lasting twelve hours. The pain radiated toward the umbilical region and was associated with nausea and vomiting. She had a past medical history of paroxysmal atrial fibrillation and an anteroseptal myocardial infarction requiring thrombolysis five weeks earlier. Two days before the presentation, she had a coronary angiogram via the right femoral artery to investigate recurrent angina. Anticoagulation with warfarin was withheld temporarily, from three days before to the day after the procedure.

Upon examination, the patient was in atrial fibrillation with a heart rate of 84 beats per minute, blood pressure 125/90 mm Hg, and temperature 37°C. She was tender in the right iliac fossa with no guarding or rebound tenderness.

Initial investigations showed hemoglobin of 12.8 g/dL, white cell count (WCC) 12.5×10^9/L, C-reactive protein (CRP) 26 mg/L, and serum creatinine 86 μmol/L (pre-admission creatinine 60 μmol/L). International normalized ratio (INR) was sub-therapeutic at 1.3. Urine dipstick examination showed 1 + blood and 1 + protein and was negative for nitrites, leucocytes, and ketones. Abdominal and chest x-rays were normal.

She was preliminarily diagnosed with acute appendicitis. However, in view of some atypical features and the recent coronary angiogram, a contrast-enhanced CT abdomen was requested. This revealed a thrombus in the right renal artery with an associated low-density area in the adjacent kidney consistent with renal infarction. Blood supply from an accessory vessel spared the upper pole of the right kidney (see Figure 1)

The patient was treated with analgesia and low molecular weight heparin (dalteparin 200 units/kg of body weight, once a day), and warfarin was continued. She worsened slightly the next day, developing a temperature of 38°C. Her WCC increased to 17.3×10^9/L, creatinine rose to 121μmol/L, and CRP increased to 219 mg/L.

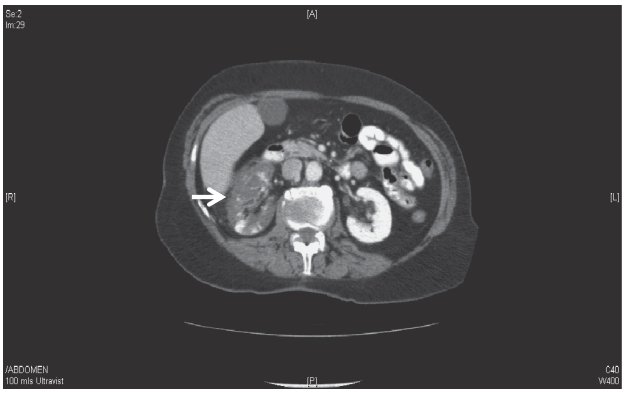

Figure 1. Contrast enhanced CT scan showing right kidney infarction.

Twenty-four hours later, her temperature settled, serum creatinine improved and subsequently settled at 85μmol/L, and WCC gradually came down to 7.8×10^9/L. At the time of discharge, CRP had fallen to 36 mg/L. Dalteparin was discontinued once INR became therapeutic between 2.0–3.0. On follow-up, six months after the initial event, she was stable with no long-term sequelae. Her creatinine remained mildly elevated but stable at 90 μmol/L from her baseline of 60 μmol/L pre-injury, and she did not have any further thromboembolic events.

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]

A brief literature review for common etiological factors, presenting features, and laboratory findings of acute renal infarction is summarized in Table 1. 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]

It typically presents with one or a combination of acute abdominal, flank, or back pain.[1–3] 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 and colleagues[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

Table 1
Predisposing factors, presenting features, and common laboratory abnormalities: a review of literature

Etiology	Richard et al., 1978 (N=17)	Blume et al., 1993 (N=14)	Domanovits, 1999 (N=17)	Hazanov et al., 2004 (N= 44)	Haung et al., 2006 (N = 20)
Atrial fibrillation	10 (59%)	13 (92%)	11 (65%)	44 (100%)	10 (50%)
Valvular heart disease	5 (29%)	5 (36%)	6 (35%)		4 (20%)
Ischemic heart disease	5 (29%)		7 (41%)		
Previous thromboembolism			6 (35%)	7 (16%)	5 (25%)
Coagulation dysfunction					2 (10%)
Idiopathic	1 (6%)	1 (7%)	3 (18%)		2 (10%)
Spontaneous renal artery dissection					1 (5%)
Presenting symptoms					
Abdominal pain	7 (41%)	4 (29%)	11 (65%)	30 (68%)	17 (85%)
Flank pain	7 (41%)	12 (86%)	6 (35%)	14 (32%)	17 (85%)
Back pain	2 (11%)		5 (29%)		2 (10%)
Nausea/vomiting	8 (47%)	12 (86%)	7 (41%)	19 (43%)	3 (15%)
Fever	10 (59%)			18 (41%)	1 (5%)
Oliguria/anuria	6 (35%)	7 (50%)		3 (7%)	
Laboratory findings					
Proteinuria	8 (47)	14 (100%)	10/15 (67%)	17 (45%)	16 (80%)
Hematuria	15 (88%)	14 (100%)	12/15 (80%)	21 (54%)	9 (45%)
Leukocytosis	17 (100%)	14 (100%)	14 (82%)	36 (82%)	20 (100%)
Elevated lactate dehydrogenase	17 (100%)	14 (100%)	17 (100%)	41 (93%)	19 (95%)

patients had proteinurea. 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 non-specific 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]

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 about twenty-four hours after the onset of symptoms, and the renal function remained relatively preserved. Thrombolysis or surgical embolectomy was therefore not justified. The treatment was mainly aimed at symptom control and prevention of further thromboembolic events.

CONCLUSION

Acute renal infarction can present with abdominal pain mimicking appendicitis. High clinical suspension 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.

DECLARATION OF INTEREST

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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