Unusual presentation of more common disease/injury

Infective endocarditis presenting with loin pain

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A 28-year-old previously healthy man presented to a peripheral hospital several hours after onset of acute right flank pain. A kidney stone was suspected clinically as the patient was otherwise well appearing, afebrile and normotensive. Renal function was normal and urinalysis showed no haematuria or white blood cells. A contrast CT scan of the abdomen revealed a filling defect in the ventral branch of the right renal artery with no distal perfusion suggesting a renal embolus. Subsequent investigations revealed blood cultures positive for coagulase negative staphylococcus and echocardiogram showed a bicuspid aortic valve, a dilated aortic root and moderately dilated ascending aorta. The patient was transferred to a tertiary care hospital and transesophageal echocardiogram revealed severe aortic insufficiency and thickening of the aortic valve suggestive of endocarditis. Following antibiotic treatment, blood cultures became negative and the patient underwent successful semi-urgent aortic root replacement. Renal function remained normal throughout.

BACKGROUND

Endocarditis is a potentially life-threatening condition that may mimic other conditions. This case is important because endocarditis presented untypically, as renal colic, in an otherwise healthy appearing young man. Renal infarction is not common and the differential diagnosis of endocarditis should always be considered.

CASE PRESENTATION

A 28-year-old man presented to a peripheral hospital with an 8-h history of acute right flank pain. Two weeks prior to presentation he reported a flu-like illness with predominantly upper respiratory tract symptoms that resolved spontaneously. He denied any recent fever, chills, dyspnoea on exertion or chest pain. He had no medical history, including no history of congenital heart disease or cardiac abnormalities, no connective tissue disorders, no renal calculi, no illicit drug use and no history to suggest any malignancy. He had no family history of kidney stones or hypercoagulable state. He was employed and active until the day of presentation to hospital. On examination the patient was haemodynamically stable, afebrile and no abnormalities, specifically no murmurs, were reported on initial physical examination besides tenderness in the right abdomen and flank region.

On arrival at the tertiary care centre 48 h later, vital signs were stable with a temperature of 37°C, blood pressure of 131/81, heart rate of 84, regular and respiratory rate of 16. He appeared clinically well but in discomfort. On physical examination the patient was of normal build. He was euvolemic. His chest was clear. Precordial examination revealed a non-displaced apex beat, no thrills or heaves, soft heart sounds and a 2/4 diastolic murmur over the aortic area. Abdominal examination revealed no organomegaly but tenderness and guarding in the right lower quadrant and flank. There were no audible bruits. Examination of the extremities revealed no rashes and no evidence of intravenous drug use. There were no peripheral stigmata of endocarditis; funduscopy was not performed.

INVESTIGATIONS

On initial presentation to the peripheral hospital laboratory results were as follows: white blood cell (WBC) $18.1 \times 10^9/l$ with $13 \times 10^9/l$ neutrophils, haemoglobin 148 g/l, platelets $240 \times 10^9/l$, creatinine 94 $\mu\text{mol/l}$, urinalysis revealed 2+ protein and 2+ leucocytes, no blood, alkaline phosphatase 109 U/l, alanine transaminase 180 U/l (normal <50), aspartate transaminase 159 U/l (normal <40) and lactate dehydrogenase 1402 (normal 100–225). CT scan and subsequent CT angiography of the abdomen revealed occlusion of the ventral branch of the right renal artery with no perfusion in its distribution affecting approximately half of the right kidney. The most likely source was an embolus. Subsequent hypercoagulable investigation was negative. Urine culture revealed no growth. Blood cultures were positive within 36 h for coagulase-negative staphylococcus.

DIFFERENTIAL DIAGNOSIS

The initial differential diagnosis based on clinical presentation was a kidney stone or possible pyelonephritis, but after CT imaging it became clear this was a renal infarct likely an embolus. Nephrology telephone consultation was obtained. The source of the embolus at the time was not clear, there was no description of a murmur, no evidence of arrhythmia, myocardial infarction, other embolic phenomena and he did not appear systemically sick or septic. A quick review of the literature revealed elevation of WBC was not uncommon with renal infarction. The differential diagnosis was idiopathic versus cardiogenic embolus. He was not initially suspected to have endocarditis, but echocardiogram and cultures were ordered for completeness.

TREATMENT

The case was discussed with interventional radiology and urology. Thrombolysis or embolectomy was considered, but not performed because of the time delay since onset of pain and the requirement of a 3-h air journey to the tertiary hospital. Renal parenchyma was unlikely still salvageable.

Table 1 Summary of published cases of renal infarction associated with infective endocarditis

Author	Patient age/gender	Presenting complaint	Diagnostic modalities	Pathogen	Management	Outcome
Current case	28/M	Flank pain	CT scan	Coagulase negative staphylococcus	Antibiotics, valve replacement	Survived
Alapont <i>et al</i> ¹¹	65/F	Lumbar pain	Blood cultures, pathology	<i>Staphylococcus epidermidis</i>	Percutaneous angiography, nephrectomy, antibiotics	Survived
Berliner <i>et al</i> ⁹	57/F	Fever/flank pain	CT scan, pathology	<i>Streptococcus agalactiae</i>	Mitral valve replacement, antibiotics	Survived
Ohebshalom <i>et al</i> ¹⁰	40/F	Fever/flank pain/haematuria	CT scan	<i>S viridans</i>	Radiographic embolisation, nephrectomy + splenectomy, mitral valve replacement, antibiotics	Survived
Allen and Warren ⁴	36/M	Flank pain/fever	CT scan	α -Haemolytic streptococcus	Angiogram, thrombectomy, antibiotics	Survived
Zakaria <i>et al</i> ⁶	29/M	Flank pain	CT scan	Unknown	Antibiotics	Survived
Liao <i>et al</i> ¹³	28/M	Abdominal pain, fever	CT scan	Unknown	Medical management	Died
Mügge <i>et al</i> ¹⁴		Abdominal pain	Autopsy	<i>Candida albicans</i>	Mitral valve replacement, medical management	Died
Chen <i>et al</i> ¹⁵	63/F	Fever, flank pain	CT scan	<i>S anginosus</i>	Antibiotics	Survived
Shoji <i>et al</i> ¹⁶	68/M	Fever, UTI	CT scan	Unknown	Valve replacement, antibiotics	Survived
Nakayama <i>et al</i> ¹⁷	46/F		Flank pain	Unknown	Antibiotics, mitral annuloplasty	Survived
Son <i>et al</i> ¹⁸	12/F	Fever, flank pain	CT scan	<i>S aureus</i>	Antibiotics, mitral valve replacement	Survived

Region in which the patient 1 was seen/treated: USA/Canada

by that time. Empiric anticoagulation was started. As soon as blood cultures were reported positive the patient was initiated on empiric vancomycin.

OUTCOME AND FOLLOW-UP

The transthoracic echocardiogram in the peripheral hospital revealed an ejection fraction of 65%, normal left ventricular wall thickness and no regional wall motion abnormalities. The right atrium was mildly enlarged. The aortic valve was bicuspid with thin mobile leaflets. The aortic root was dilated at 48 mm with a 52 mm sinus of valsalva aneurysm. There was moderate to severe aortic regurgitation. No dissection flap was seen. No thrombus or mass was seen. The patient was airlifted to the tertiary hospital where transoesophageal echo confirmed these findings. No dissection was seen. The anterior cusp of the aortic valve was thickened. Blood cultures became negative after 48 h. CT angiogram of the chest and abdomen revealed no other abnormalities and bone scan was negative for osteomyelitis. Phenotypically the patient did not have features of Marfan's syndrome. Two weeks after presentation the patient underwent aortic root replacement. No vegetations were seen but a non-coronary leaflet was ruptured and had a 2x2 cm hole. Histological examination of the aortic valve and root confirmed infective endocarditis and mild degenerative change of the aortic root. Antibiotics were continued for 6 weeks. Serum creatinine remained unchanged and normal after 6 months.

DISCUSSION

Renal infarction is a very rare entity with an estimated incidence of 0.007% of emergency room presentations.^{1 2} The most common aetiology of clinically significant renal infarction includes embolism due to cardiogenic causes

and renal trauma.^{3 4} Although clinically symptomatic renal emboli is rare in patients with endocarditis, postmortem study of patients found septic emboli in 24–31% of patients.⁵

Huang *et al*⁶ collected 38 cases of renal infarction over a 10-year period aiming to identify the most common clinical features. They found 21.1% of all renal infarction cases (8/38) were caused by infective endocarditis or valvular heart disease. In most cases, diagnosis was delayed because of atypical presentation. Although not specific, the uniformly common presentation was the presence of flank pain. Fever and signs of sepsis were not common, haematuria was present in 71% and proteinuria in up to 90% of cases.^{1 3 6 7} Lactate dehydrogenase, as an indicator of cell necrosis, has shown to be the most sensitive laboratory finding in cases of renal infarction although not specific to cases of endocarditis.^{3 8} CT scanning is accepted as the appropriate imaging modality replacing angiography, which has been considered the gold standard.^{3 4 9–10}

There are few other examples of renal infarcts caused by endocarditis in the literature over the last two decades and a summary of the available data is presented in table 1. Alapont *et al*¹¹ reported a case of significant renal haemorrhage (Wunderlich syndrome) in the setting of endocarditis caused by *Staphylococcus epidermidis* as in our case. Renal infarction of any cause is associated with mortality and the need for major invasive surgery in 50% of cases.^{1–3 12} Our patient did well clinically, although the diagnosis of endocarditis was made 2 days after presentation. In the literature, 2 out of 11 patients died as a result of the combination of endocarditis and renal infarct underscoring the severity of the condition and the need for heightened awareness of both renal infarction and the possibility of associated endocarditis allowing prompt diagnosis and treatment.

Learning points

- ▶ Consider renal infarction in patients presenting with flank pain, fever and urinary abnormalities.
- ▶ Consider endocarditis early when otherwise healthy patients present with renal infarction.
- ▶ Serum LDH is a sensitive, though non-specific, marker for renal infarction but may assist in narrowing differential diagnosis from renal calculi and pyelonephritis.
- ▶ CT angiography is the most appropriate initial test for early diagnosis of renal infarction.
- ▶ Endocarditis should always be considered in the differential diagnosis of atypical presentations when an aortic diastolic murmur is present.¹⁹

Competing interests None.

Patient consent Obtained.

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