



An uncommon cause of acute flank pain: renal infarction

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Renal infarction is a rare condition due to the occlusion of the renal artery by thromboemboli of cardiac origin or by in situ thrombosis. However, the frequency of renal infarction may be higher than detected, because the diagnosis is often missed or delayed because of nonspecific presentation of symptoms. A prompt diagnosis is a challenge, and it is crucial in preserving renal function. Here, we describe a case of cardioembolic renal infarction that, despite being immediately detected and treated, led to impaired renal function due to the prolonged duration of ischemia.

A 29-year-old woman with a history of iatrogenic dilated cardiomyopathy (NYHA I)—secondary to anthracycline therapy performed to treat a leukemia that had occurred in childhood—was admitted to the Emergency Department complaining of acute right flank pain started about 12 h before. She reported an acute onset of pain, immediately after awakening, not improved by painkillers. Vital signs were within normal limits. Physical examination revealed right flank tenderness to palpation. Laboratory findings demonstrated a slight leukocytosis, marked elevation of C-reactive protein (CRP 280 mg/dl), increased serum creatinine (creatinine 1.38 mg/dl), increase of the cellular injury enzyme lactate dehydrogenase (LDH 1559 UI/l), alanine aminotransferase (ALT 94 UI/l) and aspartate aminotransferase (AST 123 UI/l); plasma concentration of pro-brain natriuretic peptide was about 17,000 ng/l. The electrocardiogram showed sinus rhythm. The abdominal ultrasound ruled out stone disease. Therefore, an abdominal computed tomography

angiography (CTA) was performed revealing a complete thrombosis of the right renal artery with secondarily absent perfusion of the ipsilateral kidney (Fig. 1a). Consequently, a mechanical thrombectomy was performed, with restoration of the renal artery flow on completion of arteriography (Fig. 1b, c); to follow, anticoagulation therapy with low-molecular weight heparin was started. Afterwards, there was both a gradual improvement of the pain and a reduction in the levels of creatinine, LDH and ALT. Nevertheless, the radioisotope renogram, performed 5 days after revascularization, showed minimal uptake within the right kidney (Fig. 1d), which was functionally excluded for probable impairment of the microcirculation. Thus, in the suspicion of cardioembolic renal infarction, a transthoracic echocardiogram was performed showing a moderately reduced ejection fraction (38%) and a significant enlargement of the left atrium (biplane volume of 77 ml). Next, a transesophageal echocardiogram was performed revealing, in the left atrial appendage (LAA), reduced flow velocity and a small echogenic image on the pectinate muscles, compatible with thrombus. Hence, the patient was discharged after placing an implantable loop recorder and starting a chronic oral anticoagulant therapy with edoxaban to prevent cardioembolic recurrences. Afterwards, at 1-year follow-up, creatinine levels were within the normal range and the renal ultrasound showed a compensatory hypertrophy of the left kidney, but the radioisotope renogram confirmed a severe loss of right kidney function with respect to the contralateral kidney (Fig. 1e).

Renal infarction is infrequent and the diagnosis is usually missed or delayed because it can mimic many other acute conditions [1]. Most cases of renal infarction are caused by thromboemboli of cardiac origin in patients with atrial fibrillation, or by other conditions predisposing cardioembolic events including cardiomyopathies, presence of valve prostheses, endocarditis and thrombosis of the left ventricle. Renal infarction can also be caused by in situ thrombosis, which may be secondary to direct damage of the renal artery (dissection, trauma, fibromuscular dysplasia or vasculitis)

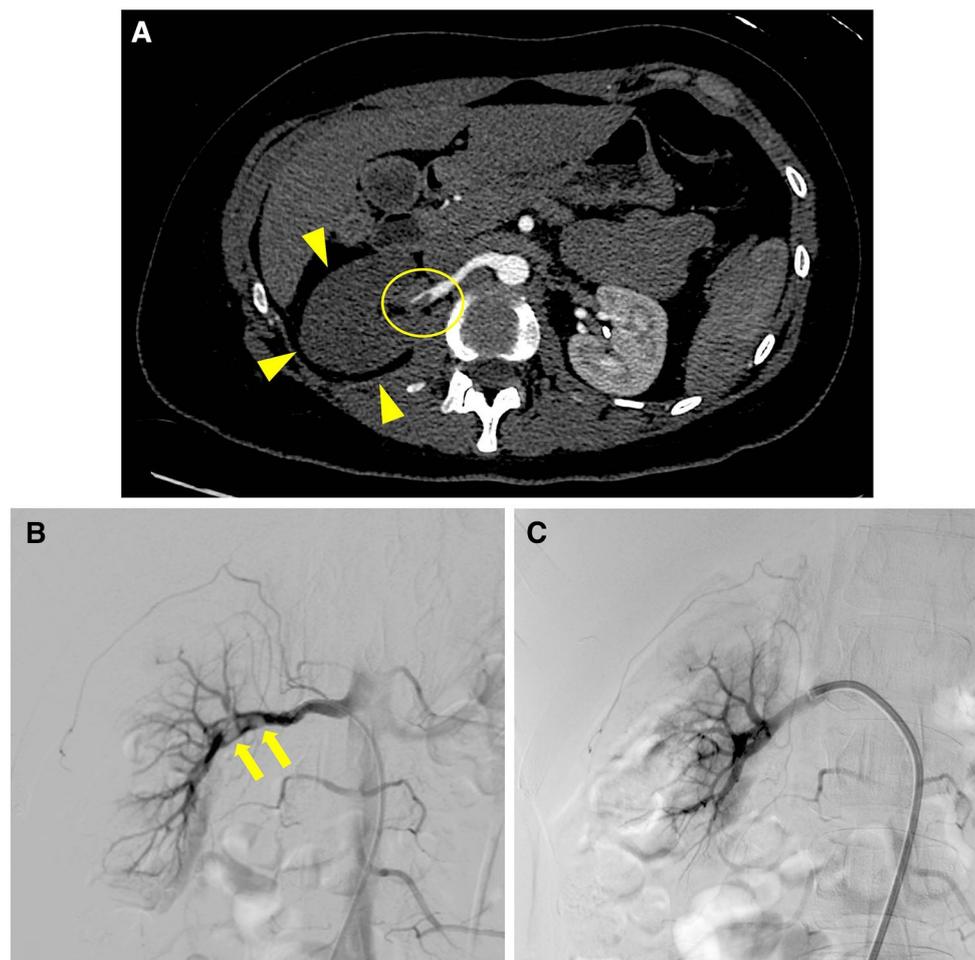
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Fig. 1 Abdominal CTA

(a) showed a complete defect of opacification in the main right renal artery, close to the hilum, referable to an embolus (yellow circle). Complete lack of right renal parenchymal enhancement was also observed (arrow heads). Renal artery angiography confirmed the presence of an embolus in the right main renal artery (arrows) with associated poor opacification of segmental arteries and the absence of parenchymal angiographic phase (b). After manual mechanical thromboaspiration, minimal increase in renal perfusion was observed (c). Radioisotope renogram performed 5 days after revascularization (d) and at 1-year follow-up (e) showed a severe loss of right kidney function with respect to contralateral kidney



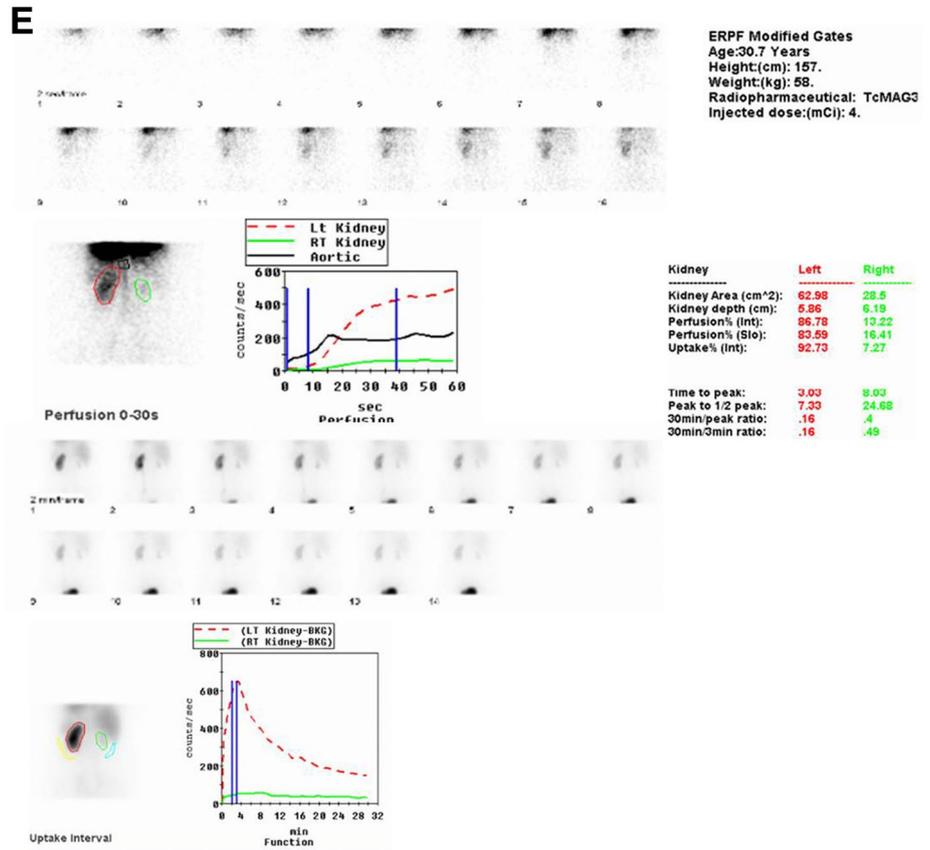
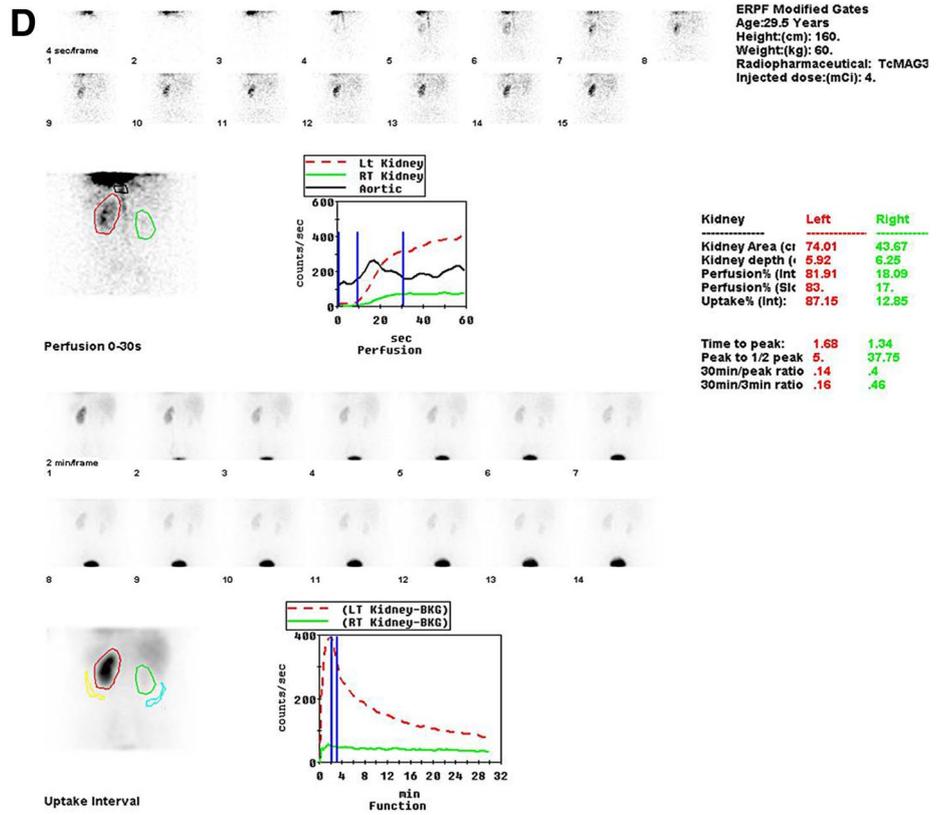
or to a hypercoagulable state (antiphospholipid antibody syndrome, iperomocisteinemia, and neoplasms). However, in a significant percentage of cases, acute renal infarction is classified as idiopathic [2]. The most common symptoms are abdominal or unilateral flank pain, nausea and vomiting [1, 2]. A leukocytosis and elevated LDH levels with little or no rise in serum aminotransferases are the most prominent laboratory findings [1–3]. Contrast-enhanced CT examination is essential for the diagnosis. In patients with acute occlusion of the main renal artery or a segmental branch artery, if the diagnosis is made up to 48 h after the onset of symptoms, endovascular treatment of thrombolysis or thrombectomy may be recommended. In the case of late diagnosis, it is advised to use anticoagulant therapy with unfractionated heparin intravenously or low-molecular weight heparin subcutaneously [4].

In the present case, the abdominal CT immediately detected renal infarction, but the late diagnosis—with respect to the onset of the symptom—did not allow the endovascular reperfusion therapy to be successful with

consequent loss of renal function. Furthermore, as highlighted, in the presence of potential embolic sources or hypercoagulable state, anticoagulant therapy should be continued to prevent recurrences. Thus, we prescribed a novel oral anticoagulant considering the patient's young age, the burden of continuing a lifelong anticoagulant therapy and the need for a better compliance with extended treatment due to no need for laboratory monitoring, lower incidence of major bleeding, and minor drug and food interactions.

In conclusion, renal infarction should be considered in the differential diagnosis of a patient complaining of unilateral flank pain, in the presence of potential predisposing factors and suggestive laboratory tests. Therefore, the diagnosis is initially based on a relevant clinical suspicion, pivotal to promptly address the appropriate diagnostic investigations to proceed with an optimal diagnostic workup, and to choose the best therapy to allow kidney rescue.

Fig. 1 (continued)



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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Statement of human rights and animal rights This case image complies with the ethical standards outlined in the journal. It involved the medical management of a patient as per society guidelines and was in accordance with the ethical standards of the institution. This case was not formal research involving human participants and/or animals.

Informed consent Informed consent was taken from the patient.

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