International Journal of Urology and Nephrology Vol. 2 (3), pp. 055-058, November, 2014. Available online at www.internationalscholarsjournals.org © International Scholars Journals

Case Report

A rare cause of renal infarct: Paradoxical embolism through the patent foramen ovale

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Accepted 1 August, 2014

Renal infarction is a rare clinical problem and the diagnosis is frequently missed or delayed because of its nonspecific symptoms. The two major causes of renal infarction are thromboembolism and in-situ thrombosis. Paradoxical embolism is defined as a systemic arterial embolism requiring the passage of a venous thrombus into the arterial circulatory system through a right-to-left shunt. Renal infarction secondary to paradoxical embolism has rarely been described. Here we present a case of renal infarction due to paradoxical embolism through the patent foramen ovale. A 26-year-old woman presented to the emergency room with a four-day history of left flank pain. The level of lactate dehydrogenase was detected five times higher than the upper limit of normal with no rise in serum aminotransferases. A contrast-enhanced computed tomography scan revealed renal infarction and perinephric mild fluid in the left kidney. An electrocardiogram showed sinus rhythm and the thrombosis panel was negative. A transesophageal echocardiography showed a patent foramen ovale with a right-to-left shunt. Although we have not found the source of embolism, paradoxical embolism through to PFO was strongly suspected. The patient was treated by anticoagulant therapy at a curative dose and the outcome was favorable.

Key words: Renal infarction, paradoxical embolism, patent foramen ovale, flank pain, transesophageal echocardiography.

INTRODUCTION

A paradoxical embolism is one that originates in the systemic veins or right side of the heart and crosses to the systemic arterial circulation through a right-to-left shunt (Corrin, 1964). The most common cardiac defect associated with paradoxical embolism is the patent foramen ovale (PFO) (Ward et al., 1995). Patent foramen ovale is a congenital cardiac lesion that frequently persists into adulthood. Although most patients with a PFO are asymptomatic it may be associated with various clinical manifestations; usually and most importantly with a cerebral embolism, less frequently with a systemic or a paradoxical embolism (Hara et al., 2005).Renal infarction due to paradoxical embolism has rarely been described. Renal infarction is a very rare condition and diagnosis can be missed because of its nonspecific symptoms like flank pain, backache, nausea and vomiting (Korzets et al., 2002). Here we report a case of renal infarction secondary to paradoxical embolism through the PFO.

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CASE REPORT

A 26-year-old woman was admitted to the emergency room with left flank pain which started 4 days ago. There was no history of fever or renal stones. No specific medical history was reported and she was not on any medications. There was consanguinity between parents and her sister had familial Mediterranean fever (FMF). There was no history of smoking or drinking.

On physical examination the patient's blood pressure, pulse rate, respiratory rate and body temperature were 120/70 mm Hg, 72 beats/minute, 14 breaths/minute and 36.7 °C respectively. Abdominal examination revealed

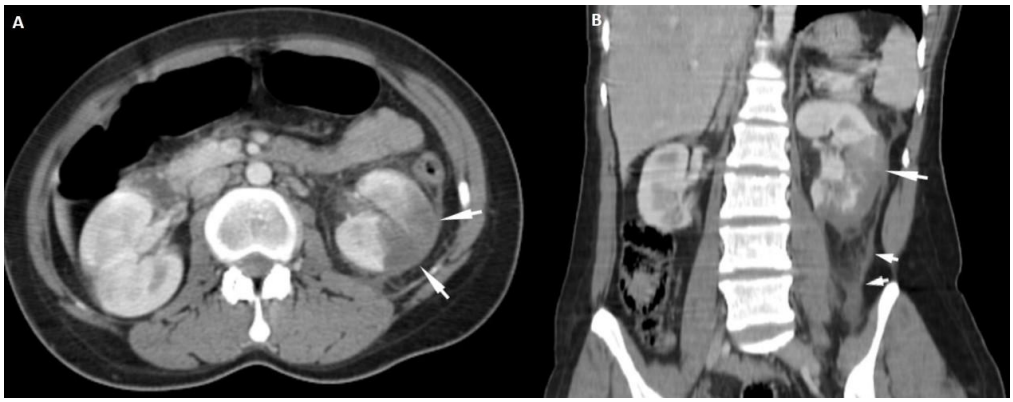

Figure 1. Contrast enhanced CT axial (A) and coronal (B) images shows less enhanced wedge shaped areas that involve both cortex and medulla consistent with renal infarct (A, B, long arrows). Also, perinephric stranding and mild fluid because of edematous changes were noted (B, small arrows).

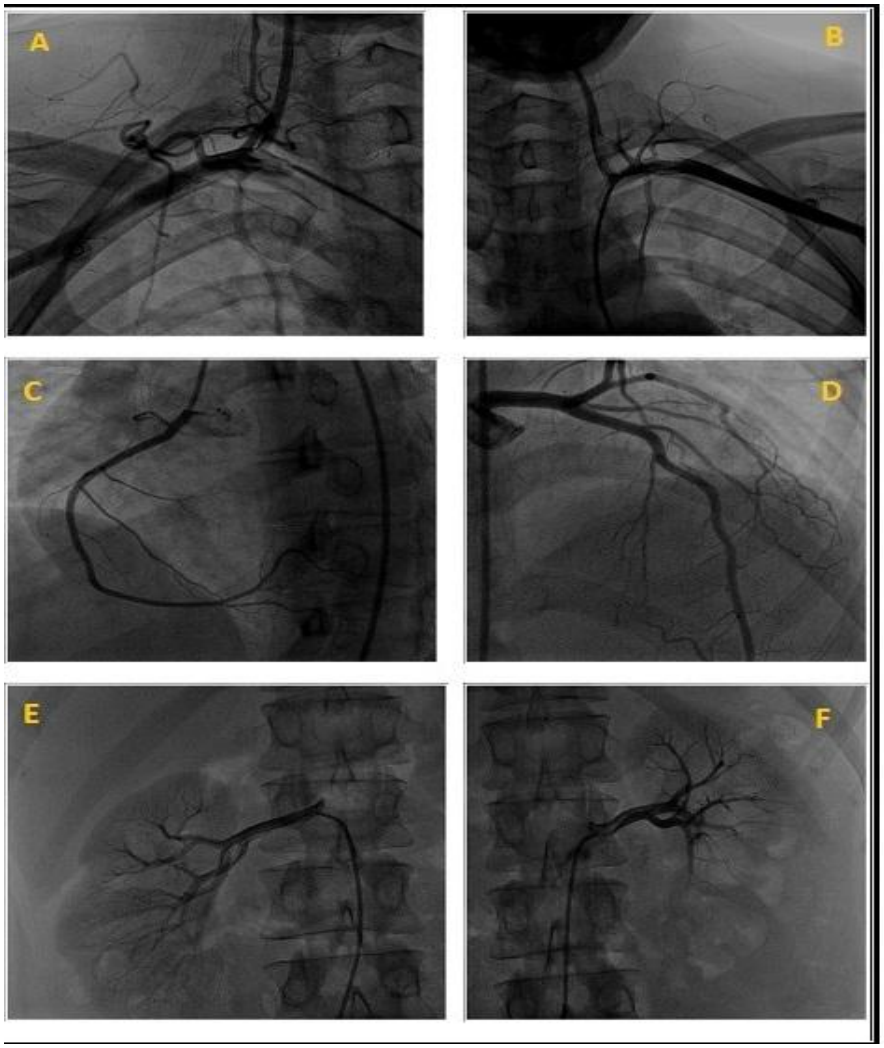

Figure 2: Carotid, coronary and renal artery angiography
A and B: Carotid angiography shows right (A) and left (B) carotid arteries were patent.
C and D: There is no occlusion in right coronary artery (C), left anterior descending artery and left circumflex coronary artery (D) on coronary angiography.
E and F: Renal angiography shows normal flow in both renal arteries but there is no blood flow in the lower lobe of the left kidney (E and F).

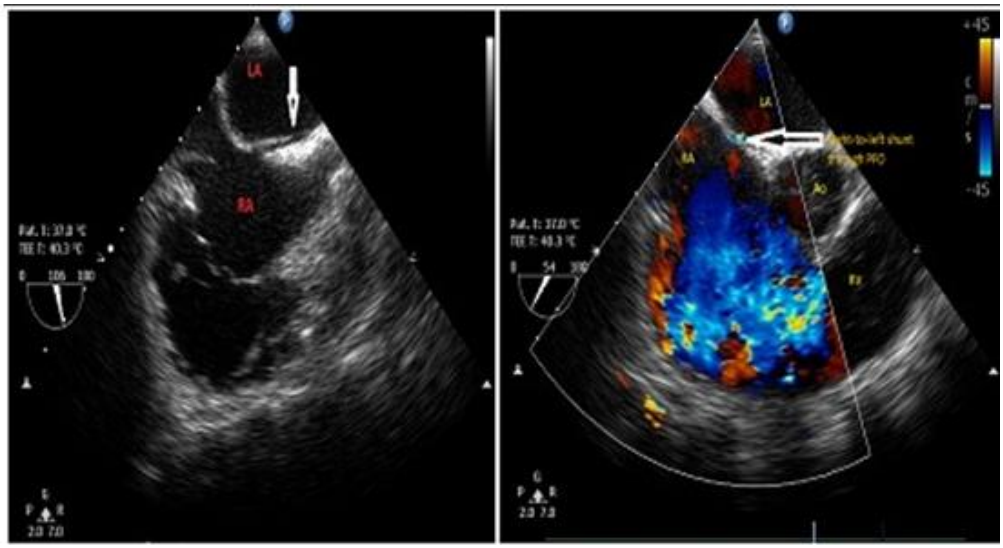

Figure 3. Transesophageal echocardiography shows transition from right to left through PFO.

tenderness but no rebound on left costovertebral angle with palpation. The breath sounds was clear and the heartbeats was regular with no murmur. She had a white cell count of 14, 2 X 10^3 (normal range: 3, 8-10 x 10^3) µ/L, hemoglobin of 11, 3 (normal range: 13-17, 5) g/dL, platelets of 178 X 10^3 (normal range: 150-400 x 10^3) µ/L. Kidney function tests were within normal limits. Biochemical parameters were within normal limits except of elevated lactate dehydrogenase value of 1037 (normal range 0-214) U/L. The activated partial tromboplastin time and prothrombin time were normal. Urinalysis showed 3+ hemoglobinuria and 5 erythrocytes without leukocytes and proteinuria. Antinuclear antibodies, anti-neutrophil cytoplasmic antibody, dsDNA antibody, anticardiolipin antibodies, antiphospholipid antibodies, anti-beta-2-glicoprotein-1 antibodies and lupus anticoagulant were negative. The level of antithrombin-3, homocysteine, protein S and protein C were within normal limits. FMF, factor-5 Leiden, prothrombin and methylenetetrahydrofolate reductase gene mutation were not detected. An electrocardiogram revealed sinus rhythm and a chest radiograph showed no abnormal findings.

A contrast-enhanced abdominal computed tomography(CT) scan determined wedge shaped areas that were consistent with renal infarct in the left kidney and perinephric stranding and mild fluid as edematous changes (Figure 1). Bilateral renal artery Doppler ultrasonography (USG) was performed to exclude the renal artery thrombus and dissection. Both renal arteries were patent. The carotid, coronary and renal angiography revealed no abnormalities except of no blood flow in the lower lobe of the left kidney (Figure 2). Transthoracic echocardiogram showed a normal left ventricular function without any evidence of regional wall motion abnormality, intramural thrombus or valvular pathology. A PFO with a right-to-left shunt was observed on transesophageal echocardiogram at Valsalva (TEE) (Figure 3). Anticoagulant therapy was initiated with 60 mg enoxaparin twice daily. After the dose of anticoagulant therapy was adjusted the patient was discharged home without any complications.

DISCUSSION

Renal infarction is one of the cause of acute abdominal and flank pain but the diagnosis is often missed or delayed due both to its non-specific clinical presentation and the rarity of the disease (Korzets et al., 2002). The incidence of renal infarction was reported to be 0,007 %-1, 4 % in the literature butit is difficult to estimate the true frequency because of the clinical picture is similar with more common disease like pyelonephritis and urolithiasis. (Korzets et al., 2002;Saunders et al., 1995).

Patients with acute renal infarction complain of the flank pain, abdominal pain, often accompanied by nausea and vomiting and less frequently fever (Chu et al., 2006). Laboratory findings include elevated peripheral white blood cell count, serum creatinine concentration and lactate dehydrogenase (LDH) and gross or microscopic hematuria. Elevated serum LDH level, often more than five times the upper limit of normal with little or no rise in serum aminotransferases is suggestive of renal infarction (Domanovits et al., 1999).In our case there was flank pain, leukocytosis, elevated LDH and hematuria with a normal level of aminotransferases.

The two major cause of renal infarction are thromboembolism, which usually originate from a thrombus in the heart or aorta and in-situ thrombosis of a renal artery, which is less common (Tunick et al., 2002). The other causes of renal infarction are fibro-muscular dysplasia of renal arteries, vasculitis, hypercoagulable states, trauma, connective tissue diseases and cardiac

diseases; sometimes it can be idiopathic (Domanovits et al., 1999; Walsh et al., 1985). The possible sources of embolism are atrial fibrillation, left atrial myxoma, infective endocarditis, cholesterol embolism and paradoxical embolism (Domanovits et al., 1999; Carey et al., 1999).

Currently, spiral CT without contrast is preferred for the evaluation of patients with acute flank pain suspected of acute ureterolithiasis and nephrolithiasis. If there are no signs of ureteronephrolithiasis, a contrast-enhanced CT scan should be performed to exclude renal infarction. The classic finding is a wedge-shaped perfusion defect (Korzets et al., 2002). Computerized scanning was the mode of imaging in our patient, too. Most of the patients diagnosed with paradoxical renal infarction present with multi organ involvement such as brain, eyes, kidneys, spleen, intestines and extremities but only renal involvement was present in our patient.

Patent foramen ovale is a congenital cardiac lesion. It is the residua of foramen ovale, which allows blood to flow across the atrial septum in fetal circulation. It closes shortly after birth in %75 (Hara et al., 2005). Although most patients with PFO are asymptomatic, it is associated to the following clinic situations: ischemic stroke, cryptogenic stroke, migraine and vascular headache, decompression sickness and air embolism, platypea-orthodeoxia syndrome, high-altitude pulmonary edema, acute myocardial infarction, systemic embolisation such as renal infarction, fat embolism, tumor embolism, and left-sided valve disease in carcinoid syndrome. The most important potential manifestation is ischemic stroke due to a paradoxical embolism (Bolderman et al., 2006; Rosenberger et al., 2003; Lumerman et al., 1999; Lessman et al., 1978; London et al., 1968; Steckel et al., 1984).

Evaluation for PFO is indicated in patients with cryptogenic stroke or other embolic events such as renal infarction. A variety of ultrasound modalities have been used to diagnose a PFO, including TTE, TEE, transcranial Doppler, transmitral Doppler and intracardiac echocardiography (ICE) (Di Tullio et al., 1993; Droste et al., 2004; Fenster et al., 2014). Among the ultrasound methods for detection of right-to-left shunts, only TEE and ICE enable visualization of the site of the shunt. Echocardiography can also detect other cardiac abnormalities associated with embolic events such as atrial septal aneurysm (ASA) and intracardiac mass (e.g. vegetation, tumor or thrombus) (Pepi et al., 2010). In summary, the most definitive diagnostic test is TEE - with contrast at rest, with cough and following Valsalva - to evaluation of PFO that indicated in patients with embolic events. Our case could only be diagnosed by TEE.

There is no standard approach in the treatment of renal infarction cases due to paradoxical embolism. While some authors prefer conservative approaches such as anticoagulant or thrombolytic drugs, others prefer the closure of the right-to-left shunt surgically or with a trans-catheter procedure (Nara et al., 2011).Closure of the PFO after the first embolic event is recommended for patients at high risk of recurrent embolic events. The risk factors associated with embolic recurrence include ASA, high shunting volume, shunting at rest, large PFO (more than 3.4 mm), higher mobility of the PFO valve, a well developed Eustachian valve, a Valsalva maneuver immediately prior to event and a history of recurrent embolic events (Guo et al., 2007). Although the source of embolism was not proven in our case, paradoxical embolism through to PFO was strongly suspected and anticoagulant therapy was started and because of there was none of the risk factors mentioned above, a surgical procedure was not planned.

In conclusion, renal infarction should be kept in mind and diagnostic tests should be considered in patients presented with flank pain and cardiac pathologies should be investigated in the etiology. Treatment should be adjusted according to the patient because of there is no data comparing surgery to anticoagulant therapy.

COMPETING INTEREST

All the authors have declared no competing interest.

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