Case report

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Multiple renal infarctions due to thromboembolism. Importance of ultrasound in diagnosis. Case report.

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Abstract

We present the case of a woman with known risk factors for embolism with multiple kidney infarctions. She was admitted with left renal colic, discrete hematuria and subfebrility. She was under acenocumarolum treatment but without efficient anticoagulation. Ultrasound performed at the emergency room revealed smaller right kidney (congenital hypoplasia) and moderately enlarged left kidney with hypoechoic areas in the parenchyma. Computer tomography scan with contrast agent revealed multiple avascular areas within the left kidney. Evolution was favorable after efficient anticoagulation. Contrast enhanced ultrasound performed two weeks later revealed only one residual avascular area. Differential diagnosis was made with acute pyelonephritis, cholesterol embolism and acute tubular necrosis in a diabetic patient.

Keywords: renal infarction, embolism, contrast enhanced ultrasound.

Introduction

Kidney infarction is a rare condition and the diagnosis can be misleading due to the higher frequency of other causes for lumbar pain, such as kidney stones and pyelonephritis. The common cause of kidney infarction is an embolism in the main renal artery or more common in their branches. The emboli can be tromboemboli originating from left heart cavities or atheroembolic (cholesterol emboli) from aortic calcifications. A precise diagnosis of thromboembolism and cholesterol embolism can be made on a clinical basis and via imagistic methods [1].

Case report

A 65 year old woman was admitted with acute, high intensity pain (which had alternated with asymptomatic periods) of the left lumbar region and left abdomen. She had had atrial fibrillation for a few months, being under no efficient anticoagulant therapy. One month prior presentation she underwent a transient ischemic stroke. Other co-morbidities were: type 2 diabetes mellitus, controlled hypertension, chronic coronary ischemia and hypercholesterolemia, and congenital hypoplasia of the left kidney was prior identified.

At presentation we found an overweight woman, with body mass index 28, in considerable distress, with lumbar pain, with transient augmentation, suggesting a left renal colic. Her body temperature was 37^0C, respiratory rate 24/min, and blood pressure 165/105mmHg. On clinical examination pallor was noticed, fine crackles at the basis of the lung, tachycardia with irregular heartbeats 112/min and the left lumbar region was very sensitive to percussion. Laboratory tests detected leukocyte count 12000/mm^3, hemoglobin 11,3g/dl, urea 50mg/dl, creatinine 1,01 mg/ml, blood sugar 130 mg/dl, LDH 680U/l, and urine sediment with microhematuria. INR (international normalized ratio) at admission was 1.5. In the emergency room an ultrasound (grey scale and Doppler) examination of the abdomen was performed. The left kidney was enlarged with hypoechoic areas and only slightly vascu-

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lar signal in color Doppler examination. In Doppler Duplex a moderately increased resistivity index was found (fig 1). Right kidney was smaller, 8.5 cm in long axis, this finding being known before and described as a congenital small kidney. Considering the patient co-morbidities (atrial fibrillation and recent ischemic stroke), we suspected a kidney infarction and a CT scan with contrast agent was performed and multiple hipodense lesions suggesting avascular areas with triangular shape in the left kidney were described (fig 2).

After anticoagulant adjustment (INR between 2.5 and 3) and symptomatic therapy, the patient evolution was favorable. After two weeks, contrast enhanced US was performed. A good vascularisation in the entire kidney was found, except in the inferior third where an avascular triangular area persisted in both cortical and medullar phase after contrast agent injection (fig 3). In the early phase (18 sec after contrast agent i.v. bolus administration) a complete avascular area in the lower pole of the left kidney was clearly delimitated. In the late phase, a slight vascular signal was seen in the periphery of the lesion after 2 min after injection.

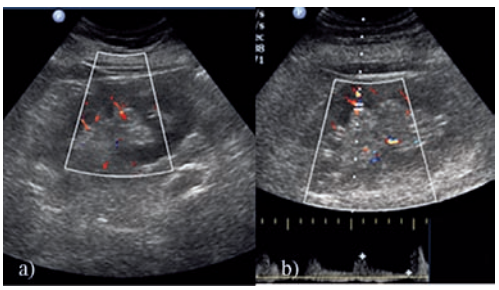

Fig 1. a) Doppler US of the left kidney: hypoechoic areas and poor arterial signal; b) Duplex Doppler US of the left kidney – high resistivity index.

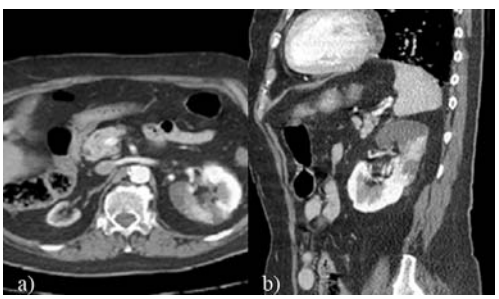

Fig 2. CE-CT scan of the abdomen a) transversal view – multiple hypodense areas in the left kidney and calcifications within the aorta; b) sagital view showing the extensive avascular areas.

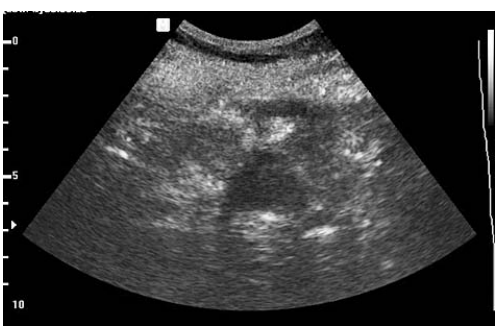

Fig 3. CE-US in the cortical phase, showing an avascular area in the lower third of the left kidney.

Discussions

Thromboembolism is generally caused by thrombi located in the left atrium or atrial appendage in cases of atrial fibrillation and from the left ventricle in cases of myocardial infarction and parietal thrombus. Also, the thromboemboli can originate from aortic instable thrombosed plaques. Atheroembolism is caused by cholesterol crystals from the aortic wall calcifications that can be mobilized into the renal arteries usually after aortic catheterization. In our case no interventional aortic procedures were made so cholesterol emboli were ruled out as the cause of the embolism. Although cholesterol embolism in the kidney is very rare, it can be misinterpretated as other causes of renal pain so the exact incidence is not known [1].

The predictive parameters for kidney infarction were studied by Domanowitz et al [2]. They found that the risk factors for kidney infarction are atrial fibrillation, previous embolism, hypertension, and ischemic cardiac disease [2]. In our patient all of these factors were found, but the most important was the inefficient anticoagulation. Due to the acute lumbar pain and leucocytosis a differential diagnosis with pyelonephritis was made, but no evidence of infection was found. Microhematuria and lumbar pain in a diabetic patient could suggest acute tubular necrosis, but it usually evolves with kidney failure [3,4]. Although the contrast enhanced US is considered to be a precise method for renal infarction, due to technical problems we performed it two weeks later. Indication of CEUS in renal vascular diseases was stated by EFSUMB in 2008, and arterial pathology is one of the indications [5,6].

Conclusions

Although a rare clinical condition, renal infarction can occur in cases with risk factors for thromboembolism or atheroembolism. Clinical features combined with imagistic methods can lead to the final diagnosis. In our case the presence of risk factors, absence of aortic cath-

eterization and rapid evolution after anticoagulation established the diagnosis of thromboembolism. Ultrasound is the preferred imagistic method being noninvasive. Grey-scale, color Doppler, Duplex Doppler and contrast enhanced US are methods easy to perform but special devices are required. In rare cases of multiple infarctions contrast enhanced CT scan is more reliable, but it cannot be used in renal failure patients.

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