Acute renal infarction of unknown origin in a young male. A case report

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Abstract
Within the variety of urologic emergencies, very few entities seem to be so deceiving like acute renal infarction. This rare disease mimics other common conditions as urolithiasis, lumbar or other abdominal lesions, detected therefore rarely early in clinical practice. In this paper, we present a case of an acute renal infarction in a healthy 42-year old man, who was initially misdiagnosed with renal colic, before the diagnosis of the disease was finally established. Riffing the relative literature, trigger points are searched, which can make the early diagnosis of a RI possible.

Introduction
Renal infarction (RI) is a deceiving entity of unknown incidence in the emergency departments (EDs). Patients suffering from RI usually present with abdominal or flank pain, elevated levels of lactate dehydrogenase (LDH) and/or hematuria1. The diagnosis is often delayed since the clinical spectrum of the disorder guides the suspicion in other common conditions like renal colic, pyelonephritis or abdominal pathology2. Although most patients with RI are old and have a high risk of thromboembolism, younger patients not carrying any obvious risks should not be excluded3. Contrast-enhanced CT is thought to be a ‘sine qua non’ in terms of RI diagnosis, therefore should be performed if suspicion is raised4. Herein, we present a case of a RI in a young man, who was initially misdiagnosed with renal colic.

Case presentation
A 42-year old male smoker present-
ed to the ED with acute onset of left flank pain and nausea for a few hours. No history of illnesses or trauma was reported. Vital signs were temperature of 36.6°C, pulse rate of 80 beats/minutes, normal respiratory rate and blood pressure. Physical examination revealed left flank tenderness. Urine analysis demonstrated hematuria, while urine pH was 5. Blood examination showed no leucocytosis and normal hematocrite. Markers of blood coagulation (PT, PTT, INR) and biochemical tests, including LDH, were normal. KUB x-ray was negative for radiopaque lithiasis. Thus, a radiolucent lithiasis renal colic was hypothesized and the patient was treated with analgesics. After the remission of pain, the patient received relational instructions and was counseled for reexamination.

After 24 hours the patient presented again reporting worsening of the flank pain. Vital signs were normal. Blood tests showed mild leukocytosis. Creatinine was elevated in 1.8 mg/dl. Liver enzymes were also mildly elevated but LDH was markedly increased in 780 IU/L. Urine analysis showed hematuria and mild proteinuria. Ultrasonography was not contributive, showing neither lithiasis nor obstruction. In front of a differentiation concern, further investigation was decided.

The patient was referred to Radiology department and CT without and after IV contrast was performed. In non-enhanced images a mild thickening of the perirenal fascia was found (figure 1a). After contrast-material administration, CT demonstrated a focal, triangular-shaped defect of renal parenchyma situated at anterior upper segment that involved
both the cortex and medulla; a featuring cortical-rim sign, due to collateral capsular perfusion, was also observed (figure 1b, 2a). Closer observation depicted an anatomic variation of left double renal artery (figure 2b) and an intraluminal thrombus of the anterior artery irrigating the anterior segment of left kidney (figure 2b, 3a). Flip-flop-enhancement sign was also seen (figure 3b). Subsequently, the diagnosis of acute segmental RI of left kidney was established.

Just following, an echocardiogram was performed demonstrating a normal left atrium and normal left ventricular size with normal ejection fraction. During hospitalization, the patient was evaluated for deep vein thrombosis, thrombophila and rheumatologic disorders; the examination panel consisted of Doppler examination of carotids arteries and veins of upper and lower limbs, measurement of blood levels of homocysteine, antithrombin, protein C and S, lupus anticoagulant and anticardiolipin antibodies. All examinations failed to designate an obvious cause of embolus.

The patient was treated with intravenous heparine and oral acenocumarole until INR in 2.5 was stabilized. The pain was totally resolved after 2 days. He was discharged from hospital after 7 days, with a creatinine level of 1.1 mg/dl. Additionally, the patient was counseled by the cardiologist to continue oral acenocumarole.

Follow-up CT at 4 months depicted an area of cortical defect in the site of the RI (figure 4), while creatinine was measured in 1.1 mg/dl which was considered as baseline. The cardiologist counseled the alteration of oral acenocumarole to prophylactic low-dose aspirin for at least further 6 months. For further monitoring of the potential sequelae regarding blood pressure and renal function the patient was referred to a nephrologist. Till now his medical course is reported uneventful.

Discussion
The actual occurrence of RI cannot be easily determined since the condition is limited reported in the literature. However, its incidence up to 1.4% in autopsy studies indicates that the disease is underestimated, demanding huge suspicion to be diagnosed ante mortem.

Advanced age and high risk of thromboembolism is almost the rule in patients with RI as it is noted in most reports. Atrial fibrillation, history of previous embolism, infectious endocarditis, valvular and ischemic heart disease, coagulation dysfunction or hematologic disease, atherosclerosis of abdominal aorta and spontaneous renal artery dissection are usually reported; atrial fibillation is the most common cause and the most common site of embolus is considered of cardiac origin. However, Bolderman et al observed a group of patients who did not carry any obvious risk but they suffered from an RI; these patients were middle-aged, smokers, being treated for hypertension or hyperlipidemia and their emboli may have been originated from the suprarenal aortic wall rather than the cardiac chambers. After evaluation, half of them were found to have underlying diseases like hyperhomocystinemia or congenital coagulation diseases, but the rest were found negative for a risk factor.

The clinical spectrum of RI consists of flank, abdominal or lower back pain, nausea/vomiting, fever/chills, even diarrhea, dyspnea or chest pain; laboratory examinations which could alert the diagnosis are LDH and the simultaneous presence of hematuria or proteinuria. Almost all patients have elevated LDH, up to 6.86-fold higher than normal, which is considered the most sensitive mark indicat-
ing further investigation. Microscopic hematuria is frequently present, although its absence may suggest a serious loss of renal function. Creatinine is not found significantly elevated and thus, not helpful but higher values may signify more severe renal damage and prolonged hospitalization. C-reactive protein, liver enzymes, blood urea or white-blood-cells may be elevated as well.

Diagnosis of acute RI via CT is established in up to 80% of the cases. It also helps ruling out other abdominal pain entities such as renal colic, appendicitis, aortic aneurysm or hepatic diseases. Furthermore, CT is noninvasive, reachable in most tertiary hospitals and is considered the standard of reference. After IV contrast administration renal infarct appears as a non-enhanced triangular-shaped zone of diminished density. Other signs are cortical-rim sign, a distal area of cortical enhancement surrounding the ischemic parenchyma and flip-flop-enhancement sign where a region of hypoenhancement on early phases becomes hyperaattenuating on delay images. In cases where non-contrast CT is performed, like in cases searching for lithiasis, imaging of perinephric stranding without hydronephrosis may be the only sign raising the suspicion of a renal infarction; if clinical and laboratory findings - like thromboembolic risk, persistence of pain and raised LDH - indicate further investigation, contrast-enhanced CT is then necessary. Doppler ultrasound may be helpful in the recognition of global rather than segmental RI, but still can be the study of choice for obstructive lithiasis or aortic aneurysm. Finally, renal angiography looks like the optimal imaging modality for final diagnosis, albeit carrying the disadvantage of invasiveness.

The management of the acute phase of RI remains unclear and lacks of standardized approach; oral or intravenous anticoagulants agents (heparine plus warfarin) or anticoagulants agents along with thrombolytics (as streptokinase) may be used. No comparative studies are available, but some authors argue in favor of conservative treatment, as far as acceptable outcome in terms of renal function is achieved in most patients, while thrombolysis carries risk of complications and gives non-superior results. Thrombolysis might be beneficial if attempted within 90 minutes from onset of pain, but even then successful revascularization may be expected in less than 50% of patients. Besides, definitive diagnosis might delay several days. In general terms, early enough diagnosis is more significant for the final outcome than treatment modality and is considered mandatory since the condition may result in unwanted sequelae like renal insufficiency or even death. Regarding the long term management of RI, there are no certain recommendations in the literature. In our case, we referred our patient to regular cardiologic follow-up, since the patient was initially treated with acenocumarole which demands regular measurement of INR and dose titration. Whereas long-term anticoagulant therapy for cardiac patients is unquestionable, the type or the duration of therapy in idiopathic RI is still unknown. Even if subsequent risk was small, it was decided our patient to be treated with a further antithrombotic prophylaxis with low-dose aspirin, as reported in the literature. Additionally, a contrast-enhanced CT was performed 4 months after the event to evaluate the condition of renal parenchyma; a DMSA scan could also be helpful. Finally, the patient was also referred to a nephrologist for the assessment of renal function and the monitoring of the possible sequel of nephrogenic hypertension.

In conclusion, RI is a rare entity which demands high level of suspicion. The typical patient is old and carries a high risk of thromboembolism, presenting with pain in the abdomen or in the flank and demonstrating microscopic hematuria, proteinuria and elevated LDH. However, young patients without obvious risk factors should not be excluded. Although renal function is likely to be preserved even in delayed diagnosis, early and definitive diagnosis and treatment is mandatory.

Abbreviations
EDs=Emergency Departments
RI=Renal Infarction,
LDH=lactic acid dehydrogenase
CT=computer tomography
PT=Prothombine Time
aPTT=partial thromboplastin time
INR=international normalized ratio
Ανάμεσα στην ποικιλία των ουρολογικών επειγόντων, ελάχιστες οντότητες δείχνουν να παραπλανούν τόσο όσο ένα οξύ νεφρικό έμφρακτο. Αυτή η σπάνια πάθηση μιμείται άλλες συχνές καταστάσεις, όπως η ουρολιθίαση, η οσφυαλγία ή άλλες κοιλιακές διαταραχές, και ως εκ τούτου σπάνια ανιχνεύεται υπό την κλινική πράξη. Σε αυτή την εργασία, παρουσιάζουμε την περίπτωση ενός οξέος νεφρικού έμφρακτου σε έναν υγιή άντρα 42 ετών, ο οποίος αρχικά διεγνώσθη εσφαλμένα με κωλικό νεφρού, έως ότου η διάγνωση της νόσου να επιβεβαιωθεί. Ξεφυλλίζοντας την σχετική βιβλιογραφία, αναζητούνται εναύσματα, που θα μπορούν να κάνουν την πρώιμη διάγνωση ενός νεφρικού εμφράγματος εφικτή.

Περίληψη

Λέξεις ευρετηριασμού

νεφρικό έμφρακτο,
θρομβοεμβολισμός, LDH,
νέος άνδρας

References