CLINICAL NOTE

Acute renal infarction: an underdiagnosed cause of abdominal pain

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Acute renal infarction as a cause of abdominal pain is rare, though its frequency is increasing along with atherosclerotic disease. Given that underdiagnosis is often a problem, it is important to bear this diagnosis in mind whenever a patient presents with abdominal and flank pain, an elevated lactate dehydrogenase level, and abnormal sediment in urine. When radiologic and ultrasound images of the abdomen are normal and no signs suggest urinary tract obstruction, contrast-enhanced computed tomography (CT) would be indicated to confirm the diagnosis; this noninvasive examination is useful because the effectiveness of treatment is time-dependent. We report the case of a woman with nonspecific abdominal pain and the aforementioned abnormal laboratory findings. CT images were diagnostic. Fibrinolytic therapy was not indicated and treatment with anticoagulants (low molecular weight heparin at therapeutic dosages) was started. The patient's condition worsened, however, and she died a few days later. [Emergencias 2010;22:117-119]

Key words: Acute renal infarction. Thrombosis, renal. Fibrinolytic therapy.

Introduction

Acute renal infarction (ARI) is an uncommon cause of abdominal pain and is under-diagnosed as a cause of kidney failure. Early diagnosis is important, since renal function can be restored with revascularization of an occluded vessel[1].

The symptoms are usually nonspecific, and may mimic other diseases such as back pain, renal colic, pyelonephritis, biliary colic and / or cholecystitis among others, hence the importance of considering this possibility in patients with cardiovascular risk factors. There are two population groups at risk of ARI: elderly men with arteriosclerotic pathology where the infarction is usually of thrombotic origin, and patients with a history of embolic disease, cardiologic or not, who are more prone to embolic ARI.

In these patients the onset of pain in the flank or renal fossa, elevated LDH and proteinuria and / or microhematuria should arouse suspicion of ARI. Early renal failure is not always present. Definitive diagnosis is made by renal arteriography. It is important to perform complementary studies such as abdominal ultrasound and then computerized tomography (CT) contrast scan and / or renal scintigraphy. Such test are not invasive and allow differential diagnosis.

The treatment of choice is local fibrinolysis, and surgery is relegated to specific cases. Another option in patients with contraindications is anticoagulation.

Case report

A 90 year-old woman attended our emergency department (ED) for abdominal pain of 24 hours evolution. Medical history included: high blood pressure (BP), chronic obstructive pulmonary disease (COPD), atrial fibrillation, sinus node disease, a dual-chamber rate adaptive (DDDR) pacemaker for interatrial block, iatrogenic hypothyroidism due to amiodarone. She was receiving treatment with: acetylsalicylic acid, retard diltiazem, spironolactone, tiotropium bromide, levothyroxine, digoxin, furosemide and omeprazole.

The patient had colic-like upper quadrant abdominal pain of 24 hours evolution, radiating to the back and accompanied by nausea and vomiting without respiratory, cardiac or voiding symptoms. Blood pressure (BP) was 119/68

mmHg, and he had an arrhythmic pulse with a ventricular rate average of 80 x' and left basal lung crackles on auscultation with right basal hypoventilation. The abdomen showed collateral circulation, was tympanic and distended, with right side pain primarily in the upper quadrant, with positive Murphy's sign and hyperperistaltism, and positive right renal succussion. The results of additional tests were: urea 76 mg / dl, creatinine 2.01 mg / dl, sodium 128 mEq / l, GOT 77 U / L, GPT 73 U / L, LDH 2097 U / L, amylase 75 U / L, hematocrit 51.2%, HB 17 g / dl, leukocytes 33 x 109 / L with 85% granulocytes, normal platelets and coagulation, pH 7.4, excess -1.9 base, and ESR 79 mm / h. Urine sediment showed 25/_l leukocytes, negative nitrites, negative red cells and protein 25 mg / dl. ECG showed atrial fibrillation with a pacemaker spike. Abdominal radiography showed dilated bowel loops with distal gas. Abdominal ultrasound showed granulomatous liver calcifications, liver splenic calcifications, acalculous gallbladder, normal pancreas and right kidney and left kidney with a cortical cyst.

The patient was admitted to the department of Internal Medicine with a diagnosis of abdominal pain secondary to a possible intra-abdominal abscess and secondary paralytic ileus. Empirical treatment was initiated with imipenem; contrast-enhanced abdominal CT showed a filling defect of the abdominal aorta, at the ostium of the right renal artery, without opacity. Contrast in the right kidney was only captured in a posterior subcapsular region. The diagnosis was right renal infarction by renal artery occlusion and thrombus in the aorta.

The patient was transferred to the department of vascular surgery where both surgical and thrombolytic treatment were rejected in favour of conservative treatment with low molecular weight heparin of 1mg/kg/12 hours. The patient evolved unfavourably and died four days later.

Discussion

Acute renal infarction is an unusual cause of renal failure, but is associated with high mortality[2]. Clinical suspicion of this entity is important and should be included in the differential diagnosis of abdominal pain in patients predisposed to renal artery thromboembolism[3,4]. It is a great "mimicker" and the initial presumptive diagnosis may be pyelonephritis, urolithiasis, diverticulitis, cholecystitis, back pain, acute abdomen, etc. Hence the importance of the suspicion to avoid late or erroneous diagnosis. The progressive increase of people aged 65 years or more and the high prevalence of cardiovascular disease, suggests that ARI may not be as rare as was thought, as shown by autopsies[5].

The most common cause is embolic, compounded by thrombosis secondary to spasm caused by the impact of the thrombus in the vascular endothelium. The thrombus is often of cardiac origin, most commonly caused by atrial fibrillation secondary to valvular disorders. Other causes include ischemic heart disease, dilated cardiomyopathy, ventricular aneurysm and atrial septal aneurysm. Less frequent causes include polycythemia vera, lupus erythematosus, Behcet's disease, polyarteritis nodosa, associated with primary or secondary antiphospholipid antibodies. Occasionally, they may be iatrogenic (selective embolization[6], placement of stents) or secondary to drugs like cocaine[7].

The clinical picture is characterized by low back or flank pain, sudden, severe, sometimes localized in the upper abdomen or chest, with vegetative symptoms such as nausea, vomiting and diaphoresis. Elevated BP or gross hematuria may be present, but proteinuria is the most common finding (present in more than 70% of cases)[8]. Laboratory tests can show leukocytosis with neutrophilia and elevated LDH, which is the most useful for diagnosis (present in virtually 100% of cases)[9], although nonspecific since this can be found in other situations (myocardial infarction, hemolysis, liver disease).

LDH activity increased during the first 24 hours and persisted up to 10 days. Our patient had atrial fibrillation as an embolic risk factor, compatible but nonspecific clinical symptoms and elevated serum LDH. Chest X-ray and abdominal ultrasound did not identify the disease but allowed differential diagnosis with cholecystitis, obstructive uropathy, etc. Diagnosis is usually by exclusion. Contrast CT scan was performed; this non-invasive technique is the diagnostic tool of choice[8,10], and selective arteriography is the most useful tool for the diagnosis of renal infarction, while also permitting local fibrinolysis.

Currently, fibrinolysis has emerged as a good alternative for treatment[11,12] but its effectiveness is time-dependent (maximum benefit within the first 12 hours). Surgery with myocardial revascularization is indicated for acute renal trauma, in both or the remaining functional kidney, but the risk is far higher than with fibrinolysis. When fibrinolysis is not feasible, anticoagulation is indicated as rescue therapy, or even prophylactic, to prevent further episodes.

Thus clinical suspicion of ARI Is important in all patients with flank pain, elevated LDH and sediment alterations. When clinical suspicion is high, radiological tests do not show evidence of lithiasis, and abdominal ultrasound is normal, additional tests are indicated, including CT scan, for an accurate diagnosis, since any delay here, as in other types of infarction (heart, stroke), will influence prognosis because treatment is time dependent.

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Infarto renal como causa infradiagnosticada de dolor abdominal

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El infarto renal agudo es una causa poco frecuente de dolor abdominal, si bien es una patología que va en aumento (en concordancia con la patología arteriosclerótica) y no en pocas ocasiones está infradiagnosticada. Por ello es importante pensar en ella en todo paciente con dolor abdominal en flanco, aumento de la láctico deshidrogenada (LDH) y alteraciones en el sedimento; si las pruebas radiológicas y la ecografía abdominal son normales, sin imágenes que nos sugieran uropatía obstructiva, la realización de una tomografía computarizada (TC) con contrate (prueba no invasiva) estaría indicada para confirmar el diagnóstico, dado que la efectividad del tratamiento es tiempo dependiente. Presentamos el caso clínico de una paciente con dolor abdominal inespecífico con las alteraciones analíticas anteriormente mencionadas, en la que la TC abdominal fue diagnóstica. No había indicación de tratamiento fibrinolítico, por lo que se inició tratamiento anticoagulante con heparinas de bajo peso molecular (HBPM) a dosis terapéuticas. A pesar de ello la paciente evoluciona desfavorablemente falleciendo días después. [Emergencias 2009;21:117-119]

Palabras clave: Infarto renal agudo. Trombosis renal. Tratamiento fibrinolítico.