Clinical Case

Acute segmental renal infarction with spontaneous resolution: a case report


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Keywords
Segmental renal infarction; Acute renal infarction

Abstract
Acute renal infarction is an entity that is rarely detected in clinical practice. Diagnosis is frequently delayed or missed due to the rareness of the disease and its nonspecific clinical presentation that generally simulates lithiasis. Up to 70% of the patients have a history of elevated thromboembolism risk. Since contrast-enhanced tomography has become the imaging modality of choice for evaluating various acute abdominal conditions, perhaps now, unsuspected renal infarction will be discovered. A 67-year-old woman with a past history of high blood pressure and nonspecific untreated cardiac arrhythmia came to the emergency department complaining of abdominal pain in the right flank. The tomography scan revealed segmental renal infarction of the right kidney affecting the anterior surface and the upper pole. The next day she had selective angioplasty with the possibility of undergoing percutaneous angioplasty to permeabilize the vessel, but instead, spontaneous reperfusion of the occluded segmental artery was observed.

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Palabras Claves
Infarto renal segmentario; Infarto renal agudo

Resumen
El infarto renal agudo es una entidad raramente detectada en la práctica clínica. El diagnóstico es frecuentemente retrasado o se pierde debido a la rareza de la enfermedad y a su presentación clínica poco específica que generalmente simula una litiasis. Hasta un 70% de los...
Introduction

Acute renal infarction is very seldom detected in clinical practice due to the rareness of the disease and its nonspecific clinical presentation\(^1\) that is frequently similar to that of lithiasis.\(^2\) Renal infarction usually occurs in the sixth to eighth decades of life.\(^2\) The majority of patients have a history of high thromboembolism risk \(^1\) and many have had a prior embolic event.\(^3\) There can be a significant loss of kidney function in complete renal infarction if the renal artery is involved, but the occlusion of a segmented artery can compromise the perfusion of one-fifth to one-third of the kidney.\(^4\)

Case presentation

A 67-year-old woman with a 20-year past history of diagnosed and controlled high blood pressure and nonspecific untreated cardiac arrhythmia came to the emergency department complaining of the sudden onset of intense abdominal pain of 3-day progression in the right flank, irradiating to the ipsilateral groin and back and accompanied with nausea and diaphoresis. Physical examination was unremarkable. The laboratory work-up revealed leukocytosis 22,000 with neutrophilia and a lactate dehydrogenase (LDH) value of 1,422.

The pain was controlled in the emergency department and noncontrast and contrast-enhanced computed axial tomography (CAT) scans identified right nephromegaly with an adequate medulla cortex relation; no solid masses, cysts, stones, or dilation were observed (fig. 1). In the angiographic phase, the right renal artery had an occluded area that affected the superior and anterior segmental arteries, causing a perfusion defect of 60% (fig. 2). There was a marked zone of low uptake in the right kidney in the nephrographic phase at the anterior surface and toward the upper pole (figs. 3 and 4). An echocardiogram was then taken that documented auricular fibrillation and a double mitral lesion, mild stenosis, and moderate insufficiency.

The next day the patient was referred for selective angiography with the possibility of percutaneous angioplasty to permeabilize the vessel. A thrombus that partially occluded the vessel lumen was observed in the superior segmental artery, but it did not limit the anterograde flow, and spontaneous reperfusion of the occluded artery was then observed (fig. 5).

Discussion

Acute renal infarction is an entity that is rarely detected in clinical practice. Diagnosis is frequently delayed or missed due to the rareness of the disease and its nonspecific clinical presentation. Since the CT scan has become the modality of choice for evaluating various abdominal conditions acutely, it is possible that renal infarction may now be discovered.
presentation. Previous reports have documented up to double the prevalence of left renal infarction, but Korzets et al. found a similar prevalence in both the left and right kidneys. A study on autopsies reported an incidence of up to 0.48%. Renal infarction usually occurs in the sixth to eighth decades of life.

Up to 70% of the patients have a history of high thromboembolism risk. The main cardiovascular diseases that present with the greatest risk for developing renal infarction are high blood pressure (64%), atrial fibrillation (50%), coronary disease (36%), congestive heart disease (23%), and rheumatic mitral stricture (23%). The majority of the patients have had a prior embolic event, and many of these patients are receiving anticoagulation therapy with a sub-therapeutic INR. This condition can be an important cause of kidney loss, and when not previously diagnosed, can indicate an underlying cardiovascular disease.

In cases of idiopathic renal infarction, the origin of renal embolism remains unknown in the majority of patients with no cardiac pathology. Embolism can more frequently originate from the aortic wall than from the cardiac cavities. A high incidence of hereditary thrombophilia and/or hyperhomocysteinemia could be a contributing factor. Other factors have been reported as possible etiologies; cocaine-induced kidney damage is a well-documented entity, but renal infarction secondary to cocaine use is not.

There can be a significant loss of kidney function in complete renal infarction if the renal artery is involved. However, the clinical impact of segmental infarction on kidney function is not completely understood.

The hypoxia that results from an imbalance between oxygen supply and demand is considered to play an important role in the pathogenesis and progression of kidney disease. Despite the importance of hypoxia, little is known in relation to the consequences of a local reduction of tissular oxygen tension and the extension and operation of adaptive responses in different anatomic zones of the kidney. Despite being an established kidney failure model, complete renal ischemia is of limited usefulness for evaluating the response to regional hypoxia, which is different from uniform hypoxia.

The ligature of a branch of the renal artery compromises the perfusion of one-fifth to one-third of the kidney and induces characteristic changes in morphology with differences in regard to the time and place of ischemia. With respect to time, the morphologic changes can be divided into an early phase (1-6 h), in which the parenchymal cells appear to be viable, an intermediate phase (1-3 days), in which tissue damage is now established, and a late phase (more than 3 days), in which tissue repair and remodeling is obvious. In terms of differences with respect to site, the distinction between 3 zones parallel to the corticomedullary axis of the kidney after one day becomes apparent: 1) tubular necrosis in the center of the infarction, 2) a zone at the edge of the infarction between the area of tubular necrosis and normal tissue, with a variety of cellular alterations, and 3) normal preserved tissue. Presumably, the oxygen level at which the tubular cells are damaged varies throughout the nephron.

Large infarctions create an oxygen gradient that is perpendicular to the corticomedullary axis of the kidney. Different levels of tissue damage are developed through this
gradient, evoking complex cell reactions and repair mechanisms. Whereas marked necrosis occurs rapidly in the center of the infarct, a band of tissue appears at the edge of the supply area of the infarcted artery after one day, in which the cells are the object of sub-lethal hypoxic damage. A comparison between the center of the infarct and the zone of the edge suggests that there is a great tubular regeneration potential after hypoxic damage.4

Up to 61% of the patients have been found to continue with normal kidney function in the follow-up after infarction.3

Ever since contrast-enhanced tomography became the imaging modality of choice for evaluating various acute abdominal conditions, this may be the first time that unsuspected renal infarction can be discovered. Its clinical manifestation is generally severe, persistent flank pain that is medical treatment-refractory, and sometimes simulates renal colic.1 Other presentations also include nausea or vomiting (46%) and fever (27%). All the clinical variations are highly variable and can imitate lithiasis.2 Small infarcts can be asymptomatic and manifest only as fatigue. Positive findings include fever, abdominal pain, and high blood pressure of recent onset. Urinalysis may reveal microscopic findings include fever, abdominal pain, and high blood pressure of recent onset. Urinalysis may reveal microscopic hematuria or proteinuria, but it can also be normal. Increased levels of serum LDH, C-reactive protein, and aminotransferases are common; LDLH is probably the most useful, albeit nonspecific, marker.2 Leukocytosis is observed in 50% of the patients and increased LDH, up to 6.48-fold its upper limit of normal, is consistent in all the patients.2

In patients with renal infarction, angiotomography is generally the initial imaging study, but the definitive diagnosis is made with angiography.2 Renal angiography is diagnostic in 100% of the cases. Other diagnostic techniques include kidney scintigram, which is abnormal in 97% of the cases, intravenous contrast-enhanced tomography, which can be diagnostic in 80% of the cases, and ultrasound, which is positive in only 11%.3 Kidney ultrasound is not sufficiently sensitive and cannot diagnose or exclude acute renal infarction, given that there are no specific changes in the infarcted kidney.4 In the tomography scan, in particular, a renal infarct can be confused with malignant disease.7 Total and segmental infarction can be differentiated clinically through imaging studies or a nuclear renal perfusion scan.2

Radiologic findings in renal infarction depend on the extension, as well as the time of progression, of the infarct. These findings are parallel to the pathophysiologic changes that occur after acute arterial occlusion. After one h of occlusion of an arcuate or interlobular artery, a triangular hyperemeric area is seen with its apex pointing to the medulla and its base parallel to the subcapsular region. After 7 days of occlusion, the infarct begins to shrink. At 28 days, there is a pronounced depression in its surface. The central necrotized area collapses to the degree that the infarct is reduced, whereas regeneration and organization take place at the periphery. Tubular calcification begins from 3 days after infarction and lasts throughout the following week. It is more noticeable in the tubules near the surface of the kidney.3

Contrast tomography is the standard noninvasive technique for visualizing a renal infarct. The classic finding is a wedge-shaped zone of reduced peripheral density, without enhancement. Tomographic findings have been described by Suzer et al.4 in a series of 37 cases. The most common finding was a hypo-attenuated area with an associated mass effect in 32% of the cases, followed by the cortical rim sign in 19%. The cortical rim sign in a nephrogram is an opacification of a border of functional nephrons, irrigated by capsular collateral vessels, that surrounds an otherwise nonfunctioning kidney.5

Treatment guidelines have not yet been established. However, early anticoagulant therapy seems to be beneficial; thrombolytic therapy is better if it is used early (90-180 min from symptom onset), whereas ischemic renal tissue benefits from reperfusion. A tendency toward kidney function improvement has been seen in the early diagnosis groups, as opposed to those of late diagnosis. Conservative treatment has been more favorable than surgery, which is reserved for patients with solitary kidney or bilateral thrombuses.2

In relation to renal function prognosis, serum creatinine and urea levels have been analyzed in patients with renal infarction, and they have been found to be normal, or not higher than 25%, at the long-term follow-up in 88% of the patients. It is therefore recommended that long-term follow-up of these patients include functional studies, rather than serum creatinine monitoring.10 The incidence of arterial hypertension following infarction has also been analyzed. In a study of 55 cases of patients with renal infarction, 25 had a history of hypertension of long progression and 30 presented with acute hypertension. In the follow-up, the patients with acute hypertension at the time of renal infarction were found to have a greater tendency to normalize their baseline tension figures than the patients with long progression hypertension. In the long-term follow-up, the patients did not have significant changes in the glomerular filtration rate. The authors concluded that a patient with renal infarction presenting with acute hypertension has a favorable prognosis in relation to long-term blood pressure figures.11

Our patient’s clinical presentation imitated right renal colic, but due to her history of cardiac arrhythmia and a marked increase in LDH, an abdominal tomography scan was ordered as the study of choice for lithiasis diagnosis, ordering the contrast-enhanced phase for corroborating clinical suspicion. The laboratory and imaging study findings corresponded to those described in the literature. The patient did not receive thrombolytic therapy, but rather the decision was made to carry out percutaneous reperfusion because the fully equipped hemodynamic unit was available. We found no cases of spontaneous reperfusion in the literature and we suggest following the international recommendations and initiating thrombolytic therapy if no hemodynamic unit is available, as was the case with our patient.

Conclusions

When a patient with an increased risk for thromboembolism presents with flank pain unexplained by other causes, renal infarction should be suspected, and under these circumstances, hematuria, leukocytosis, and elevated serum LDH strongly support the diagnosis.1
Ethical responsibilities

Protection of persons and animals. The authors declare that no experiments were performed on humans or animals for this study.

Data confidentiality. The authors declare that no patient data appear in this article.

Right to privacy and informed consent. The authors declare that no patient data appear in this article.

Financial disclosure

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Conflict of interest

The authors declare that there is no conflict of interest.

References


