Acute Renal Infarction. A Case Report

N. Laraba
Internal Medicine Department, University Hospital of Bab El Oued, University of Algiers, Algeria

N. Bellik
Nephrology Department, University Hospital of Bejaia, University of Bejaia, Algeria

M. A. Habouchi
Radiology Department, University Hospital of Bab El Oued, University of Algiers, Algeria

K. Belgine
Gastroenterology Department, University Hospital of Mustapha, University of Algiers, Algeria

K. Abbaci-Deghor
Internal Medicine Department, University Hospital of Bab El Oued, University of Algiers, Algeria

Abstract
Renal infarction is a rare disease often overlooked due to its very unspecific clinical presentation. We report the particular case of an idiopathic bilateral acute renal infarction occurring in a young adult male. The diagnostic delay in our patient is significant: ten days. However, a Doppler ultrasound allows screening. The diagnosis of certainty is made by the angioCT scan: essential examination to make the lesional balance sheet and to evaluate the prognosis on the renal function. Anticoagulant treatment is the treatment of choice provided it is instituted early.

Introduction
Renal infarction is a rare condition often overlooked due to its misleading clinical presentation. The pain associated with acute renal infarction is nonspecific and can be attributed to other pathologies affecting the digestive organs, spine, or even cardiac issues [1]. Its incidence, as assessed in emergency department admissions in a study conducted in the United States, ranges from 0.004% to 0.007% [2]. However, the incidence evaluated in autopsy series reaches up to 1.4% [3-4]. This discrepancy between the two figures underscores the diagnostic challenge. Delayed diagnosis can adversely affect prognosis by precipitating renal function decline, particularly in cases of embolic etiology [2]. In the latter case, early initiation of anticoagulant therapy is crucial. Apart from embolic origin, there are numerous etiologies of acute renal infarction. Autoimmune diseases such as polyarteritis nodosa, systemic lupus erythematosus, Behçet’s disease, or any other type of vasculitis can cause renal infarction. Identifying the underlying cause directly impacts therapeutic management. Herein, we report a specific case of bilateral idiopathic acute renal infarction occurring in a young adult male.

Case Presentation
Mr. M. T., aged 48, with no known cardiovascular risk factors or thromboembolic history, presents with a picture of refractory renal colic despite prescribed anti-inflammatory treatments. On examination, ten days after the onset of symptoms, the patient is in fair general condition, afebrile, and hemodynamically stable. He complains of unbearable left flank abdominal pain without parietal defense. He reports food vomiting at the peak of pain and longstanding dysuria. The rest of the examination is unremarkable. Biologically, there is proteinuria at 300mg/24h with leukocyturia. No hematuria is observed. There is an inflammatory syndrome with an elevation of C-reactive protein to 38mg/l, and fibrinogen levels to 5g/l. The complement C4 fraction is 0.487 g/l. Liver function tests show moderate cytolysis without cholestasis. Lactate dehydrogenase (LDH) levels are elevated at 300 IU/l, which is two and a half times the normal range. Renal function is normal. Estimated glomerular filtration rate using the Cockcroft and Gault formula is within normal limits. Urine culture and sensitivity are unremarkable. Blood electrolytes, complete blood count, and lipid profile are normal. Abdominopelvic ultrasound shows
no abnormalities, with no dilatation of the urinary tract and preserved renal dimensions. Abdominopelvic computed tomography, prompted by persistent pain, reveals bilateral renal infarction. A complementary angioscannography (see Figure 1) reveals thrombosis of the lower segmental branch of the left renal artery associated with bilateral renal infarcts.

![Abdominopelvic Angio-CT Scan Revealed Thrombosis of the Lower Segmental Branch of the Left Renal Artery Associated with Bilateral Renal Infarcts, Particularly in the Left Lower Pole](image)

Figure 1 (a, b, c): Abdominopelvic Angio-CT Scan Revealed Thrombosis of the Lower Segmental Branch of the Left Renal Artery Associated with Bilateral Renal Infarcts, Particularly in the Left Lower Pole

Treatment with low molecular weight heparin enoxaparin at therapeutic dose was initiated with early switch to oral anticoagulation such as Vitamin K antagonist. The patient underwent exploratory investigations to identify the etiology. Toxic causes were ruled out. Both ECG and 24-hour Holter monitoring showed no rhythm disturbances. Transthoracic and transesophageal echocardiography revealed no notable abnormalities, and no intracavitary thrombus was detected. An autoimmune workup yielded normal results: antinuclear antibodies (ANA), anti-neutrophil cytoplasmic antibodies (ANCA), anti-endothelial cell antibodies. Antiphospholipid antibodies including lupus anticoagulant, anti-β2GP1, and anti-cardiolipin antibodies were negative. Plasma homocysteine levels were normal. Other thrombophilia markers such as Antithrombin III, protein C, and protein S were also negative. There was no mutation detected in factor V Leiden (resistance to activated protein C) or factor II. A myeloproliferative syndrome was ruled out with a negative test for the V617F JAK2 gene mutation. Negative results from flow cytometry excluded
paroxysmal nocturnal hemoglobinuria. The diagnosis of bilateral idiopathic renal infarctions was established. Following treatment, the patient's condition improved with resolution of lumbosacral pain and complete regression of the inflammatory syndrome. However, sequelae included systolic hypertension with a systolic blood pressure of 170 mmHg. Treatment with calcium channel blockers was initiated for hypertension management. Renal function remained satisfactory. A renal scintigraphy with DTPA-99mTc performed eight days after antithrombotic treatment showed both kidneys being adequately perfused with functional asymmetry. Relative renal function of the right kidney was 61%, while that of the left kidney was 39%. Bilateral parenchymal defects, more pronounced on the left, were observed as sequelae.

Discussion
Renal infarction is a rare condition, often challenging to diagnose due to the absence of specific symptoms. In our patient, renal infarction presented as left-sided renal colic, while the right-sided infarction, albeit less extensive, remained completely asymptomatic. Clinical manifestations of renal infarction vary widely. Commonly reported symptoms include pain, similar to that observed in renal colic, fever, nausea, and vomiting. New-onset hypertension or recent blood pressure imbalance may also be indicative of renal infarction. Microscopic or macroscopic hematuria may be present biologically, although it was absent in our patient, possibly due to the timing of the examination (ten days after symptom onset). Additionally, albuminuria or leukocytosis with elevated lactate dehydrogenase (LDH) levels, as observed in our patient, are frequently reported in the literature. Elevated liver enzymes such as aspartate transaminase (AST) and alanine transaminase (ALT) may lead to misdiagnosis as hepatic pathology, especially when right-sided pain is present.

Ultrasound examination coupled with color or power Doppler imaging may contribute to diagnosis by revealing absent flow in the affected kidney. However, Doppler imaging was not performed in our patient. Contrast-enhanced ultrasound is emerging as a diagnostic modality in this indication. Contrast-enhanced computed tomography (CT) is crucial for diagnosis, demonstrating vascular abnormalities such as decreased or absent enhancement of the ischemic renal parenchyma during contrast injection. Definitive diagnosis is typically established through renal arteriography or CT angiography, allowing for exclusion of arterial dysplasia, aneurysms, or arteriovenous malformations.

Renal infarction can result from various causes, with the most common being embolic origin. This involves occlusion of a renal artery by a thrombus, typically of cardiac origin due to conditions such as atrial fibrillation, myocardial infarction, mitral stenosis, atrial myxoma, or infective endocarditis. Thrombus migration from an atherosclerotic plaque or an aneurysm is also possible. In-situ thrombosis leading to renal artery occlusion can occur in conditions such as polyarteritis nodosa, thromboangiitis obliterans, thrombotic thrombocytopenic purpura, or renal artery dissection or aneurysm. Genetic or acquired thrombophilic traits, myeloproliferative syndromes, or solid cancers can also cause renal infarction. Additionally, substances like cannabis, ecstasy, and other drugs can induce renal artery thrombosis. The diagnosis of idiopathic renal infarction can only be made after ruling out all the aforementioned etiologies, as in the case of our patient.

The prognosis of renal function depends on the prompt initiation of anticoagulant therapy and the timeliness of diagnosis. For patients with unilateral involvement, systemic anticoagulation with heparin or fibrinolysis, or percutaneous catheter thromboembolectomy may be the treatment of choice. Surgical embolectomy is traditionally preferred for bilateral or solitary kidney involvement, although it carries a high operative mortality rate. There is currently no consensus on the therapeutic approach to renal infarction.

Conclusion
Acute renal infarction is a rare condition that should be systematically considered in cases of renal colic resistant to treatment, especially when LDH levels are elevated. Its etiologies are diverse, and anticoagulant therapy is the treatment of choice provided it is initiated promptly.

References