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Bilateral Renal Infarction Revealing Primary Antiphospholipid Syndrome: A Case Report and General Review of Literature

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Abstract

Introduction: Renal infarction is a rare, nonspecific condition that is difficult to diagnose. We report a case of bilateral renal infarction presenting with acute low back pain that revealed primary antiphospholipid syndrome. **Case Presentation:** A 41-year-old female patient presented to the emergency department with sudden-onset bilateral low back pain radiating to the flanks, without fever, accompanied by chills and vomiting. Her medical history included cryoablation for *Bouveret's* disease and current smoking. Laboratory results showed serum creatinine at 93 µmol/l, hemoglobin at 11.5 g/dl, leukocytosis at 22 G/l, platelets at 450 G/l, C-reactive protein at 180 mg/l, LDH at 682 IU/l, and a negative HIV serology test. The CT scan revealed hypodense parenchymal foci in the kidneys (nephritis) and foci of necrosis (absence of enhancement after contrast injection). A diagnosis of acute pyelonephritis was suspected, and the patient was treated with ceftriaxone. Given the negative blood culture and urine culture on day 6, the diagnosis was reoriented toward a bilateral renal infarction in the process of developing. Transthoracic echocardiography and Holter electrocardiogram were normal. The antiprothrombinase-type lupus anticoagulant was positive. The remainder of the immunological workup was normal. Management consisted of administering tinzaparin sodium for 3

months, followed by warfarin, for primary antiphospholipid syndrome. The course of the disease was favorable, marked by resolution of the pain and inflammation. **Conclusion:** Renal infarction is a rare condition with a polymorphic clinical presentation. It should be considered in any case of acute low back pain that is more or less associated with elevated LDH levels. Even in the absence of a suggestive clinical picture, antiphospholipid syndrome should be investigated.

Keywords

Renal Infarction, Low Back Pain, Acute Pyelonephritis, Antiphospholipid Syndrome

1. Introduction

Renal infarction is an acute vascular event leading to impaired renal perfusion, which causes ischemia and tissue necrosis in a more or less extensive area of the kidney [1]. It is a rare, nonspecific condition that is diagnostically complex even in its typical clinical form (lumbosacral and abdominal pain in 97% of cases), as it mimics conditions such as urinary lithiasis and acute pyelonephritis. Although the timing of diagnosis and treatment determines the reversibility of renal lesions, renal infarction remains a difficult diagnosis, often made late or even retrospectively [2].

In the absence of prospective multicenter studies, it remains difficult to accurately and reliably assess the epidemiology of this condition. In a French study based on a database of patients followed for hypertension over 17 years, the annual incidence was 0.018% and the prevalence was 0.3% in this specific population [3].

Various mechanisms (embolic, thrombotic, dissection, etc.) of renal infarction have been identified, reflecting the multiplicity of underlying causes and factors. However, despite a comprehensive evaluation, the exact cause of renal infarction may remain unknown [4] [5].

In this case report, we describe a rare case of renal infarction presenting with acute low back pain that revealed primary antiphospholipid syndrome.

2. Case Presentation

A 41-year-old female patient presented to the emergency department with sudden bilateral low back pain radiating to the flanks, without fever, accompanied by chills and vomiting. There was no history of urinary symptoms, nor had she taken any antibiotics and/or nonsteroidal anti-inflammatory drugs. Her medical history included cryoablation for *Bouveret's* disease and current smoking. The patient had never been pregnant, was not using contraception, and had no known history of thromboembolic disease. There was no known family history of systemic disease.

Physical examination revealed a preserved general condition, blood pressure of

133/73 mmHg, a heart rate of 83 beats per minute, and a temperature of 36.5˚C. The abdomen was distended, depressible, and painless on palpation. The urine test strip was positive for hematuria (2 crosses) and proteinuria (1 cross). Laboratory results showed serum creatinine at 93 µmol/L, hemoglobin at 11.5 g/dL, leukocytosis at 22 G/L, platelets at 450 G/L, C-reactive protein at 180 mg/L, AST at 39 IU/L, ALT at 45 IU/L, GGT at 259 IU/L, LDH at 682 IU/L, and a negative HIV serology test (Table 1).

Axial CT scans with contrast injection during the arterial phase showed bilateral renal corticomedullary triangular hypodense areas that did not enhance after contrast injection, with infiltration of the renal fat (Figure 1).

A diagnosis of acute pyelonephritis was considered, and the patient was treated with ceftriaxone.

Given the negative blood culture and urine culture on Day 6, the diagnosis was reoriented toward a bilateral renal infarction in the process of developing. The antiprothrombinase-type systemic lupus anticoagulant was positive according to the diluted Russell's viper venom clotting time test. Repeated positivity over a 12-week period for anti-cardiolipin IgG and anti-beta-2-glycoprotein 1 IgG antibodies supported the diagnosis of antiphospholipid syndrome (APS). The rest of the immunological workup was otherwise normal (Table 2).

Transthoracic echocardiography and Holter electrocardiogram were normal. Doppler ultrasound of the renal arteries and lower extremities showed no abnormalities.

A follow-up renal CT scan on Day 20 showed bilateral hypodense areas in the renal parenchyma of unchanged extent, with no other associated abnormalities.

Management consisted of administration of tinzaparin sodium for 3 months, followed by warfarin for 18 months, targeting an INR between 2 and 3. The course

Table 1. Patient's laboratory data.

Reports	Data (Reference values)	Results
Renal and Urinary	Serum creatinine (53 - 120 µmol/L)	93
	Urinary sediment	Red Blood Cells++, White Blood Cells+
	24-hour proteinuria (<150 mg/24 H)	0,10
	Urine culture	Negative
Hematology, Inflammation, and Biochemistry	Leukocytes (4 - 10 G/L)	22
	Hemoglobin (13 - 17 g/dL)	11,5
	Platelets (150 - 400 g/L)	450
	CRP (<6 mg/L)	**180**
	LDH (90 - 320 IU/L)	**682**
	AST (8 - 30 UI/L)	39
	ALT (8 - 35 UI/L)	45
	HIV serology test	Negative

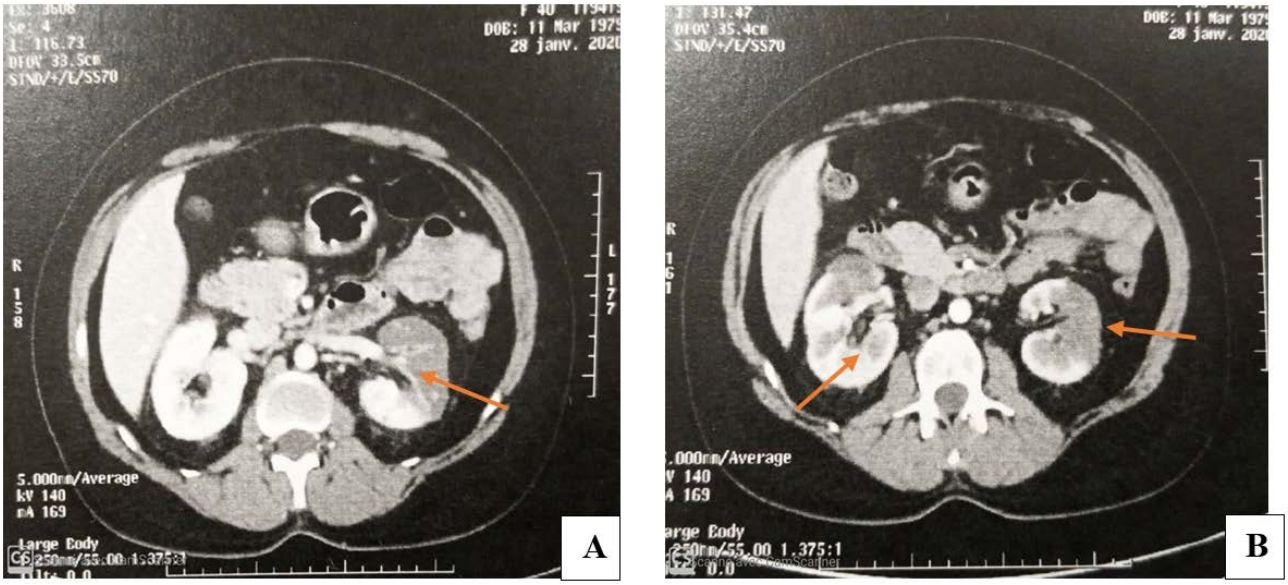

Figure 1. (A) and (B) Axial CT scans with contrast injection at the arterial phase showing bilateral renal corticomedullary triangular hypodense areas that do not enhance after contrast injection, corresponding to foci of renal infarction.

Table 2. Patient's laboratory data for autoimmunity and others.

Autoimmunity and others (*Reference value, dosage methods*)	Week 1	Week 6	Week 12
Lupus anticoagulant (*Coagulation test using diluted Russell's viper venon*)	**positive**	**positive**	**positive**
Anti-cardiolipin IgG **(<40 *U/mL, ELISA*)**	**99**	**83**	**101**
anti-beta-2-glycoprotein IgG **(<20 *U/mL, ELISA*)**	**128**	**134**	**117**
Antinuclear factor (<40 *AU/ml, IFI*)	37	19	26
Anti-native DNA antibody (<30 *IU/mL, ELISA*)	23	-	-
Antithrombin III (80% - 120%)	97	-	-
Proteins C and S (60% - 140%)	113	-	-
Anti-nucleosome Ab (< 20 U/mL)	7		
Ac Scl70 (< 0.9 U/mL)	0.4		
MPO-ANCA (< 20 AU)	14.2	-	13,03
PR3-ANCA (< 20 U/mL)	9.1	-	10.2
Ac-Anti MBG (<10 U/mL)	3.4		
Supplements (C3: 0.7 - 1.7 g/L; C4: 0.1 - 0.4 g/L)	Normal	-	Normal

of the disease was favorable, marked by resolution of the pain and inflammation and the disappearance of the circulating anticoagulant after approximately 2 years of follow-up.

3. Discussion

3.1. Epidemiology

Estimating the true incidence of renal infarction is difficult in clinical practice, as many cases of renal infarction are diagnosed late or incorrectly; as a result, post-

mortem studies may provide more accurate data. Postmortem studies suggest that the incidence of renal infarction is around 14 per 1000, or 1.4% [6] [7]. According to several studies in the literature, the average age of patients with renal infarction is estimated at approximately 63 years, with no significant difference between the sexes [5] [8]. In a French multicenter retrospective study, the average age at diagnosis was 52.9 ± 16.6 years. Risk factors for renal infarction include atrial fibrillation, hypertension, diabetes, and a history of embolic infarction [5]. Aside from active smoking, there were no other notable risk factors for renal infarction in this patient.

3.2. Physiopathogenesis

The exact pathophysiology of renal infarction remains unclear. However, several research studies have been conducted to investigate the impact of hypoxia on renal cellular functions and the adaptive responses induced by renal infarction. Thrombotic mechanisms involve endothelial injury, which triggers the formation of a thrombus in situ. Another ischemic mechanism occurs when emboli originating in the heart—in cases of atrial fibrillation—become dislodged and eventually obstruct the renal artery [9].

A decrease in local renal filtration capacity due to loss of nephrons in the infarcted area may be observed, which can lead to acute kidney injury depending on the extent of the lesion. In the long term, segmental fibrotic scarring carries a risk of secondary renal hypertension (activation of the renin-angiotensin-aldosterone system in healthy tissue in response to hypoperfusion) and chronic kidney disease if the lesions are extensive or recurrent [10].

3.3. Diagnosis

The clinical symptoms of renal infarction are nonspecific. The suggestive clinical presentation includes lower back and abdominal pain, fever, nausea, hematuria, and, occasionally, hypertension [11]. Our patient, who had normal blood pressure, presented with flank pain, chills, vomiting, and microscopic hematuria—all of which are common but nonspecific signs of renal infarction.

Biochemically, elevated LDH levels and moderate renal insufficiency are often observed. Renal involvement during a renal infarction is multifactorial. No link has been established between renal involvement and the extent of the infarction. Some authors report a slight increase in creatinine in cases of unilateral renal infarction [10] [11]. In our case, LDH was very elevated (which may reflect this bilateral renal necrosis), with preserved renal function.

More sporadically, the presence of a systemic inflammatory response syndrome remains possible, although it is rarely reported in certain cases. In a French multicenter study, the median CRP level among patients was 60 mg/L and was higher in groups where the infarction was of cardiac origin or related to thrombophilia [5]. Given the significance of the inflammatory markers associated with low back pain and chills, the initial diagnosis for our patient strongly favored acute pyelo-

nephritis. It was only after the negative results of the bacteriological cultures on day 6 that the diagnosis shifted toward a renal infarction.

The diagnosis relies on a high index of suspicion and appropriate imaging—in this case, a renal CT angiogram with contrast injection, as described here, or a CT scan—which very clearly demonstrated an extensive absence of bilateral renal cortical enhancement. Given its low prevalence, the absence of specific clinical signs, and significant variability in symptoms (particularly with acute pyelonephritis), the diagnosis of renal infarction is often difficult to establish [12]. This could partly explain the diagnostic delay: 5.4 ± 6.5 days between the onset of symptoms and radiological confirmation, as reported in the study by *Bourgault et al.* [5].

The causes of renal infarcts are numerous and varied. They include renal artery anomalies, embologenic heart diseases, vasculitides, and, very rarely, thrombophilic conditions that may be hereditary or acquired in the context of antiphospholipid syndrome [13].

Diagnosis of APS in this context requires confirmed arterial thrombosis as well as the persistent presence of lupus anticoagulant on two separate occasions [14]. Our patient met these criteria: she had confirmed arterial thrombosis (bilateral renal infarction) associated with repeated positivity over 12 weeks for the lupus anticoagulant (as determined by the diluted Russell's viper venom clotting time test), IgG anticardiolipin antibodies, and IgG anti-beta-2-glycoprotein 1 antibodies, thereby confirming the diagnosis of primary antiphospholipid syndrome. The search for sources of embolism in the heart and peripheral circulation proved negative in this patient, given the normal results of the transthoracic echocardiogram, Holter electrocardiogram, and Doppler ultrasound of the renal arteries and lower extremities. Other potentially confounding conditions, such as ANCA-associated vasculitis, renal infarction associated with systemic lupus erythematosus, and polyarteritis nodosa [15]-[17], were unlikely in our patient, especially since there were no extrarenal signs, and both renal function and the rest of the immunological workup were unremarkable.

4. Treatment and Outcome

In general, the management of renal infarction depends on the underlying cause, the extent of the lesions, the time to diagnosis, and the patient's comorbidities. There is no absolute consensus on the treatment of renal infarction in primary acute kidney injury, and in published case series, treatment is predominantly conservative and medical. The gold standard of care is systemic anticoagulation with heparin, followed by a vitamin K antagonist and/or antiplatelet agents [18]. In our patient, the administration of tinzaparin followed by warfarin contributed to achieving good outcomes.

Although the duration of anticoagulant therapy following a renal infarction is not standardized in a number of cases, it seems clear that long-term anticoagulant therapy should be maintained in cases of renal infarction occurring in the context of hereditary or acquired thrombophilia [13] [14] [19] [20]. The patient was on

warfarin for 18 months, and no thromboembolic recurrence was noted.

In the literature, many patients with unilateral renal infarction maintained normal overall renal function during follow-up, in contrast to some cases of renal failure associated with bilateral involvement [10]. Overall renal function was preserved in our patient throughout the follow-up period. In Yang's study, chronic kidney disease developed in 27.4% of patients during follow-up, and the predictive risk factors for acute kidney injury were diabetes and elevated CRP levels.

Early screening and treatment appear essential for preserving renal function. In addition to proper adjustment of anticoagulant therapy, long-term management also requires attention to cardiovascular risk factors and avoidance of additional prothrombotic factors (e.g., smoking, estrogen-containing contraceptives), which is an important consideration [21].

This clinical case highlights the complexity involved in diagnosing renal infarction in the emergency setting, given the polymorphic nature of the clinical presentation. Although renal biopsy is an essential tool for confirming the diagnosis, it does not appear to be indispensable when clinical and laboratory findings are consistent, as demonstrated in this case.

5. Conclusion

Renal infarction is a rare condition with a polymorphic clinical presentation. It should be considered in any case of acute low back pain, with or without elevated LDH levels. While diagnosis remains difficult in most cases, determining the underlying cause remains a significant challenge, and thrombophilia should be investigated in all cases of renal infarction.

Consent

Informed consent was obtained from the patient.

Author Contributions

ASF, CB diagnosed, treated and clinically monitored the patient. ASF conducted the literature search and wrote the manuscript. CA, SM, CM, SOYD, HY, and FS reviewed the manuscript. SC reviewed the manuscript images.

Conflicts of Interest

The authors declare that they have no competing interests.

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