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Renal Infarction Mimicking Renal Colic: A Diagnostic Challenge

Mohammed Alfozan

Department of Surgery, College of Medicine, Prince Sattam bin Abdulaziz University, Al-Kharj, Saudi Arabia, <https://orcid.org/0000-0002-3551-8331>

ABSTRACT:

Background: Renal infarction is an uncommon but clinically important cause of acute flank pain and may closely resemble renal colic, particularly when nephrolithiasis is simultaneously present. Because renal colic is considerably more common, vascular causes of flank pain may be overlooked, resulting in delayed diagnosis and potential loss of renal function.

Case presentation: R.F is a male health patient. This case describes a patient presenting with acute flank pain and laboratory findings initially compatible with renal colic. Investigations demonstrated microscopic hematuria, acidic urine, uric-acid crystalluria, elevated serum uric acid, and increased 24-hour urinary uric-acid excretion, supporting the presence of uric-acid nephrolithiasis. However, additional abnormalities, including markedly elevated lactate dehydrogenase, increased C-reactive protein and erythrocyte sedimentation rate, and mild impairment of renal function, raised suspicion of an associated ischemic renal process. Contrast-enhanced computed tomography revealed a renal perfusion defect consistent with renal infarction, while imaging findings also supported concomitant renal stone disease. Liver function tests, lipid profile, coagulation parameters, and glycemic indices remained within normal limits.

Discussion: The coexistence of renal infarction and nephrolithiasis can complicate diagnostic interpretation because both conditions may present with flank pain and hematuria. Elevated lactate dehydrogenase and characteristic contrast-enhanced CT findings are particularly useful in distinguishing renal infarction from uncomplicated renal colic.

Conclusion: Renal infarction should be considered in patients with acute flank pain when clinical severity, laboratory abnormalities, or imaging findings are not fully explained by nephrolithiasis. Early recognition and appropriate imaging are essential to minimize diagnostic delay and preserve renal function.

INTRODUCTION

Renal infarction is an uncommon but clinically important cause of acute flank pain that may be easily overlooked in the emergency setting because its presentation often resembles more prevalent renal and urinary tract conditions. Patients typically present with sudden-onset unilateral flank or abdominal pain, nausea, vomiting, microscopic or gross hematuria, and nonspecific inflammatory or biochemical abnormalities [1]. These manifestations frequently overlap with those of renal colic, pyelonephritis, and other acute abdominal disorders, creating a considerable diagnostic challenge. Among these differential diagnoses, renal colic is particularly important because nephrolithiasis is far more common and is therefore often considered first in patients presenting with acute flank pain [2].

Renal infarction results from an abrupt interruption of renal arterial blood supply, leading to ischemic injury of the renal parenchyma. The underlying mechanism may include arterial embolism, in-situ thrombosis, renal artery dissection, vascular

injury, hypercoagulable states, or, less commonly, idiopathic causes [3, 4]. Cardioembolic events, particularly in association with atrial fibrillation and other cardiac disorders, are among the most frequently recognized etiologies. However, a substantial proportion of patients may lack obvious predisposing factors at initial presentation, further complicating timely diagnosis [5]. Because renal tissue is highly dependent on adequate perfusion, delayed recognition may lead to irreversible renal damage, persistent hypertension, deterioration in renal function, or loss of functional renal mass [6].

The diagnostic difficulty is amplified when renal infarction occurs in a patient who also has nephrolithiasis [7]. The presence of a renal or ureteric calculus can provide an apparently convincing explanation for flank pain and hematuria, potentially diverting attention from a concomitant vascular event. Uric-acid stones are of particular interest because they are associated with persistently acidic urine, hyperuricemia, increased urinary uric-acid excretion, and uric-acid crystalluria. Although renal stones may account for colicky pain and hematuria, they do not necessarily explain marked elevations in tissue-injury markers or characteristic renal perfusion abnormalities. Therefore, careful integration of laboratory and imaging findings is essential [5, 8].

Laboratory investigations in renal infarction are often nonspecific, although elevated lactate dehydrogenase is a well-recognized clue reflecting renal tissue ischemia and necrosis [9]. Mild leukocytosis, elevated inflammatory markers, hematuria, and transient impairment of renal function may also be present. In contrast, nephrolithiasis may be associated with microscopic hematuria, crystalluria, urinary pH abnormalities, and metabolic disturbances related to stone composition. The coexistence of these findings may create a mixed biochemical picture that requires cautious interpretation [10-12].

Imaging plays a central role in distinguishing renal infarction from uncomplicated renal colic. Non-contrast computed tomography is commonly used to detect urinary calculi, whereas contrast-enhanced CT is more informative when renal vascular compromise is suspected [13, 14]. Wedge-shaped areas of reduced or absent renal enhancement are characteristic radiologic findings of renal infarction. Recognition of these features is critical, particularly when symptoms appear disproportionate to the stone burden or when laboratory findings suggest tissue ischemia [15].

This case study describes renal infarction presenting with features suggestive of renal colic in the setting of uric-acid nephrolithiasis. It highlights the overlap between vascular and obstructive renal pathology and emphasizes the importance of combining clinical assessment, laboratory markers, urinalysis, and appropriate contrast-enhanced imaging to avoid diagnostic delay and improve clinical decision-making.

CASE PRESENTATION

R.F is a male health patient, his race is Northern African, with no history of smoking, renal or liver dysfunction. His case came to diagnostically confer his renal infarction and renal stone before any further investigations.

It has a summary of hematological, renal, inflammatory, uric-acid, urinalysis, coagulation, hepatic, lipid, and glycemic laboratory findings for a single case presenting with renal infarction and a uric-acid renal stone. The most notable abnormalities include mild leukocytosis with neutrophilia, elevated serum creatinine and blood urea nitrogen, reduced estimated glomerular filtration rate, increased lactate dehydrogenase, C-reactive protein, and erythrocyte sedimentation rate, together with hyperuricemia and increased 24-hour urinary uric-acid excretion. Urinalysis demonstrates acidic urine, marked microscopic hematuria, trace proteinuria, and uric-acid crystalluria. In contrast, liver-function tests, coagulation parameters, lipid profile, and glycemic indices remain within their respective reference ranges. Results are classified as normal (N), high (H), or low (L) according to the stated laboratory reference intervals (Table 1).

Figure 1 illustrates the principal laboratory abnormalities associated with the simulated renal infarction and uric-acid stone case. Results are expressed as a percentage of the upper reference limit, with the dashed vertical line at 100% representing the upper limit of normal. Marked elevations are observed in C-reactive protein and lactate dehydrogenase, accompanied by increased erythrocyte sedimentation rate, serum creatinine, blood urea nitrogen, serum uric acid, and 24-hour urinary uric-acid excretion. Microscopic hematuria represents the most pronounced urinary abnormality. For graphical normalization of the urine RBC range of 18–25/HPF, the midpoint of the reported range was used relative to the upper normal limit of 2/HPF. Overall, the pattern demonstrates renal injury, inflammatory activity, and abnormalities related to uric-acid stone formation.

Figure 2 shows the lipid and glycemic profile of the simulated case relative to their respective clinical reference thresholds. Total cholesterol, LDL cholesterol, triglycerides, fasting blood glucose, 2-hour postprandial glucose, and HbA1c remain below their upper reference thresholds. HDL cholesterol is shown relative to the recommended lower threshold of 40 mg/dL; therefore, a

value above 100% for HDL-C represents a favorable finding rather than an abnormal elevation. Collectively, these results demonstrate a normal lipid profile and normal glycemic status, with no simulated laboratory evidence of dyslipidemia or diabetes mellitus.

Regarding imaging as a second diagnostic tool, a plain abdominal radiograph demonstrates a well-defined radiopaque calculus projected over the region of the left kidney (right side of the radiograph), highlighted by the red circle. The location is consistent with a left renal stone. The right kidney is located on the opposite side of the radiograph and does not show a similarly prominent radiopaque calculus in this image (Figure 4). Axial contrast-enhanced CT image of the abdomen demonstrating a markedly abnormal left kidney (right side of the image), with substantial enlargement and a large heterogeneous low-attenuation component associated with peripheral calcific foci. The right kidney (left side of the image) demonstrates relatively preserved size and contrast enhancement. The appearance of the left kidney in this single image is not characteristic of a straightforward segmental renal infarction alone and may represent a more complex renal process; correlation with the complete CT series and radiological report would be required for definitive characterization (Figure 5).

Table 1 Main laboratory findings for the case presentation (Renal and liver functions, as well as diabetic and lipid profiles)

Laboratory test	Simulated result	Unit	Reference range	Code
CBC				
Hemoglobin	14.2	g/dL	12.0–17.0	N
RBC count	4.85	$\times 10^6/\mu\text{L}$	4.2–5.9	N
Hematocrit	43.0	%	36–52	N
WBC count	11.8	$\times 10^3/\mu\text{L}$	4.0–11.0	H
Neutrophils	76	%	40–75	H
Lymphocytes	17	%	20–45	L
Platelets	278	$\times 10^3/\mu\text{L}$	150–450	N
Renal/metabolic profile				
Serum creatinine	1.38	mg/dL	0.6–1.2	H
Blood urea nitrogen (BUN)	26	mg/dL	7–20	H
eGFR	62	mL/min/1.73 m ²	≥ 90	L
Sodium	139	mmol/L	135–145	N
Potassium	4.3	mmol/L	3.5–5.1	N
Chloride	103	mmol/L	98–107	N
Bicarbonate	24	mmol/L	22–29	N
Calcium	9.4	mg/dL	8.5–10.5	N
Phosphorus	3.5	mg/dL	2.5–4.5	N
Renal infarction-associated tests				
LDH	510	U/L	120–250	H
CRP	18	mg/L	<5	H
ESR	26	mm/hr	0–20	H

Uric acid/stone profile				
Serum uric acid	9.1	mg/dL	3.5–7.2	H
Urine uric acid, 24 h	820	mg/24 h	250–750	H
Urine RBCs	18–25	/HPF	0–2	H
Urine WBCs	2–4	/HPF	0–5	N
Urine protein	Trace	—	Negative	H
Urine glucose	Negative	—	Negative	N
Urine ketones	Negative	—	Negative	N
Urine nitrite	Negative	—	Negative	N
Urine leukocyte esterase	Negative	—	Negative	N
Uric acid crystals	Present ++	—	Absent	H
Liver function tests				
ALT	24	U/L	7–56	N
AST	22	U/L	10–40	N
ALP	88	U/L	44–147	N
GGT	29	U/L	9–48	N
Total bilirubin	0.7	mg/dL	0.2–1.2	N
Direct bilirubin	0.2	mg/dL	0.0–0.3	N
Albumin	4.3	g/dL	3.5–5.0	N
Total protein	7.1	g/dL	6.0–8.3	N

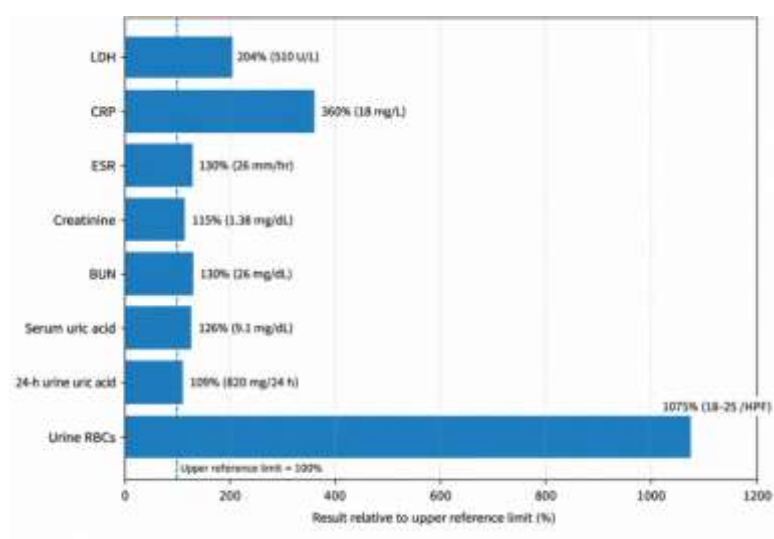


Figure 1 Renal infarction and uric-acid stone laboratory abnormalities: LDH, CRP, ESR, creatinine, BUN, serum uric acid, 24-h urinary uric acid, and urine RBCs, shown relative to reference limits.

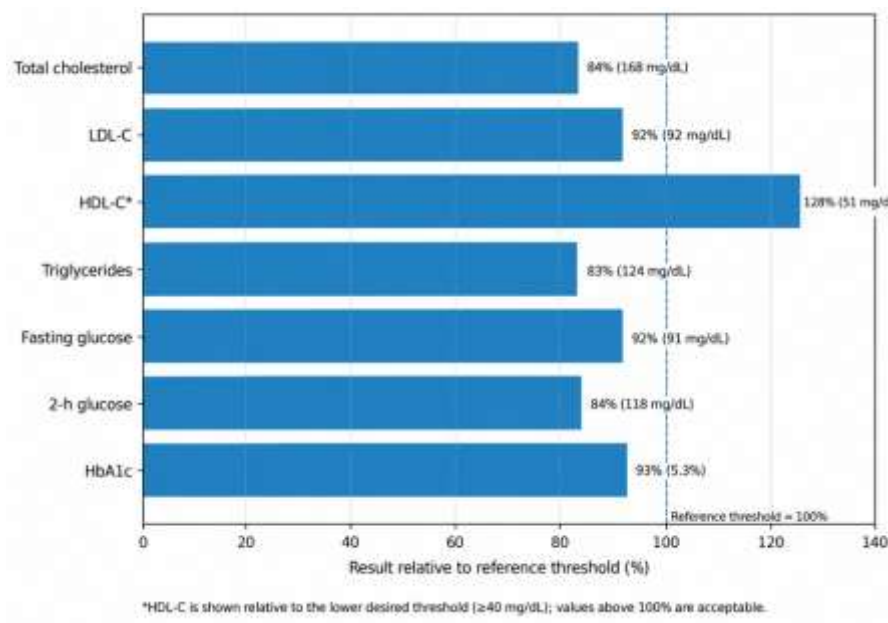


Figure 2 Lipid and glycemic profile: total cholesterol, LDL-C, HDL-C, triglycerides, fasting glucose, 2-h glucose, and HbA1c, demonstrating a normal metabolic profile.



Figure 3 Plain abdominal radiograph showing a left renal calculus



Figure 4 Axial contrast-enhanced CT showing marked abnormality of the left kidney

DISCUSSION

The discussion may begin by emphasizing that acute flank pain was the principal presenting symptom and the starting point of the diagnostic approach. Although flank pain is commonly associated with renal colic, it is a nonspecific symptom that may also occur in several renal, vascular, and abdominal disorders. Therefore, any physician, including a general practitioner or emergency physician, should approach acute flank pain systematically using clinical assessment, laboratory investigations, urinalysis, and appropriate imaging to establish the underlying cause and exclude alternative diagnoses [6].

In the present case, the laboratory findings supported the presence of urinary stone disease, particularly through microscopic hematuria, acidic urinary pH, uric-acid crystalluria, elevated serum uric acid, and increased 24-hour urinary uric-acid excretion. These findings strongly suggested a uric-acid-related stone-forming environment. However, laboratory investigations alone cannot accurately determine the size, exact anatomical location, or degree of obstruction caused by the stone. Furthermore, although the biochemical pattern may suggest uric-acid nephrolithiasis, definitive determination of stone composition generally requires direct stone analysis or appropriate imaging characterization [16, 17].

Renal function tests also demonstrated abnormalities, including increased serum creatinine and blood urea nitrogen with a reduction in estimated glomerular filtration rate. Nevertheless, these abnormalities are not specific for renal infarction and may occur in several forms of acute renal injury. Similarly, inflammatory markers and the marked elevation of lactate dehydrogenase may raise clinical suspicion of renal ischemia, but renal infarction cannot be confirmed on laboratory findings alone [13].

The major diagnostic challenge was therefore resolved by imaging. Contrast-enhanced computed tomography demonstrated renal perfusion abnormalities and a focal perfusion defect consistent with renal infarction, providing evidence of vascular compromise that could not be established by routine laboratory testing. Imaging also allowed visualization and localization of the renal calculus, thereby demonstrating the coexistence of vascular and stone-related renal pathology [18].

The combined diagnostic approach consequently supported the presence of both renal infarction and nephrolithiasis in the same patient. However, the simultaneous occurrence of these conditions does not, by itself, establish a causal relationship between them. The current findings demonstrate coexistence rather than causation. Other potential etiologies of renal infarction, including cardioembolic disease, renal vascular abnormalities, hypercoagulable states, and other systemic risk factors,

should therefore be considered and investigated before any direct relationship between the renal stone and renal infarction can be proposed[20,19].

CONCLUSION

Renal infarction should be considered in patients with acute flank pain when clinical severity, laboratory abnormalities, or imaging findings are not fully explained by nephrolithiasis. Early recognition and appropriate imaging are essential to minimize diagnostic delay and preserve renal function. A complementary diagnosis should be implemented to refine the renal infarction and renal stones with a definite type.

Acknowledgment

Not available.

Conflict of interests

The author declares no conflict of interest.

Ethical considerations

Informed written consent was obtained from this case to ensure his confidence in reporting his findings and visualized reports for a scientific purpose.

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