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Renal Infarction Caused by Isolated Spontaneous Renal Artery Intramural Hematoma

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Funds Collection G

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Conflict of interest: None declared

Patient: Male, 46
Final Diagnosis: Renal infarction
Symptoms: Flank pain
Medication: —
Clinical Procedure: CT
Specialty: Nephrology

Objective: Rare disease

Background: Acute renal infarction is an uncommon condition resulting from an obstruction or a decrease in renal arterial blood flow. Isolated spontaneous renal artery intramural hematoma is a rare cause of renal infarction.

Case Report: A 46-year-old healthy man presented to our emergency room because of sudden onset of severe right flank pain. An enhanced abdominal computed tomography scan showed a low-attenuated lesion in the lateral portion of the right kidney but no visible thromboembolisms in the main vessels. Computed tomography angiography revealed acute infarction resulting from intramural hematoma of the anterior segmental artery of the right kidney, with distal occlusion.

Conclusions: The rarity and non-specific clinical presentation of renal infarction often lead to a delayed diagnosis that may result in impaired renal function. Clinical suspicion is important in the early diagnosis, and intramural hematoma of the renal artery should be considered the cause of renal infarction even in healthy patients without predisposing factors.

MeSH Keywords: Hematoma • Infarction • Renal Artery

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/895285

Background

Although there were some of reports that the incidence of renal infarction (RI) is 0.007% or 1.4% [1,2], the true incidence of renal infarction is actually unknown. The cause of renal infarction is usually due to thromboembolism with emboli arising from the heart or aorta in atrial fibrillation. Renal infarction can also be caused by other conditions, like hypercoagulable status, infective endocarditis, endovascular intervention, and renal artery dissection cause. The clinical presentations of RI are flank pain, nausea, vomiting, and mild fever, which are nonspecific. The rarity and non-specific clinical presentation of RI often lead to a delayed diagnosis that may result in impaired renal function. Among the cause of RI, renal artery dissection (RAD) is often caused by an iatrogenic injury, trauma, fibromuscular dysplasia, atherosclerotic disease, and connective tissue disorder. Isolated extra-aortic arterial dissection is very rare and has been reported in renal and carotid arteries in a few studies at no more than 200 cases. Moreover, isolated spontaneous renal artery intramural hematoma (IMH) is also very rare. If physicians miss the diagnosis due to its rarity, renal function may deteriorate. Herein, we report a case of RI due to isolated spontaneous IMH treated successfully with only medical therapy.

Case Report

A 46-year-old man presented to our emergency department complaining of severe right flank pain in the late afternoon. Previously, he visited out-patients clinic with the same complaint in the early morning and was not able to find any cause of pain via kidney-ureter-bladder sonography, so he went home with some pain killers. As time went by, the pain persisted and worsened, but it did not aggravate on movement or radiate. The patient denied any trauma histories and medical histories, including hypertension. He was 30-pack-year current smoker and drank 1 bottle of soju (20% alcohol strength by volume) daily. He was a captain of an ocean-going ship.

The patient's vital signs were: blood pressure (BP), 160/90 mmHg; heart rate, 89 beats/min; respiratory rate, 20 breaths/min; and temperature, 36.5°C. On physical examination, his abdomen was soft with mild decreased bowel sound and without tenderness or rebound tenderness, but there was tenderness on his right flank. His white blood cell count was 13 140/mm^3 (neutrophil 72.3%, lymphocyte 19.9%); hemoglobin level, 16.0 g/dl; lactate dehydrogenase (LDH), 1261 IU/L; creatine phosphokinase, 92 U/L; blood urea nitrogen, 11.8 mg/dL; and creatinine, 0.87 mg/dL: other results are listed in Table 1. Urinalysis indicated proteinuria of 1+ and red blood cells at 0–2/high power field. Protein-to-creatinine ratio in spot urine samples was 209.8 mg/g. C-reactive protein level was 11.30 mg/dL. There were no specific findings on his chest radiograph and electrocardiograms.

Abdominopelvic computed tomography (APCT) revealed perfusion defects that sharply demarcated low-attenuated lesions in the lateral portion of the right kidney (Figure 1), consistent with acute infarction. The patient was started on anticoagulation therapy with heparin. We performed coagulation tests to exclude coagulopathy and the results were within normal range (Table 2). Arrhythmias, such as atrial fibrillation, were not observed on Holter monitoring. There were no thrombi or vegetations on transthoracic echography. Three days later, a multidetector computed tomography angiography (MDCTA) was performed to evaluate any vascular causes of RI. MDCTA revealed IMH of the anterior segmental artery of the right kidney, with distal occlusion (Figure 2). After obtaining the findings from the MDCTA, we discontinued the heparin infusion and started tight BP control with amlodipine 5 mg and valsartan 80 mg.

He felt free for pain and his BP was controlled to 120/70 mmHg within 3 days. Two months later, he had no symptoms related to RI and BP was 115/65 mmHg with normal renal function (creatinine: 0.88 mg/dL). The follow-up APCT showed an interval decrease in the volume of infarcted necrotic parenchyma of the right kidney, which was considerably smaller than the left one (Figure 3).

Discussion

Acute RI is an uncommon condition resulting from an obstruction of or a decrease in renal arterial blood flow. Although some studies have estimated the incidence of RI, the actual incidence is still unknown. Based on the relatively lower incidence compared to other intra-abdominal disease, it can be easily missed and undiagnosed. The common clinical presentation is persistent steady flank and abdominal pain. Other symptoms and signs, like fever, anorexia, nausea, vomiting, hematuria, proteinuria, and/or anuria, may accompany the pain. However, these clinical presentations can also indicate many other intra-abdominal diseases, such as renal stones, acute pyelonephritis, appendicitis, and/or diverticulitis. Therefore, the diagnosis is often missed and delayed, leading to renal impairments [3]. The major cause of RI is emboli due to cardiac disease, such as atrial fibrillation, atherosclerosis, valvular heart disease, myocardial infarction, ventricular aneurysm, and dilated cardiomyopathy [4]. Other causes are trauma, hypercoagulable states, hyperviscosity syndrome, fibromuscular dysplasia, factor V Leiden mutation, dissection of renal artery or aneurysm, and idiopathic RI [4]. The cause of RI in our case was IMH. IMH is one of the variants of dissection that is included in acute aortic syndrome. In cases of dissection, blood

Table 1. Complete blood count and basic metabolic panel.

Blood test (unit)		Test value	Reference range
White blood cell (/mm^3)		13,140	4,000–10,000
Diff	Neutrophil (%)	72.3	40–80
	Lymphocyte (%)	19.9	15–50
Hemoglobin (g/dL)		16.0	14.0–18.0
Platelet (/mm^3)		202,000	140,000–440,000
Protein (g/dL)		7.3	6.7–8.3
Albumin (g/dL)		4.2	3.1–5.2
Aspartate transaminase (IU/L)		132	7–38
Alanine transaminase (IU/L)		96	4–43
Total bilirubin (mg/dL)		1.3	0.2–1.1
Lactate dehydrogenase (IU/L)		1261	130–270
Creatinine phosphokinase (U/L)		92	50–200
Blood urea nitrogen (mg/dL)		11.8	8.0–20.0
Creatinine (mg/dL)		0.87	0.6–1.2
Amylase (U/L)		79	54–168
Lipase (U/L)		34	7–60
C-reactive protein (mg/dL)		11.30	0.00–0.50

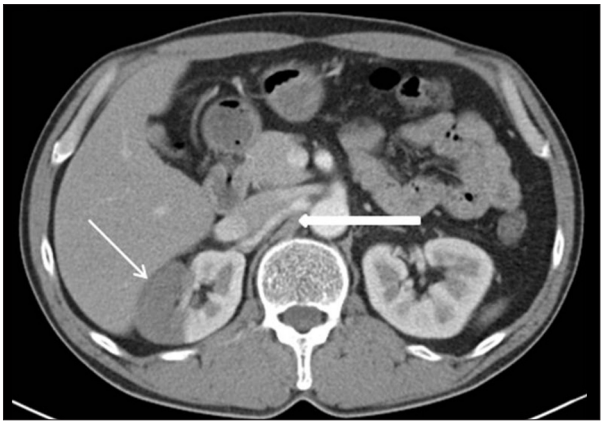

Figure 1. The abdominopelvic computed tomography (horizontal view) reveals perfusion defects, which sharply demarcated a low attenuated lesion in the lateral portion of the right kidney (arrow). No clearly visible thromboembolism in main vessels (bold arrow).

flows into the intima and enters the media layer, and the intimal flap is able to distinguish between the true and false lumens of the artery. In cases of IMH, hemorrhage occurs from the weakened vaso vasorum into the media layer and an absence of communication between the dissection sac and the true lumen of the artery. The natural history of IMH is poorly understood. Some studies have reported that patients with IMH have a better prognosis than those with dissection, whereas others have reported that mortality and morbidity resulting from IMH are similar to those resulting from dissection [5]. The etiology is also not well understood. Some associated conditions include uncontrolled hypertension, fibromuscular dysplasia, and atherosclerosis. Most often, renal artery dissection or IMH is accompanied with aortic dissection. Isolated spontaneous renal artery IMH without aortic dissection is rare, especially in a healthy individual.

Renal angiography is the most sensitive test for the diagnosis of RI and IMH (100% sensitivity); however, it is invasive. MDCTA is noninvasive, convenient, accurate, and plays an important role for the diagnosis of renal vascular disease [6]. Ultrasonography is also noninvasive; however, its sensitivity is only 11% [7]. In this case, we did not reveal the RI on initial ultrasonography. In recent years, enhanced CT has gradually replaced renal angiography in the diagnosis of RI [7, 8]. In RI, laboratory findings typically show elevated AST, ALT, and/or LDH levels. Particularly, elevated LDH level is the most common laboratory abnormality [7–9].

The treatment of RI remains controversial. The treatments include conservative therapy, such as hydration and blood pressure control, anticoagulation therapy such as heparin infusion or a dose of warfarin, and intravascular therapy such as thrombolysis and angioplasty. Clinical features, causes, and time interval from the onset of symptoms to the initiation of treatment are considered when selecting the treatment. The prognosis varies according to the site, size, and causes of RI.

Table 2. Coagulation test.

Blood test	Test value	Reference range
Prothrombin time(s)/international normalized ratio	11.6/1.08	10.0–13.0/0.85–1.30
activated partial thromboplastin time(s)	43.3	30–47
C protein activity (%)	141.8	70–140
S protein activity (%)	90.8	60–130
Lupus angicoagulant	Negative	Negative
Anti cardiolipin Ab IgM/IgG	Negative/negative	Negative/negative
Factor V Leiden mutation	Negative	Negative
Anti-thrombin III (%)	108.3	80–125
Factor VIII (%)	118.7	60–140

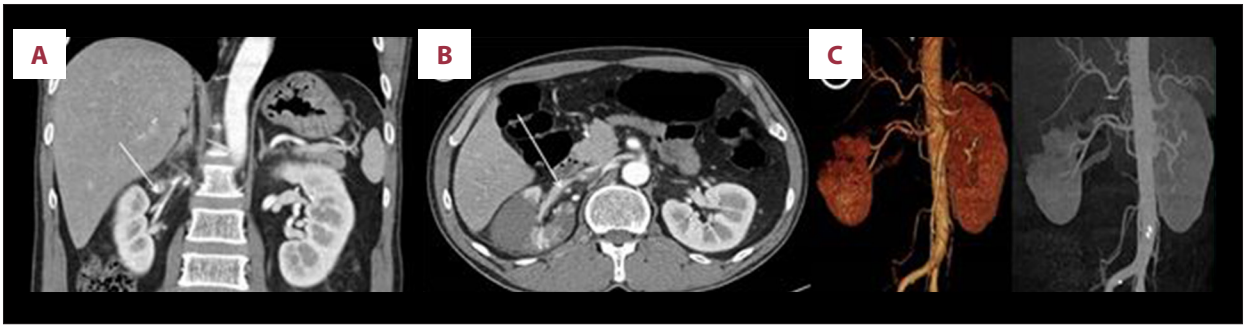

Figure 2. The multidetector computed tomography angiography reveals intramural hematoma of anterior segmental artery of right kidney, with distal occlusion (arrow). (**A**: coronal, **B**: horizontal, **C**: 3D and maximal intensity projection image).

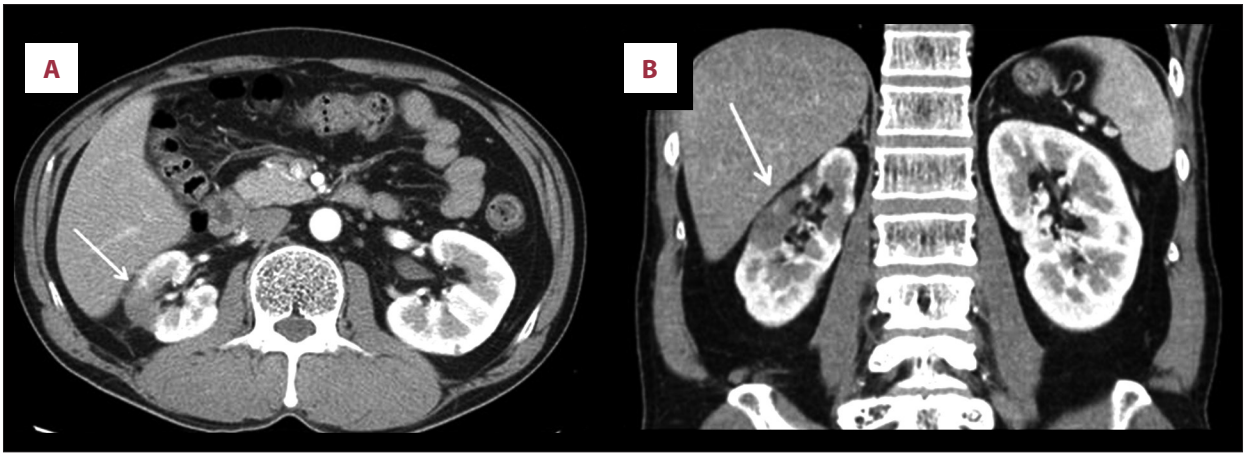

Figure 3. The abdominopelvic computed tomography (**A**: horizontal view, **B**: coronal view) reveals interval decreased volume of infarcted necrotic parenchyme, right kidney (about 40% involvement).

In this case, other renal vascular diseases were excluded on the basis of results from MDCTA. Because the patient denied a past history of hypertension, we concluded this was not a renal artery disease resulting from chronic hypertension; his high blood pressure was secondary to acute renal infarction. The patient was a captain of an ocean-going ship, but he denied exposure to repetitive trauma (for example, trauma resulting from movement of the ship) or any other situations that may result in an abrupt increase in blood pressure.

The general principles of the management of IMH and aortic dissection are similar. Controlling BP is important for managing

IMH. Failure of medical therapy to control of blood pressure requires surgical treatments or other interventions [10]. Some studies have reported that anticoagulation therapy is useful, but there is insufficient evidence supporting this [11]. In this case, the injured kidney was smaller. However, we did not perform the angioplasty or stenting because his renal function was stable and the site of IMH was not suitable for stenting due to high risk of additional infarction and the optimal time for intervention had passed. Therefore, only anti-hypertensive treatment was administered.

In patients presenting with acute flank or abdominal pain, it is important to consider an RI and to perform an optimal diagnostic workup. Renal artery dissection or IMH was previously most often diagnosed using renal angiography, but recently renal angiography has been replaced by MDCTA. The primary treatment is conservative therapy, such as taking antihypertensive drugs, but time intervals to treatment, the possibility of recovering renal function, underlying diseases, uncontrolled blood pressure, and disease progression should be considered when selecting a treatment strategy.

Conclusions

The rarity and non-specific clinical presentation of renal infarction often lead to a delayed diagnosis that may result in impaired renal function. Clinical suspicion is important in the early diagnosis and intramural hematoma of renal artery should be considered as a cause of renal infarction. Physicians should consider RI and RAD or IMH in patients with abdominal pain without risk factors when other intra-abdominal diseases are ruled out.

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