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Case Report

Spontaneous Renal Artery Dissection Complicated by Renal Infarction: Three Case Reports

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Spontaneous renal artery dissection (SRAD) is a rare disease entity. The diagnosis is usually delayed because clinical presentation is non-specific. We report three cases of symptomatic SRAD complicated by renal infarction which occurred in previously healthy middle-aged male patients. They visited the hospital due to acute abdominal or flank pain. They had no specific underlying disease or trauma history. The laboratory tests and physical examination were normal. They were not suspected of having SRAD initially, but computed tomography (CT) revealed dissection of the renal artery with distal hypoperfusion leading to renal infarction. They were treated conservatively with anticoagulation and/or antiplatelets for 6 months. They had a 6-month regular follow-up with CT, where resolution was confirmed in one patient and all patients remained asymptomatic. These cases emphasize the importance of clinical suspicion of SRAD in previously healthy patients who complain of abdominal pain without specific findings on initial investigation.

Key Words: Kidney, Dissection, Renal artery, Infarction, Endovascular procedures

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INTRODUCTION

Spontaneous renal artery dissection (SRAD) is defined as dissection of the renal artery in the absence of trauma or iatrogenic injury [1]. Because of its rarity, the current literature remains limited to case reports since the first case was reported by Bumpus in 1944 [2]. The true incidence, etiology, and natural history of this phenomenon are not precisely defined. Many cases occur in healthy individuals although SRAD has been known to be associated with atherosclerosis, intimal fibroplasia, malignant hypertension, Marfan's syndrome, Ehlers-Danlos syndrome and severe physical exertion [3]. The clinical presentation is non-specific and varies from acute abdominal pain to severe hypertension. Conventional angiography has been known as a gold standard for diagnosis. However computed tomography angiography (CTA) is nowadays used for diagnosis and follow up [4]. Imaging may demonstrate renal infarction with intimal flap in the renal artery. Therapeutic options for treatment include conservative treatment and revascularization [5]. Despite its rarity, there were three cases of SRAD during two years in our institution. We analyzed these three cases of SRAD and performed a review of the literature. We discuss the epidemiology, etiology, diagnostic work up and treatment plan associated with SRAD in general.

CASE

1) Case 1

A 50-year-old man experienced sudden onset of left lower quadrant abdominal pain 3 days ago. He visited a local clinic and had x-ray, urinary, and blood tests.

There were no specific findings, and the pain subsided spontaneously. Therefore, he was discharged with analgesics. However, he visited our hospital with the same pain for 20 minutes. The pain was dull and constant. He did not present any urinary symptoms. He was a 21 pack-year smoker and drank 2 times a week, but otherwise was previously healthy. His body mass index (BMI) was 27.26 kg/m^2. He denied any trauma or sudden changes in position.

His abdomen was soft, non-tender, non-distended, and bowel sounds were normal. Initially, blood pressure was 173/100 mmHg with an otherwise unremarkable physical examination. Laboratory evaluation revealed a serum creatinine level of 0.95 mg/dL and white blood cell (WBC) count of 10,200 cells/mm^3, with 68.8% neutrophils. Urinalysis was negative. Plain X-ray of the kidney ureter bladder (KUB) was normal. A contrast-enhanced CT scan revealed a renal artery dissection with renal infarction. There was circumferential wall thickening of the left mid to distal renal artery with thrombus and wedge-shaped perfusion defect at the left kidney lower pole posterolateral aspect (Fig. 1). The patient was anticoagulated with intravenous heparin to prevent thrombus extension. At the same time, antihypertensive therapy was started with nicardipine. The patient's pain improved within 1 day and was discharged with antiplatelet and antihypertensive medications. After 6 months, follow-up CT showed no change of renal infarction in the lower pole of the left kidney. He was advised to continue the medication and return to clinic every 6 months with CT.

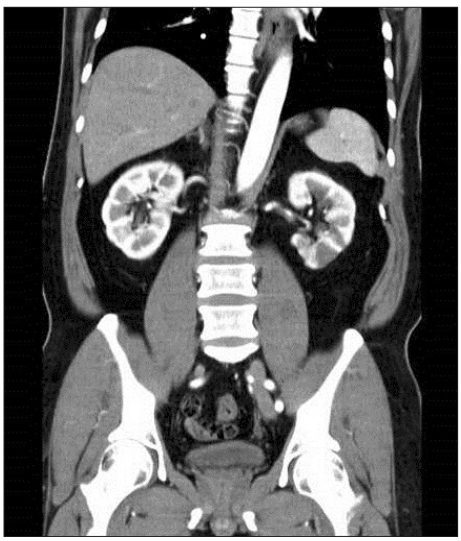

Fig. 1. Case 1. CT scan demonstrated circumferential wall thickening of the left mid to distal renal artery with thrombus and wedge-shaped perfusion defect at the left kidney lower pole posterolateral aspect.

2) Case 2

A 42-year-old man presented with sudden onset right flank pain which started one week ago. The pain resolved by itself but presented again with the same right flank pain of approximately 24-hour duration. There was also associated lower back pain, nausea, vomiting, chilling sense and generalized myalgia. The pain was dull and constant in nature. He denied any urinary symptoms. He had started taking an antihypertensive agent one month ago and the blood pressure was well controlled. He denied any trauma or sudden changes in position.

His BMI was 25.88 kg/m^2. Right lower quadrant tenderness and right costovertebral angle tenderness were positive. His initial blood pressure was 160/103 mmHg and the temperature was 36.3°C (97.34°F). The laboratory value was significant for WBC 15,400 cells/mm^3, with 85.9% neutrophils. However, C-reactive protein (CRP) level was normal. His serum creatinine level was 1.02 mg/dL urinalysis was negative. Plain X-ray of the KUB was normal. A contrast-enhanced CT scan disclosed a focal dissection at the right distal renal artery with infarction (Fig. 2A).

The patient was anticoagulated with enoxaparin 80 mg every 12 hours. At the same time, antihypertensive therapy was started with amlodipine besylate 5 mg every 12 hours and analgesics were given. The pain was settled within 4 days. A follow-up CT was conducted on hospital day 7. The image showed no change of infarction in the anterior aspect of the right kidney, with no change of thrombus in the right renal artery. Interestingly, a new dissection was identified in the left renal artery. He was discharged with antiplatelet and antihypertensive medication on hospital day 11. The patient returned to clinic 18 months later, and CT showed old renal infarction sequelae in the right kidney with disappearance of thrombus in the left renal artery and no evidence of perfusion defect (Fig. 2B). He was advised to continue the prescribed medications and visit the hospital every 6 months with CT.

3) Case 3

A 35-year-old man visited our hospital complaining of left lower quadrant abdominal pain. The first attack had occurred 2 days ago, which resolved without any treatment. However, the same pain had started again one hour ago. He had no past medical history. However, he recalled having practiced golf a few days ago but denied having suffered trauma on the left side. He had no urinary symptoms. His abdomen was soft, non-tender and only mildly distended. Bowel sounds were normal.

His BMI was 25.61 kg/m^2. Initially, blood pressure

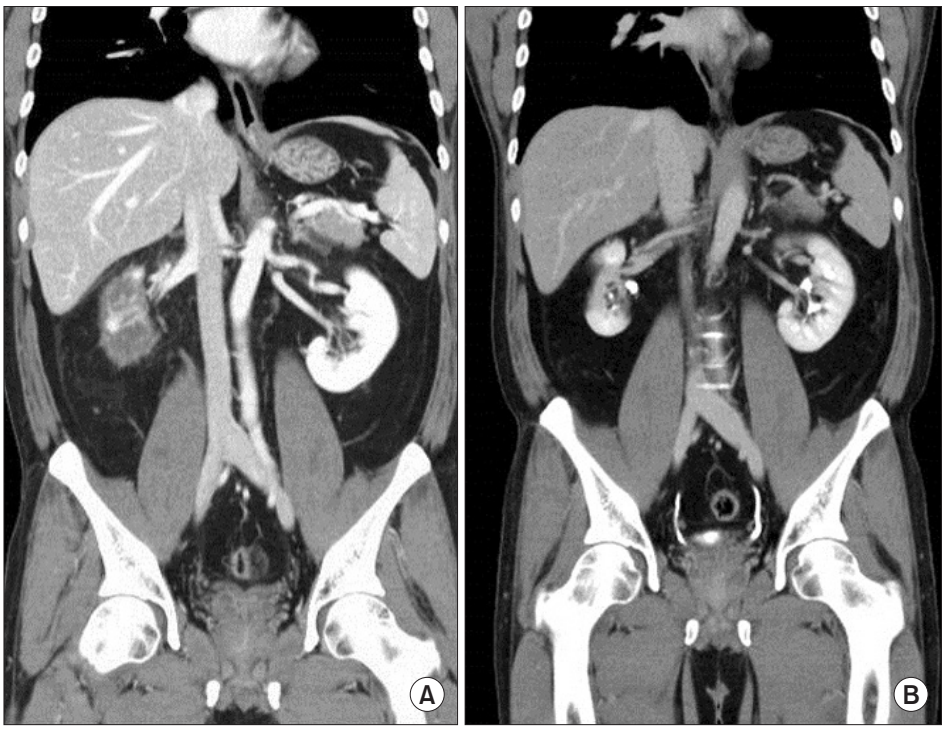

Fig. 2. Case 2. (A) CT scan disclosed a focal dissection at the right distal renal artery with infarction. (B) Eighteen months later, CT scan showed old renal infarction sequelae in the right kidney with disappearance of thrombus in the left renal artery and no evidence of perfusion defect.

was 161/95 mmHg with an otherwise unremarkable vital sign. The physical examination and laboratory tests were normal. Left renal infarcts associated with left renal artery dissection were identified on CT (Fig. 3). Dalteparin 10,000 IU for 24 hours with antiplatelet agent was started. The blood pressure was normalized after pain control. However on hospital day 2, he had a fever of up to 38.2°C (100.76°F) with leukocytosis (WBC 11,400 cells/mm^3, with 77.2% neutrophils) and elevated CRP (1.42 mg/dL). He was given intravenous 3rd generation cephalosporin. The pain was relieved within hospital day 2 without analgesics. He was discharged with an antiplatelet agent and antibiotics. He is scheduled for a follow-up visit with CT.

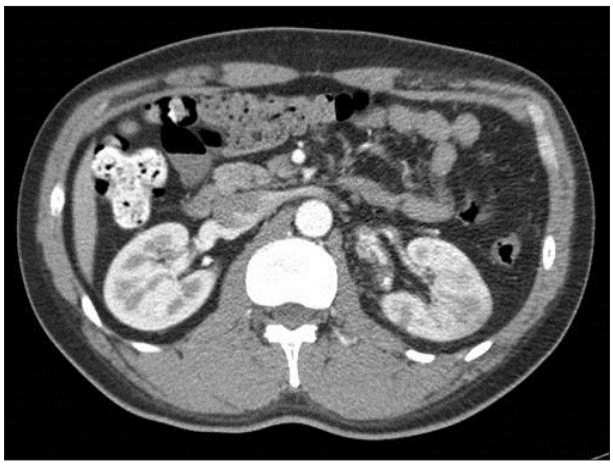

Fig. 3. Case 3. Left renal infarcts associated with left renal artery dissection were identified on CT.

DISCUSSION

In the cases we report, there are several common points. First, they are all male patients. Actually, SRAD is dominant in men. Male:female ratio is known to range between 4:1 and 10:1 [6]. Second, they were middle-aged patients. It usually occurs between the fourth and sixth decades of life [7]. Third, they have no definite underlying disease except one patient who started antihypertensive agents one month ago. Fourth, they presented with abdominal pain and hypertension. This hypertension was temporary and was normalized after several days of antihypertensive medications. Fifth, renal artery dissection with renal ischemia was found. Dissection of the renal artery leads to formation of hematoma between the intima and media layers, which as it progresses, causes severe stenosis of the renal artery leading to renal parenchymal ischemia along the distribution of the renal artery dissection. Such renal ischemia results in increased secretion of renin, which leads to renovascular hypertension. If the renal artery is totally occluded, renal failure may also develop [1]. Sixth, they experience recurrent episodes of pain attack with spontaneous resolution in between. Like in our cases, most SRADs develop silently, without severe symptoms at onset. In addition, SRADs found during diagnostic work-

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up of hypertension are mostly found in a chronic stage and therefore the spontaneous course during the acute stages of dissection is unknown [8]. Therefore, it is probably a more common phenomenon than reported because many may be silent or may resolve spontaneously [9]. Last, the physicians who first met the patients did not suspect of renal artery dissection. The first impressions were acute gastroenteritis, acute appendicitis and urinary obstructive disease, respectively. As in our cases, the diagnosis is often delayed because of the vague and various symptoms. Therefore, a high index of suspicion is required to diagnose SRAD in previous healthy patients presenting with abdominal pain without any other definite signs or symptoms.

The etiology of SRAD is not well known. The association with connective tissue disorders including fibromuscular disease, Ehlers-Danlos syndrome and Marfan's syndrome was documented in a few case reports. In addition, malignant hypertension, atherosclerosis, blunt trauma and strenuous exercise have also been increasingly reported as a possible etiology. Cocaine abuse and extracorporeal shock wave lithotripsy were reported in rare cases [10]. The association with anti-phospholipid antibody has been reported [11]. Arterial dysplasia and abnormalities involving the vasa vasorum have been viewed as predisposing factors for SRAD. Rupture of the vasa vasorum may result in hemorrhage with subsequent intramural hematoma, which may lead to medial ischemia and a further compromise in vessel wall integrity. Unusual physical stress can lead to traction of arteries and abnormalities in the integrity of connective tissue may present as predisposing factors that can result in rupture of the vasa vasorum leading to a subsequent cascade of events such as intramural hematoma and SRAD. Conventional angiography has been known as a definitive diagnostic modality of SRAD, because it can precisely demonstrate the extent and nature of the vascular involvement while identifying potential treatment options [12]. However currently, CTA or magnetic resonance angiography (MRA) has substituted conventional angiography because of their noninvasiveness and accuracy [4]. In this series, we had no problems with the diagnosis and follow up of SRAD using CT. Given the increasing accuracy of MRA and CTA imaging, these new technologies may gradually replace traditional arteriography as the gold standard [13].

The treatment of SRAD is divided into two categories (Fig. 4). One is conservative treatment and the other is revascularization. Like in our cases, medical treatment alone is performed in many cases and includes control of pain, normalization of blood pressure, and systemic anticoagulation. Although comparative studies with long-term follow-up are lacking, non-operative treatment or clinical observation is preferred and is sufficient in many cases [7]. Treatment of SRAD-associated hypertension should follow the recommendations published in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC VII). According to the recommendations, the goal blood pressure should be less than 140 mmHg systolic and less than 90 mmHg diastolic. Hypertension with systolic blood pressure (SBP) between 140 and 159

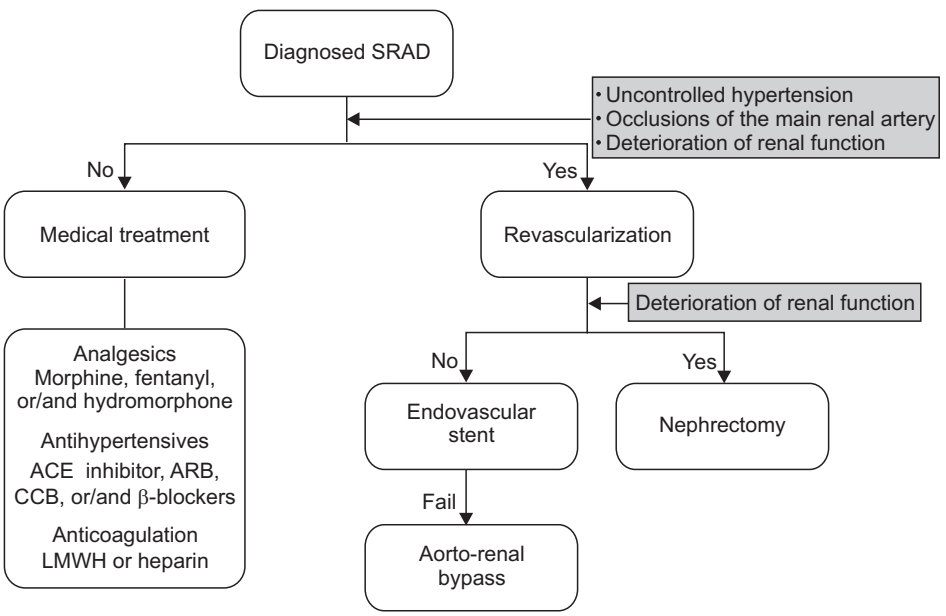

Fig. 4. Treatment algorithm for SRAD. SRAD, spontaneous renal artery dissection; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; CCB, calcium channel blockers; LMWH, low-molecular-weight heparins.

mmHg or diastolic blood pressure (DBP) between 90 and 99 mmHg should be controlled with a single antihypertensive medication. Hypertension characterized by SBP 160 mmHg or DBP 100 mmHg should be treated with a two-drug combination. Refractory hypertension can be treated with optimization of dosages or by adding additional drugs until the goal blood pressure is achieved [14]. Angiotensin-converting enzyme inhibition or angiotensin receptor blockade may be beneficial particularly in renovascular atherosclerosis without the absence of acute kidney injury or rise in serum creatinine of greater than 30% [15]. There is still controversy as to the need for use of anticoagulation and/or antiplatelet agents in visceral artery dissections. However, we adopted a strategy for use of short-term anticoagulation during the hospitalization period with the intention of preventing false lumen thrombus propagation from occluding the true lumen flow to the kidneys. This was followed by antiplatelet agent for 6 months for preventive measures.

There are two options for revascularization including endovascular stenting and open surgery. The decision to proceed with revascularization is made in the presence of intractable severe hypertension despite maximal medical management, hemodynamically significant occlusions of the main or major segmental renal arteries and significant deterioration of renal function. Endovascular intervention includes placement of self-expanding stents, stents with coiling, and thrombolysis followed by stenting. Good short and long term outcomes of intervention have been reported in few selected cases [1,4,16].

The surgical treatments include arterial bypass and nephrectomy. Nowadays, endovascular intervention is becoming the preferred method of treatment with safe and effective outcomes. However, patients with failed medical and endovascular therapy should be considered for operative treatment, if a surgically correctable lesion is present [5]. Aorto-renal bypass is the most common surgical approach with extracorporeal reconstruction and autotransplantation [17]. Nephrectomy should be considered in large renal infarction with significant deterioration of kidney function or severe refractory renovascular hypertension since ongoing uncontrolled hypertension can cause significant damage to the contralateral kidney and other end organs. In addition, failure of endovascular intervention with worsening hypertension can also be considered as a relative indication for nephrectomy, provided that the underlying lesion is not amenable to surgical repair [8].

Other spontaneous isolated visceral artery dissections, including superior mesenteric artery (SMA), inferior mesenteric artery (IMA), and celiac artery dissections, are also rare disease entities. The most frequently reported cases are renal artery, followed by SMA, celiac artery, and IMA dissections. Their etiology and pathophysiology are also not well known due to their rare incidence, and the methods of diagnosis do not differ either [18]. However, there is still controversy as to the need for use of anticoagulation and/or antiplatelet agents in visceral artery dissections. The method of conservative treatment varied among the reported literatures; ranging from dual anticoagulation and antiplatelet agents, single anticoagulation or antiplatelet alone, or no medication use at all [19,20]. However contrary to SMA or celiac dissections, the location of the lesions varied and there was no prevalent location in SRAD. The reason for this is unknown, but a possible theory is that there is no abrupt curvature near the os of the renal artery as in the case of SMA, and therefore the hydrostatic pressure exerted on the wall leading to tear at such location may not be so evident in SRAD. Therefore, focal ischemic changes in mid-to distal renal artery dissections (after branching) are identified in many cases. In addition, abdominal pain is a more accurate predictor in SMA dissection since it involves the small bowel. In SMA dissection, the indication for revascularization was persistent abdominal pain not relieved by conservative treatment lasting 7 days or longer, or signs and symptoms suggestive of bowel ischemia and large aneurysmal dilation of the SMA likely to rupture [19]. However, in SRAD, indications for revascularization were uncontrolled hypertension, occlusions of the main renal arteries and significant deterioration of renal function.

In conclusion, SRAD occurs most frequently in middle-aged male patients. The diagnosis is usually delayed because of the vague and nonspecific symptoms. Therefore the diagnosis of SRAD requires a high index of suspicion in patients complaining of abdominal pain without other specific findings. CTA or MRA is mainly used for diagnosis, and treatment options including conservative management or revascularization should be individualized based on the clinical findings.

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