



Case Report

Bilateral spontaneous renal artery dissection

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Summary Spontaneous renal artery dissection is a rare condition that precedes renal infarction. We describe a 48-year-old, normotensive healthy woman presenting with left flank pain of sudden onset. Enhanced abdominal computed tomography demonstrated a dissecting intimal flap of the left renal artery complicating renal infarction. Doppler ultrasonography, selective angiography, and intravascular ultrasound revealed a dissecting intimal flap, with a large false lumen and narrow true lumen, of the renal artery bilaterally. Conservative management was undertaken with anticoagulant and analgesic therapy, and the patient was discharged after an uneventful clinical course.

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Introduction

Aortic dissection frequently extends to the renal arteries, but spontaneous renal artery dissection (SRAD) is rare and its natural history is not yet well defined. Misdiagnosis and diagnostic delay of SRAD are common, because of the confusing clinical presentation [1]. In 40% or more of cases, renal infarction occurs and some physicians have seen cases of repeat episodes and progression of dissection [2]. But, the etiology of SRAD is not well understood and the best ther-

apeutic strategy is still under debate. We herein present a case report of a bilateral SRAD patient who recovered from conservative management and was discharged after an uneventful clinical course.

Case report

A 48-year-old, previously normotensive healthy woman presented with left flank pain of acute onset and was admitted to a medical service. Her symptoms began suddenly while she was resting at home and persisted for approximately 6 h. She had no significant medical or family history, and there was no history of trauma. The patient's blood pressure at presentation was 134/60 mmHg, the pulse was 50/min, and the temperature, 36.8 °C. On physical examination, she

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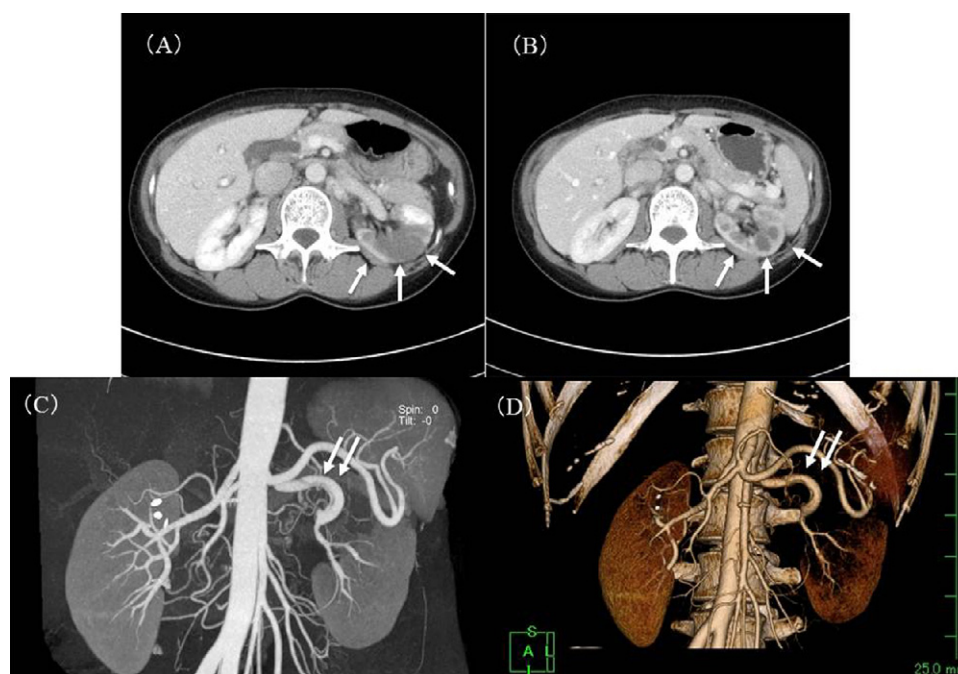


Figure 1 Enhanced abdominal computed tomography. (A) At admission, evidence of left kidney infarction (arrows). (B) On day 7 of hospitalization, recovery from the kidney infarction is evident (arrows). (C, D) At admission, 3D reconstructions showing left renal artery dissection with an intimal flap (arrows) and defect in the upper pole of the left kidney.

was alert, and her abdomen was soft and flat, with normal bowel sounds. She complained of left flank pain, and palpation revealed left costo-vertebral tenderness. Laboratory studies revealed a white blood cell count of $12 \times 10^3/\mu\text{L}$, serum lactate dehydrogenase of 219 IU/L, and serum creatinine of 1.2 mg/dL (106.08 $\mu\text{mol/L}$). Urinalysis revealed 3+ hematuria and 2+ proteinuria, but the examination was negative for glucose and nitrite, and also for leukocyte esterase activity. Enhanced abdominal computed tomography (CT) performed showed a perfusion defect in the left kidney (Fig. 1A) and an intimal flap in the left renal artery (Fig. 1C and D). The patient was administered systemic anticoagulant therapy with 10,000 IU/day of unfractionated heparin for 7 days and received an analgesic drug, under the diagnosis of left renal infarction (Fig. 1B). On day 7 of hospitalization, enhanced abdominal CT revealed normal enhancement except in the upper pole of the left kidney and evidence of recovery from the left kidney infarction. A renogram and renal scintigram with $^{99\text{m}}\text{Tc}$ -diehylenetriamine pentaacetic acid showed marked decline of the glomerular filtration rate and reduced uptake in the left kidney (Fig. 2); however, the findings of Doppler ultrasonography led to a suspicion of bilateral renal artery dissection with an intimal flap (Fig. 3A–D). On day 30, she was transferred to our hospital for further evaluation.

We performed selective renal artery catheterization to determine the treatment strategy because she complained of left flank pain continuously (Fig. 4A and B). After introduction of a guiding catheter into renal artery ostium, selective hand-injection, and wiring was performed very carefully because there was no real arterial wall surrounding the false lumen, and perforation could lead to devastating complications. Intravascular ultrasound (IVUS) imaging showed a narrow true lumen and a large false lumen of the renal

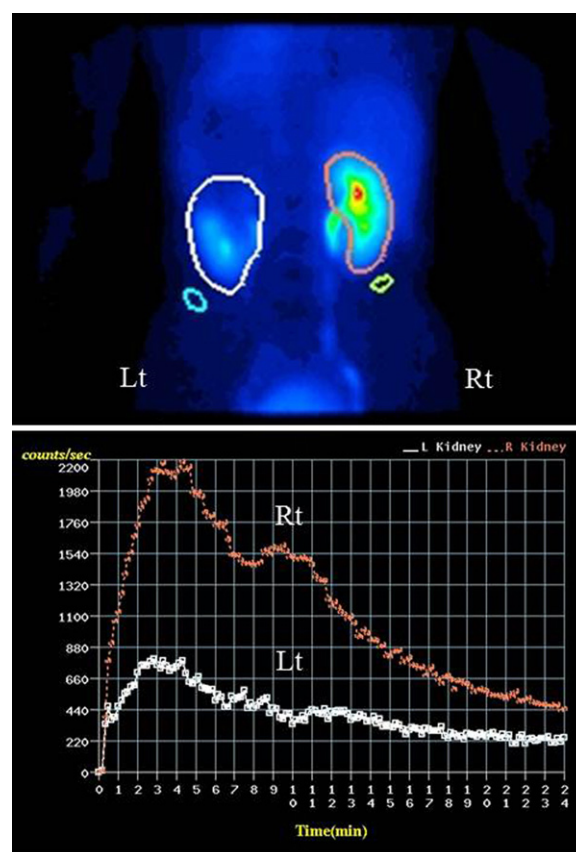


Figure 2 Renogram and renal scintigram with $^{99\text{m}}\text{Tc}$ -diehylenetriamine pentaacetic acid. Marked decline of the glomerular filtration rate and a large defect in the upper pole of the left kidney are evident.

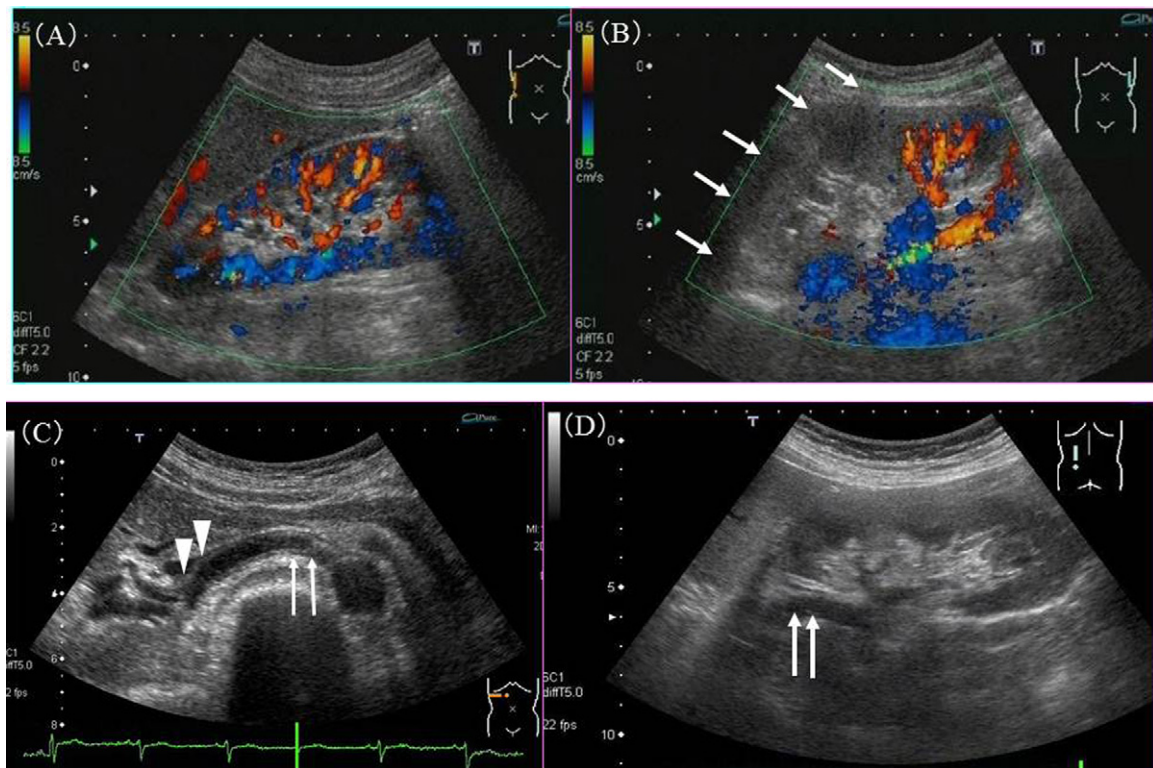


Figure 3 Doppler ultrasonography (A, right kidney; B, left kidney; C, right renal artery; D, left renal kidney and artery). (A) No atrophic changes of the right kidney are evident, and the renal perfusion appears to be adequate. (B) No atrophic changes of the left kidney are seen either; however, the upper pole of the left kidney shows deficient renal perfusion (arrows). (C) Dissection of the right renal artery was suspected; proximal entry (arrows) and distal re-entry (arrowheads). (D) Suspected intimal flap in the left renal artery (arrows).

artery bilaterally (Fig. 4C and D). We felt that percutaneous interventional treatment was impossible, because wiring would have been difficult due to the narrow true lumen. We decided to prescribe additional analgesic drugs for this patient, as she rejected surgical management. Two months later, her symptoms recovered little by little.

We attempted to determine the reason for the bilateral renal arterial dissection; however, the etiology remained unknown; factors such as cardiovascular disease, systemic collagen vascular disease, trauma, arteriosclerosis, hypertension, iatrogenic accident, Marfan's syndrome, and Ehlers–Danlos syndrome could, however, be ruled out. She was discharged from the hospital and remained asymptomatic and normotensive through the early follow-up. Six months later, she is symptom-free and blood pressure is normal, as is her serum creatinine level (0.90 mg/dL; 79.56 $\mu\text{mol/L}$). Doppler ultrasonography revealed a patent large false lumen of the renal artery.

Discussion

SRAD was first reported in 1944 by Bumpus [3]. Before 1970, it was usually detected at autopsy [4]. Since then, SRAD has been diagnosed with increasing frequency during the patients' lifetimes, and isolated cases/small series have been reported. SRAD occurs at an estimated incidence of 0.05%, with a predilection for middle-aged males

[1]. Bilateral SRAD is an extremely rare condition, reportedly accounting for 12–18% of all cases of SRAD [5–7]. Despite the clinical significance of aortic dissection, there is an ongoing debate with regard to the pathophysiology of the initial damage to the intima. Several authors have suggested local breakdown of the intima, or intramural hemorrhage from the vasa vasorum [8]. Although the etiology of SRAD has not yet been precisely defined, conditions reported to be associated with the development of SRAD include fibromuscular dysplasia (FMD), malignant hypertension, severe atherosclerosis, Marfan's syndrome, Ehlers–Danlos syndrome, systemic collagen vascular disease, trauma, and iatrogenic accidents [1]. FMD frequently coexists with SRAD and in one study, pathologic evidence of FMD was demonstrated in 45% of the patients [5]. Especially, renovascular FMD tends to affect women between 15 and 50 years of age [9]; while SRAD shows a strong male predominance (10:1), FMD is distributed at a female:male ratio of about 4:1 [10]. Therefore, we suspect that FMD may have been the predisposing factor for the renal artery dissection in our patient.

Although the reported incidence is low, SRAD probably occurs more commonly than is reported, because many cases may be symptom-free or show spontaneous resolution [11]. Importantly, chronic dissections which are often asymptomatic are most frequently those associated with FMD of the renal artery [12]. For this reason, we consider that the right renal artery dissection in our patient did not occur

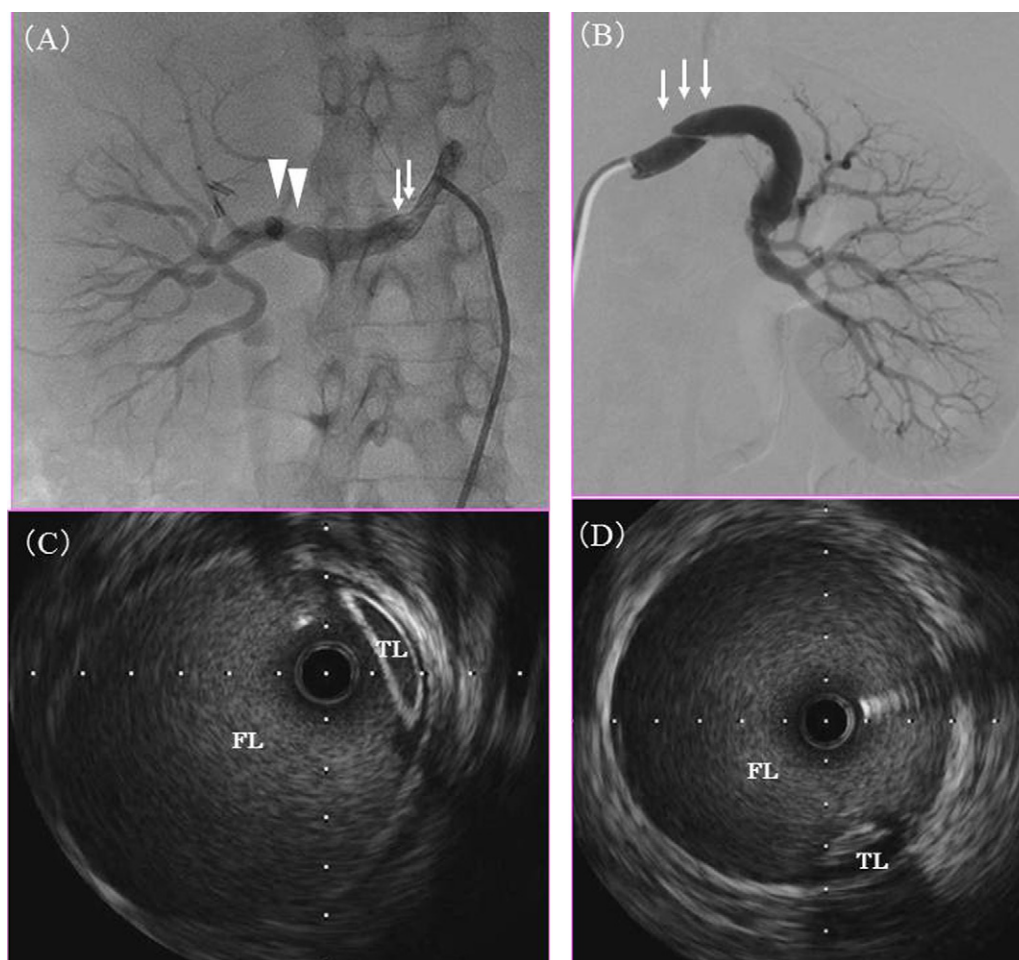


Figure 4 Selective renal artery angiography and intravascular ultrasound images (A, C, right renal artery; B, D, left renal artery). (A) Right renal artery dissection is confirmed, with the proximal entry (arrows) and distal re-entry (arrowheads). (B) Left renal artery dissection with an intimal flap (arrows) and almost no enhancement of the upper pole of the left kidney. (C, D) Intravascular ultrasound images showing a narrow true lumen (TL) and large false lumen (FL) of the renal artery bilaterally.

simultaneously with the left, but was previously silent. On the other hand, renal infarction was caused by left renal artery dissection and marked decline of glomerular filtration rate in our case. Lacombe reported that re-entry of dissection was postulated as the most probable mechanism for silent dissections that do not entail arterial hypertension [5]. In fact, after studying Doppler ultrasonography, selective renal artery angiography, and IVUS images we suspected distal re-entry in only the right renal artery in our case. So, we guess that spontaneous re-entry considered as a healing process occurred and did not cause renal infarction in the right renal artery.

The therapeutic strategy for SRAD should be based on the results of careful examinations, including tests of renal function and renal angiography, and also on the technical feasibility; however, treatment of SRAD remains controversial [13]. Surgery may be indicated when medical therapy fails to control the blood pressure, improve symptoms, or renal failure is obvious [5]. A few cases of percutaneous interventional treatment have been reported [8,12]. In our case, IVUS was helpful, and particularly allowed us to distinguish the large false lumen from the narrow true lumen [12]. Stent placement within the true lumen as treatment

of the renal artery dissection would have been extremely difficult because of the narrow true lumen on both sides. It has been reported previously that conservative management may be an appropriate therapeutic option in cases with uncontrolled hypertension and progressive renal dysfunction [1,7,14]. Revascularization should be considered only in patients with complete occlusion of the main renal artery, malignant hypertension, renal function impairment, worsening symptoms, or refractory renovascular hypertension. However, more long-term follow up is essential to understand the prognosis of renal artery dissection.

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