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Isolated spontaneous renal artery dissection: diagnosis and endovascular management

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ABSTRACT
Spontaneous, isolated focal renal artery dissection is an extremely rare cause of renovascular hypertension. Imaging technology for the renal arteries has evolved recently with the development of multidetector computed tomography angiography (CTA), and intravascular ultrasound. We describe a 52-year-old man with spontaneous renal artery dissection complicated by renovascular hypertension, successfully diagnosed initially with CTA, then evaluated by intravascular ultrasound, and finally, treated successfully with stenting.

Key words: • *renovascular hypertension* • *computed tomography* • *intravascular ultrasonography* • *stent*

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Renal artery dissection is a rare cause of renovascular hypertension (1, 2). Acute dissection may present spontaneously or as a complication of angiography. The most common symptom in acute dissection is abdominal pain, and the most common sign is hypertension (3). The etiology of spontaneous renal artery dissection is not precisely defined. Although fibromuscular dysplasia, severe atherosclerosis, malignant hypertension, and Marfan syndrome are known to be associated with renal artery dissection according to some reports, this condition can develop in a healthy person with normal blood pressure (2). Treatment of renal artery dissection remains controversial. Surgical intervention is usually suggested for cases with medically uncontrolled hypertension or progressive renal dysfunction (4–6), although some authors have observed that medical treatment alone provides blood pressure control (7). Only a few cases of percutaneous interventional treatment are reported in the literature (8, 9).

Case report

A 52–year-old man with a five-year history of controlled hypertension presented to our hospital complaining of uncontrolled blood pressure for one week. Despite monotherapy (metoprolol 50 mg/day), his initial blood pressure was 170/100 mmHg with an otherwise unremarkable physical examination. His father died of a myocardial infarction, and his brother died of aortic dissection. The patient's blood urea nitrogen and creatinine levels were within normal limits. Urinalysis by dipstick was negative for protein and blood. After the antihypertensive therapy was changed to amlodipine 10 mg/day and carvedilol 25 mg/day, his blood pressure decreased to 150/90 mmHg. Computed tomography angiography (CTA) of the aorta and renal arteries showed a 1-cm long focal dissection at the distal truncal right renal artery (Fig. 1); however, a fenestrated renal artery could not be excluded completely (10). There was no history of catheter angiography that might have caused iatrogenic injury to the renal artery.

Selective right renal artery catheterization was then performed, which confirmed that the focal lesion was limited to the main trunk of the renal artery, and did not extend to the division branches. Selective catheterization should be performed very carefully because there is no real arterial wall surrounding the false lumen, and perforation could lead to catastrophic complications. Selective hand-injection was performed carefully. The tip of the catheter in the false lumen revealed the extent of the dissection (Fig. 2). An intravascular ultrasound (IVUS) catheter was then advanced over a wire passing through the caudal channel. The live images demonstrated that the caudal channel did not have an intimal echogenic reflection, suggesting that this lesion was a dissection rather than a fenestration (Fig. 3). In addition, the thickened intimal flap with a

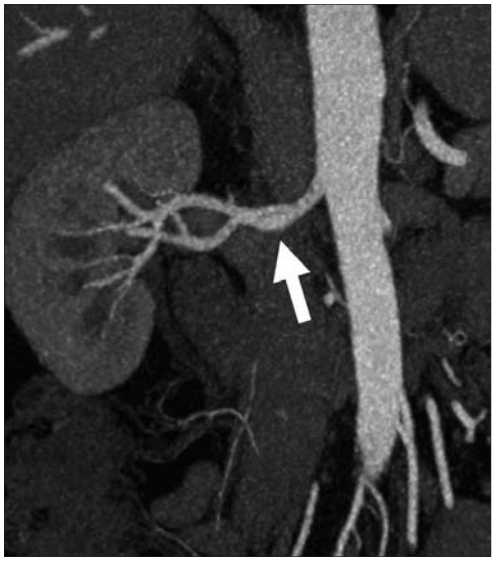

Figure 1. Reformatted coronal oblique image from the initial diagnostic CT angiography shows two lumens at the distal truncal right main renal artery *(arrow)* which is suggestive for focal renal artery dissection.

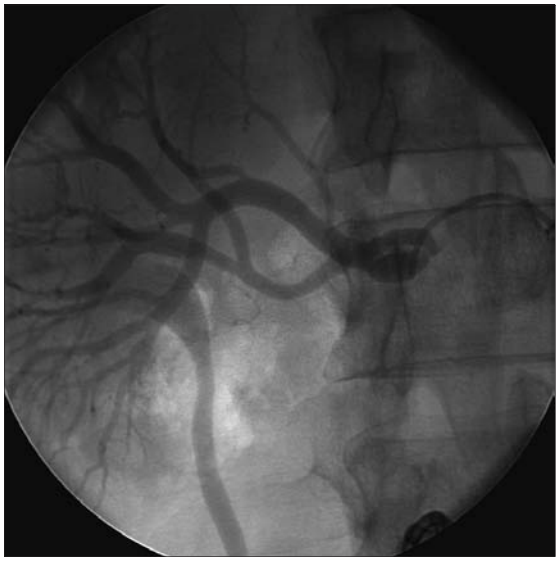

Figure 2. Selective right renal artery injections with the catheter tip in the caudal lumen shows filling of both lumens, which all are limited to the main trunk confirming the CT angiography findings.

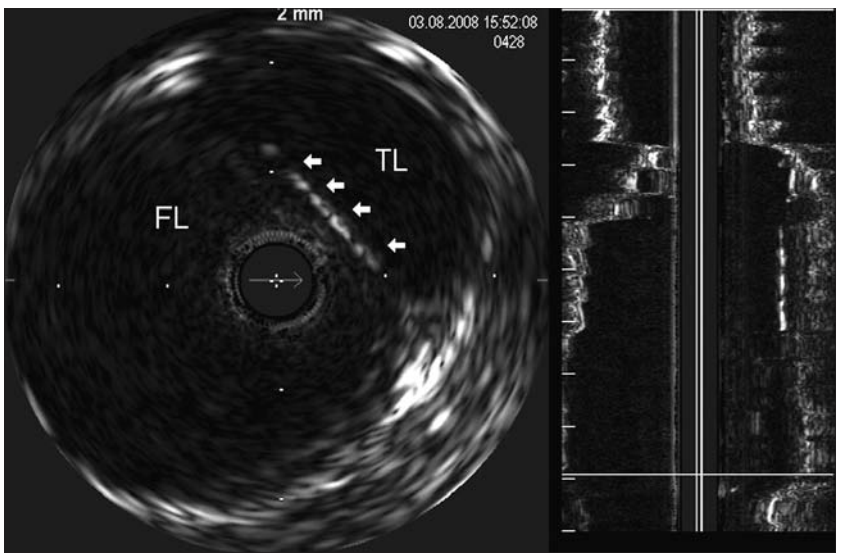

Figure 3. Intravascular ultrasound image obtained by the US catheter within the caudal lumen (FL, false lumen) showing the intimal flap with bright intimal reflection *(arrows)* only at the side of the cranial true lumen (TL).

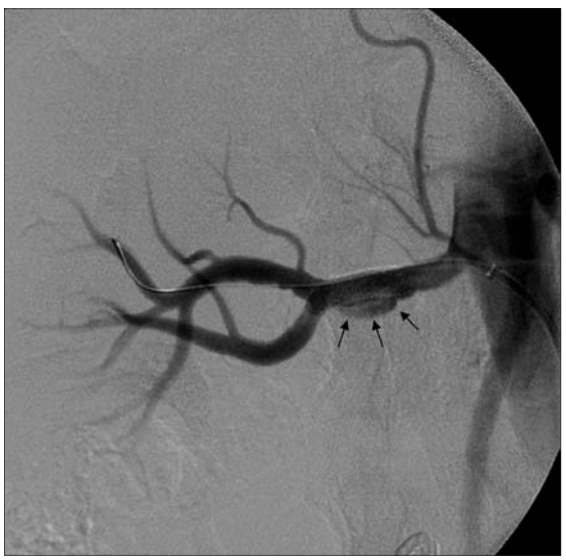

Figure 4. Final digital subtraction angiography image after the endovascular treatment. Cranial lumen, which was compressed by the false lumen, is now widely open after stenting, with compressed false lumen just below the stent *(arrows).*

single layer of intimal echogenic reflection suggested that the IVUS catheter was passing through the false lumen of subacute-chronic focal dissection. Pressure measurements were then obtained, revealing a 35-mmHg systolic pressure gradient across the dissection.

Following a successful cannulation of the distal renal artery branches through the true lumen, a stent 6 mm in diameter and 18 mm long was deployed across the dissected truncal segment of the main renal artery. Care was taken to avoid extending the stent into the branches. Control arteriogram revealed a significantly enlarged true lumen with a widely patent stent in excellent position (Fig. 4). The false lumen became compressed by the stent with faint filling of contrast medium. The patient was treated with clopidogrel 75 mg for 6 weeks, and lifetime aspirin. A week after the procedure, his blood pressure was 120/70 mmHg and it remained within normal limits for the remainder of the follow-up period. Although the CTA obtained at 2-month follow-up revealed very slight residual filling of the false lumen, CTA at 6-month follow-up revealed a widely patent renal artery stent with no residual filling of the false lumen.

Discussion

Renal artery dissection may be caused by iatrogenic injury, trauma, or arterial disease including fibromuscular disease, atherosclerotic disease, or connective tissue disorders (11). Clinical presentation of dissection of the renal artery may be classified as acute or chronic. Acute dissections are classified as spontaneous, iatrogenic, or agonal, whereas chronic dissections are classified as functional and silent (3).

Despite the clinical significance of aortic dissection, there is an ongoing debate with regard to the pathophysiology of the initial damage to the intima. The trigger in aortic dissection is believed to be a tear or hemorrhage in the aortic wall that leads to a separation of the media with potential end-organ damage from propagation of the dissection, and obstruction of the origins of vital vessels such as the mesenteric, renal, or iliac arteries (12). Studies of the initiating mechanisms of dissection also lead us to consider the factors that determine the extent of the dissection. Several studies reported in the literature attempt to explain the correlation of the following factors with the dissection: increased blood pressure, heart rate, sympathetic activity, basal vascular tone, and vasoconstrictive hormones, prothrombotic tendency, platelet aggregability, plasma viscosity, hematocrit, and circadian rhythms (13).

Another important question raised in this report is why in some patients the dissection involves only the ascending aorta, and in others, it extends to the level of the iliac arteries. Also raised is the question of why a patient may develop only a focal renal artery dissection, while sparing the remainder of the renal artery and the aorta.

Acute agonal renal artery dissections have been discovered at autopsy in patients who died from critical systemic illness such as sepsis, malignancy, stroke, chronic renal failure, or cirrhosis (3). Iatrogenic dissections may be caused by guidewires, catheters, and angioplasty balloons. Because there was no history of catheter angiography in this patient, the diagnosis in this case merits comment. A renal complication of primary hypertension is the most likely etiology; however, the patient also had a brother who died recently of aortic dissection, thus raising the possibility of a hereditary systemic disorder or predisposing hemodynamic factors, but our patient did not have any known connective tissue disease or congenital disorders of the vascular system. One other possible diagnosis in this case is segmental mediolytic arteriopathy, which is a rare, non-inflammatory, non-atherosclerotic arteriopathy that involves the splanchnic and renal arteries (14).

Chronic dissections may result in renovascular hypertension, or be entirely asymptomatic. Although an occasional patient will recall an episode of flank pain, the majority of these dissections are asymptomatic, the only indication of their existence being the development or worsening of hypertension (3). Chronic dissections are most frequently associated with fibromuscular dysplasia of the renal artery, which accounts for 5–10% of renal artery stenosis. These dissections are typically encountered during the evaluation of renovascular hypertension caused by either the dissection itself or associated fibromuscular dysplasia (15).

The utility of intravascular ultrasound is well described in the literature for determining the false and true lumens in aortic dissections (16). Although there is very limited information on IVUS in the evaluation of visceral/renal artery dissections, IVUS was helpful in this case, particularly to distinguish the false from the true lumen. The stent placement within the true lumen for the treatment of the dissection in our case was primarily tailored according to the IVUS images. Similarly, the hemodynamic significance of focal dissection of the renal artery is based on information from the aortic dissection literature (17). In addition, as mentioned above, the diagnosis of fenestrated renal artery could not be totally excluded by CTA. To the best of our knowledge, the only reported case of renal artery fenestration is very similar to this case with regard to imaging (10). The IVUS exam together with DSA helped us to reach the diagnosis of dissection in our case, and thus assisted in the management of our patient. If the authors of the fenestration case had had a chance to examine their patient with IVUS, the final diagnosis might have changed.

Despite technological developments that have evolved in the past decade, the pathological process resulting in arterial dissection remains poorly understood . Renovascular hypertension is widely accepted as a treatable and even curable etiology for systemic hypertension; however, long-term patency of the stents placed for renal artery stenosis, and the effect of stenting on kidney function remain of major concern in this field. It is most likely that the timing of renal artery intervention is crucial to providing the greatest benefit to patients. In order to preserve renal function, an aggressive approach in the recognition and treatment of this entity is required to recognize affected patients prior to irreversible impairment of kidney function. We believe that the case presented here is an important example of renovascular hypertension diagnosed at a very early stage and treated appropriately. IVUS examination should be a part of renal artery evaluation, particularly with non-atherosclerotic pathology, such as non-atherosclerotic aortic dissection.

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