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Wide-necked renal artery aneurysm: endovascular treatment with stent-graft

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ABSTRACT

Aneurysms of the renal artery are rare and have an estimated incidence of 0.09% in the general population. They may be diagnosed incidentally or during the evaluation of related symptoms. They may be followed up or treated either surgically or endovascularly. We present a successful percutaneous treatment of a renal artery aneurysm with stenosis by a stent-graft in a 55-year-old woman, who was diagnosed during the evaluation of labile hypertension. Follow-up was for 6 months.

Key words: • *renal artery* • *aneurysm* • *endovascular treatment* • *stents*

Renal artery aneurysms may be asymptomatic and diagnosed incidentally or diagnosed during the evaluation of related symptoms. Aortic aneurysms account for most of the aneurysms in the abdominal region, while visceral aneurysms are rare. Although renal artery aneurysms are rare, they account for approximately 25% of all visceral aneurysms and have a pathophysiology that is unclear. The likelihood of rupture seems to increase as the diameter exceeds 2 cm. Medical management, endovascular exclusion, or surgery are the treatment alternatives (1–4).

We present the successful endovascular treatment of a wide-necked left renal artery aneurysm with stenosis in a 55-year-old woman by the use of a stent-graft.

Case report

A 55-year-old woman was referred for endovascular treatment because of renal artery stenosis. She had labile hypertension and has been under antihypertensive treatment with 2 drugs (amlodipine 10 mg/day and ramipril 10 mg/day) for 2 years. In the last month, her blood pressure became more labile and an additional antihypertensive drug (metoprolol 50 mg/day) was added to her regimen. Her blood pressure remained unstable (150–190/95–120 mmHg) despite using 3 drugs for 3 months. She denied any hematuria, flank pain, or trauma. Laboratory tests revealed her renal functions were within the normal range. Color Doppler ultrasonography (CDUS) was performed, but the result was suboptimal due to her obesity. Thereafter, contrast enhanced magnetic resonance angiography was performed, which revealed a critical stenosis in the mid portion of the left renal artery (Fig. 1). Based on the contrast enhanced magnetic resonance angiography findings, stent placement was planned. Selective left renal angiography was performed via standard retrograde femoral access with a 5 F cobra catheter. During the angiography, the left main renal artery was tortuous and had a caudal angulation in the mid portion. At the angulation point, an approximately 20-mm, wide-necked, saccular aneurysm was present, which was applying external compression to the renal artery. Both caudal angulation of the renal artery and external compression of the aneurysm were causing moderate stenosis (Fig. 2). We decided to treat the lesions via the axillary route with a stent-graft. The procedure was performed through a 7 F introducer. At the beginning of the procedure, heparin 5000 IU was administered. A 7 F guiding catheter (Guider 40XF, Boston Scientific, USA) was used to catheterize the left renal artery and a 0.014" guidewire (Roadrunner, Cook, USA) was introduced through a Tuohy-Borst. Since there was tortuosity and angulation at the aneurysm site, we were able cross the lesion after several attempts with the guidewire (Fig. 3a). A premounted, balloon-expandable, 5 × 25 mm stent-graft (Jostent Stent Graft, Abbott

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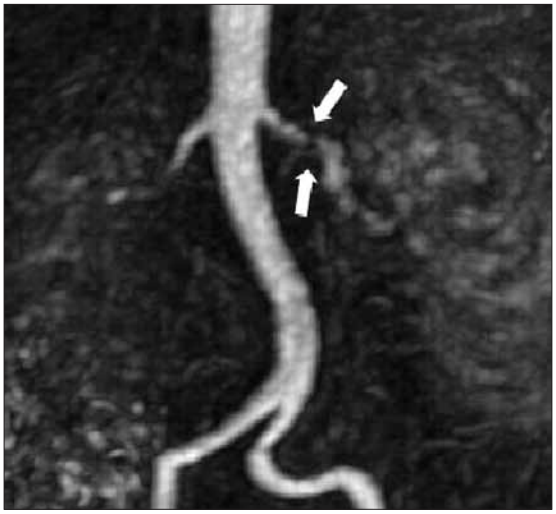

Figure 1. Contrast enhanced MR angiogram shows high-grade stenosis *(arrows)* in the mid portion of the left renal artery.

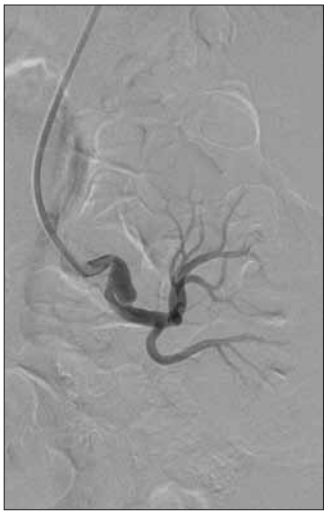

Figure 2. Selective left renal artery angiogram shows caudal angulation in the mid portion and a wide-necked aneurysm.

Vascular, Netherlands) was successfully deployed at 14 atm, covering the neck of the aneurysm. A final renal angiogram showed complete exclusion of the aneurysm, a patent lumen, and no sign of residual stenosis (Fig. 3b). There were no complications and her blood pressure was within the normal range (115–125/75–80 mmHg) immediately after the procedure. The patient was discharged on the following day and put on an anticoagulant regimen of clopidogrel 75 mg/day for 3 months and aspirin 300 mg/day for lifetime. She was also asked to record her blood pressure, without taking any medication. At the first week follow-up, her blood pressure was stabilized at 120–135/85–95 mmHg. Her antihypertensive medications were then discontinued and she was asked to record her blood pressure weekly. At the sixth-month follow-up, her blood pressure was within the normal range and she was not taking any medication. Follow-up computed tomography (CT) angiography was performed and revealed that the aneurysmal sac had resolved and the stent was patent (Fig. 4). She was asked to come to routine follow-ups, or when and if she experienced an increase in or lability of her blood pressure.

Discussion

Primary renal artery aneurysms are relatively rare. They have an estimated incidence of 0.09% in the general population, 0.1%–2.5% in angiographic series, and up to 9.7% in autopsy series (1, 2). They may constitute up to 25% of all visceral artery aneurysms and are usually detected during the fourth and sixth decades of life. Males and females are equally affected and aneurysms are most commonly located along the main renal artery. Renal artery aneurysms exhibit calcification in 18% of cases and 8.5% are >20 mm (1, 3). In our case, a wide-necked, saccular aneurysm >20 mm was located in the mid portion of the main renal artery and there was no calcification.

There are 4 basic types of renal artery aneurysms: saccular, fusiform, dissecting, and intrarenal. Saccular aneurysms account for up to 80% of renal artery aneurysms (3). Even though the underlying pathophysiology is unclear, they appear to be related to arterial fibrodysplasia exaggerated at branch points in the renal vasculature due to discontinuity in the internal elastic lamina (4). More commonly, the underlying etiologies are atherosclerosis or fibrous dysplasia. Secondary renal artery aneurysms are seen in such conditions as malignancies, infection (mycotic), and trauma, in association with systemic diseases, such as polyarteritis nodosa, neurofibromatosis, William's syndrome, midaortic syndrome, autoimmune vasculitis, and tuberous sclerosis, or are iatrogenic (e.g., renal biopsy) (3).

Fibromuscular dysplasia is an idiopathic process that leads to stenosis of medium to small arteries as a result of segmental overgrowth of fibrous and muscular elements of the arterial media and, to a lesser extent, the adventitia. Although it is first described in renal arteries, fibromuscular dysplasia most commonly affects the cervical internal carotid artery. It is usually bilateral and is associated with spontaneous arterial dissections, as well as aneurysms. It is more common in women and radiographically usually appears as a string of beads. There are several types of fibromuscular dysplasia, which are based on their histology: medial fibroplasia (60%-70%), perimedial fibroplasia (15%-25%), medial hyperplasia (5%-15%), medial dissection (5%), intimal hyperplasia (1%-2%), and adventitial fibroplasia (<1%). Dissections and aneurysms are mostly seen in medial fibroplasia and medial dissection subgroups. Although the radiological appearance strongly suggests the disease, only pathological examination confirms the diagnosis (5). If the findings (female patient, mid portion of the main renal artery, aneurysms associated with stenosis) are taken into consideration, fibromuscular dysplasia may be considered as the underlying etiology in our case.

The clinical significance of these aneurysms varies from that of an incidental finding to hypertension, flank pain and hematuria due to renal artery embolization, infarction, or rupture with resultant mortality (3, 6). Estimates of the risk for rupture vary, but appear to be low, except in pregnant women and noncalcified saccular aneurysms (1, 3, 6, 7). However, rupture carries a mortality rate of up to 80%. Although hypertension is also a common finding in this population of patients, it is debatable whether renal artery aneurysms alone cause hypertension (3, 4). Branch renal artery ischemia due to compression or displacement by the aneurysm, peripheral renal infarction due to microembolization from a mural thrombus within the aneurysm, relative renal ischemia related to altered antegrade flow or turbulence caused by the aneurysmal segment, associated stenosing fibrous renal artery disease, arteriovenous fistula as a consequence of aneurysmal erosion into a renal vein, and concurrent essential hypertension unrelated to renal artery pathology may cause hypertension (3, 4). In our case, hypertension was thought to be secondary to arterial ischemia due to congenital dysplastic stenosis or due to

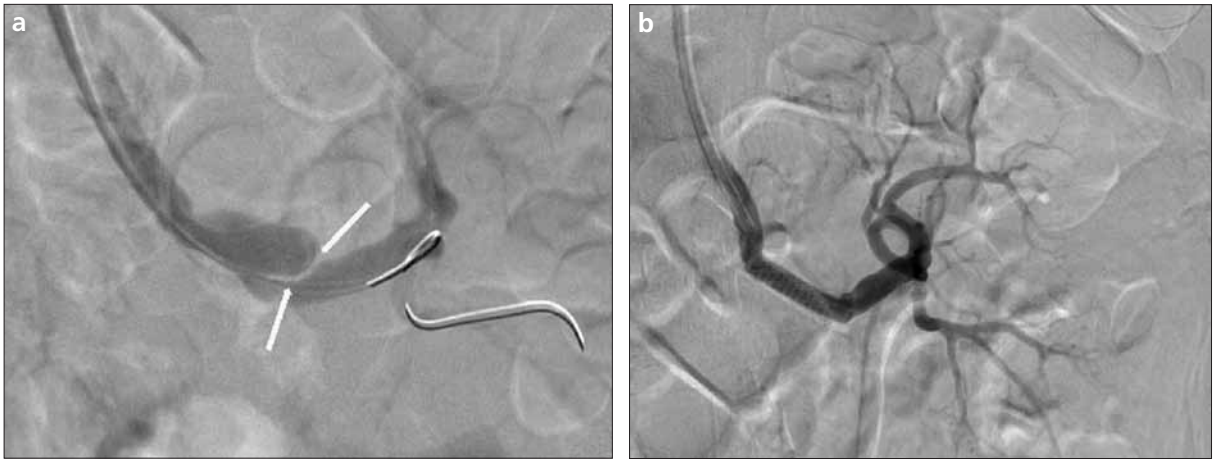

Figure 3. a, b. Selective left renal artery angiogram after crossing the lesion via the guidewire shows external compression on the renal artery by the aneurysmal sac *(**a**, arrows)*. Follow-up angiogram after deployment of the stent-graft **(b)** shows complete exclusion of the aneurysm and a patent lumen.

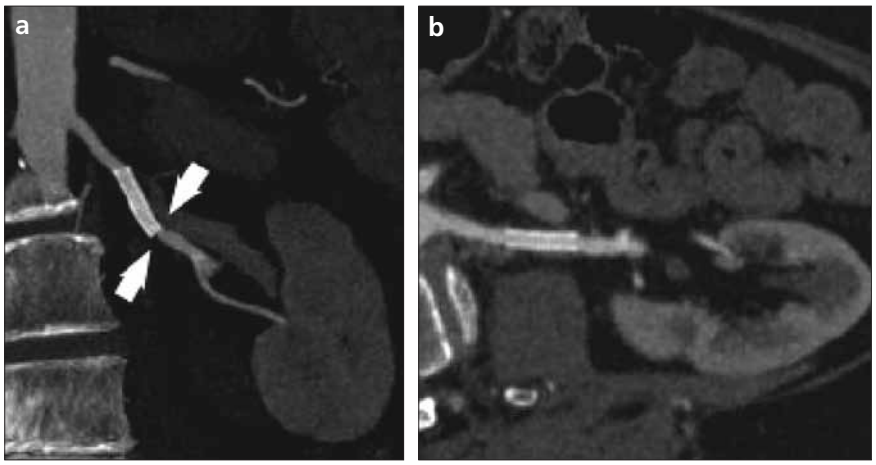

Figure 4. a, b. Coronal **(a)** and axial **(b)** reformatted CT angiography images taken during the sixth-month follow-up show the patent stent and complete resolution of the aneurysmal sac. Note that there seems to be a mild stenosis at the distal end of the stent in the coronal reformatted image *(**a**, arrows)*, while the lumen is seen patent in the axial reformatted image.

mild stenosis caused by the angulation and external compression of the main renal artery by the aneurysm.

The standard therapy for large renal aneurysms is surgery. Surgical options include aneurysm resection, aortorenal bypass, reno-renal interposition, reimplantation, patch angioplasty, and nephrectomy (6, 8). Indications include an expanding or symptomatic aneurysm, aneurysmal size >2 cm, renal infarction, intractable hypertension, or in the context of anticipated pregnancy (3, 4, 6, 8). Of the indications for treatment, size of the aneurysm is the most controversial. Reports have documented rupture at sizes <2 cm, but other reports suggest no necessity of treatment for sizes <2 cm (4, 9–11). Surgical series have reported a cure of hypertension in 50%–88% of cases, especially in children, and preservation of branch renal arteries is achieved in up to 86% of cases (12–14). Potential complications include the need for nephrectomy, branch occlusion, ureteral stricture, postoperative graft occlusion, and death (3). Surgical approaches for treatment of aneurysm are technically challenging, requiring retroperitoneal dissection for exposure, and carry a morbidity rate of up to 28%, making endovascular approaches a viable alternative (3, 4, 8).

Percutaneous approaches have been reported as balloon-assisted coil or onyx embolization, stent-assisted coil embolization, and instillation of alcohol into the aneurysm (3, 6, 15, 16). While this technique offers sparing of branch vessels, there is always a possibility of delayed recanalization of the aneurysm, and non-target embolization or migration of the coils. There is also a risk that there may be a spillover thrombosis that may protrude or embolize into the parent vessel. Moreover, this strategy is not readily applicable to aneurysms that have a wide neck. Stent-grafts offer direct sealing of the neck of the aneurysm and aneurysmal occlusion. In lesions located at the bifurcation, sacrifice of branch vessels can occur with concomitant loss of renal mass accompanied by significant patient discomfort and prolongation of hospitalization (3, 6, 7, 9). In our case, we decided on the treatment based on the patient's uncontrolled labile hypertension and the size of the aneurysm. Stent-graft was chosen because of the wide neck and the location of the aneurysm, where the stent could be deployed safely without sacrificing any side branch. Self-expandable stent-grafts also could be used for the endovascular treatment of aneurysms, especially for large vessels, but they are not preferred for lesions associated with small vessels because of the large profile of the shaft of the stents (i.e., a 9 F introducer is necessary for a 6 × 30 mm Wallgraft stent, Boston Scientific, USA). Additionally, they are not preferred for lesions that need precise stent positioning because of the possible jumping and shortening of the stent during deployment, which may lead to side branch loss and inadequate exclusion of the lesion. We chose not to use a self-expandable stent-graft in the presented case because of the need for a large introducer (9 F), which was not suitable for axillary access, and unpredictable shortening of the stent-graft. Even though low-profile stent-

grafts are now available (Fluency, Bard, USA), they were not at the time this case was treated.

In conclusion, percutaneous treatment of renal artery aneurysms with stent-grafts seems to be a safe and effective procedure; however, the long-term patency in the renal vasculature remains unknown, necessitating close follow-up of the patients treated with this minimally invasive technique.

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