To the Editor:

Renal artery stenosis after renal sympathetic denervation (RDN) with radiofrequency ablation in patients with resistant hypertension is a rare complication of this medical procedure. In a study of 153 patients, only 1 new renal artery stenosis was reported at 3 years after renal denervation. In a study of 88 patients, no renal artery stenosis was reported at 6 month follow-up. To date there are few reports of renal stenosis after RDN.1–3

We present a 39-year-old patient with high blood pressure (BP) of 10-year evolution, left ventricular hypertrophy, and left putaminal hypertensive intracranial hemorrhage, which occurred 1 year before denervation, On examination, BP was 170/100 mm Hg, despite treatment with 6 antihypertensive drugs. Resistant hypertension was confirmed by 24-h ambulatory blood pressure monitoring (ABPM). Mean 24-hour ABPM measurement was 144/91 mm Hg (diurnal BP 155/99 mm Hg; nocturnal BP 128/79 mm Hg). Secondary hypertension was excluded, and no renal artery stenosis was found. Seven radiofrequency ablations were applied on the right side and 5 on the left side (Figures 1 and 2). Ablations were applied in a spiral pattern at a minimum distance of 5 mm.1 Renal artery alterations were visible immediately after denervation. A vasospasm occurred in both renal arteries directly after RDN (Figures 1 and 2); in the right side, it resolved with intravenous nitroglycerin, but there was no response in the left side. The patient was asymptomatic within the days after denervation, and renal function remained normal. A Doppler ultrasound study performed after 48 hours showed an increase of velocity in the middle third of both renal arteries, slightly higher than normal. One month later, there were no changes in the renal function; however, in the 24-hour ABPM measurement, BP was raised considerably (24-hour ABPM: 167/98 mm Hg; diurnal BP 183/108 mm Hg; nocturnal BP 149/85 mm Hg). Two months later, ultrasound showed an increase of peak velocity of 338 m/s in the left renal artery. Computed tomographic angiography showed 75% stenosis in the left renal artery and 30% in the right renal artery (Figure 3).

RDN is an increasingly common therapeutic option for patients with refractory hypertension in spite of medical therapy. Currently available data1–4,6–8 indicate that the procedure is effective in lowering BP in selected cases, showing few adverse events (2%). The renal artery stenosis has been reported only in 2 cases,9,10 one at 3 months, and the other at 5 months after RDN.

Our case shows the development of a bilateral renal artery stenosis after RDN. We found an early renal artery spasm, which was probably caused by direct manipulation of the catheter (Figures 1 and 2). This renal artery alteration continued in the patient follow-up. We then assumed that the stenosis detected by computed tomographic angiogram (Figure 3) could have been caused by RDN. No atheroma or edema was found during the RDN procedure. In this case, BP raised considerably. Extending this technique is likely to increase the frequency of this complication, so we consider that early imaging follow-up and control of renal arteries could be appropriate in these patients.

Disclosures

None.


**Figure 1.** Left renal sympathetic denervation (RDN) angiogram. 

A, Pre-RDN procedure. Left renal artery. 

B and C, Ablation areas on left renal artery. 

D, Left renal spasm after RDN.

**Figure 2.** Right renal sympathetic denervation (RDN) angiogram. 

A, Pre-RDN procedure. Right renal artery. 

B and C, Ablation areas on right renal artery. 

D, Right renal spasm after RDN.

**Figure 3.** Computed tomographic renal angiography control, 2 months after renal sympathetic denervation.
Bilateral Renal Artery Stenosis After Renal Denervation
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