Renal artery stenosis presenting as hypertension with hypokalemia

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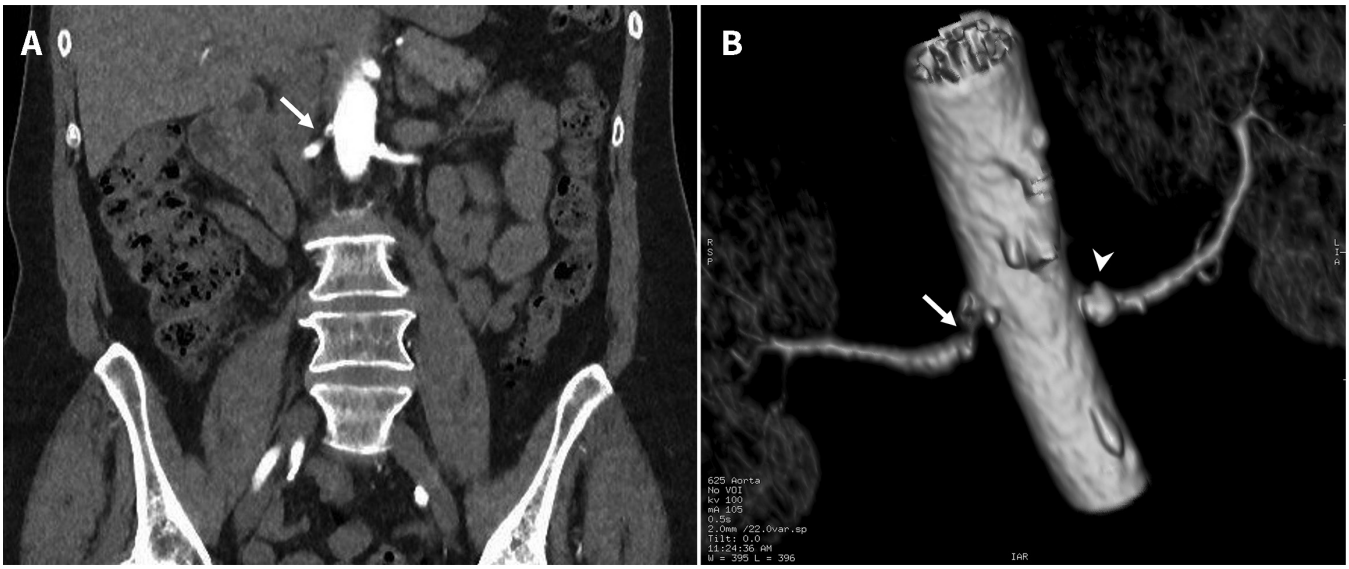

Figure 1: (A) Computed tomographic renal angiogram of a 69-year-old woman showing right renal artery stenosis (estimated occlusion up to 90%) at site of origin (arrows) in the coronal plane and (B) three-dimensional reconstruction. The left renal artery shows heavy ostial calcification (arrowhead) but no stenosis.

A 69-year-old woman was seen by her family physician with new-onset, asymptomatic hypertension and hypokalemia. Her physical examination was normal and her mean 24-hour ambulatory blood pressure was 220/110 mm Hg. Her serum creatinine level was 74 (normal 44–97) µmol/L, but her serum potassium was low at 2.8 (normal 3.5–5.3) mmol/L. Her family physician prescribed 10 mg amlodipine once daily and referred her to our department.

We found that the patient's plasma metanephrines were normal; however, her plasma renin was 12.0 (normal 0.3–2.2) nmol/L/h, her aldosterone was 1765 (normal < 630) pmol/L and her aldosterone-to-renin ratio was 147 (normal < 800). We performed a renal angiogram, which showed right-sided atherosclerotic renal artery stenosis (Figure 1). We managed her symptoms conservatively with 5 mg amlodipine daily and 10 mg atorvastatin daily, with annual monitoring for complications in a multidisciplinary renovascular clinic. Her average home blood pressure readings are 127/75 mm Hg, her potassium is normal and her estimated glomerular filtration rate is 75 mL/min.

Hypertension with hypokalemia (spontaneous or drug-induced) suggests an aldosterone-mediated pathophysiology, typically primary aldosteronism (eponymously called Conn syndrome), which has a prevalence of 8% among patients with primary hypertension.[1,2] However, not all instances of hypertension with excess aldosterone are caused by primary aldosteronism (Appendix 1, available at www.cmaj.ca/lookup/doi/10.1503/cmaj.220091/tab-related-content). In primary aldosteronism, renin is suppressed, leading to a high aldosterone-to-renin ratio. In contrast, a high renin level and low-to-normal aldosterone-to-renin ratio are indicative of hyperreninemic secondary hyperaldosteronism. This results from excessive activation of the renin–angiotensin–aldosterone system in response to reduced renovascular blood flow, owing to renal arterial obstruction from atherosclerosis (prevalence 24% among older individuals with resistant hypertension) or from fibromuscular dysplasia (usually among younger women).[2]

Treatment of the underlying cause can resolve secondary hyperaldosteronism. One study found that revascularization with renovascular stenting for atherosclerotic renal artery stenosis reduces antihypertensive pill burden but not adverse cardiovascular or renal outcomes, compared with medical therapy alone.[3]

Revascularization can be considered in patients with uncontrolled blood pressure refractory to medical therapy or with fluid retention or breathlessness from renal or congestive heart failure, or those with progressive, asymptomatic decline in renal or cardiac function. Resistant hypertension may require a 3-drug regimen comprising an angiotensin-converting-enzyme inhibitor or angiotensin-II receptor blocker, a calcium-channel blocker and a thiazide.[2]

References

1. Funder JW, Carey RM, Mantero F, et al. The management of primary aldosteronism: case detection, diagnosis, and treatment: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab* 2016;101:1889-916.
2. Carey RM, Calhoun DA, Bakris GL, et al. Resistant hypertension: detection, evaluation, and management: a scientific statement from the American Heart Association. *Hypertension* 2018;72:e53-90.
3. Gupta R, Assiri S, Cooper CJ. Renal artery stenosis: new findings from the CORAL trial. *Curr Cardiol Rep* 2017;19:75. doi: 10.1007/s11886-017-0894-2.

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