SHORT REPORT

A Rare Case of Aortic Dissection and Primary Hyperaldosteronism

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Introduction

Type-B aortic dissection can be fatal and cause significant morbidity amongst survivors.1 It is most commonly seen in hypertensive patients. Hyperaldosteronism is a recognized cause of non-essential hypertension and is associated with higher cardiovascular complication rates than would be expected with hypertension alone.2 We report a rare triad of aortic dissection, hypertension and an aldosterone-secreting adrenal tumour. To our knowledge, there are seven other reported cases of aortic dissection in patients with primary hyperaldosteronism.3

Case Report

A 39-year-old Romanian man presented with left-sided renal angle pain. He was a smoker and had been diagnosed with hypertension 10 years previously. He took no anti-hypertensive or other medications. His blood pressure (BP) on presentation was 180/110 mmHg. Clinical examination was

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otherwise unremarkable. Initial biochemical results demonstrated hypokalaemia (serum potassium: 2.5 mmol/l), normal random serum cortisol (126 nmol/l), elevated plasma aldosterone (680 pmol/L), suppressed plasma renin activity (0.2 pmol/mL/hour) and elevated aldosterone:renin ratio (3400). A diagnosis of primary hyperaldosteronism was made. A computed tomography (CT) scan demonstrated a left-sided adrenal mass consistent with adrenal adenoma. He was commenced on an aldosterone-antagonist and discharged. The patient’s General Practitioner continued to be responsible for BP modification.

Six months later he re-presented with low back pain, hypertension (240/100 mmHg) and pyrexia. CT angiography demonstrated an acute aortic dissection distal to the origin of the left subclavian artery and extending to the left common iliac and right external iliac arteries respectively (i.e. Stanford type-B or DeBakey type-3; Fig. 1) Transoesophageal echocardiogram confirmed the absence of a Stanford type-A proximal extension, despite retrograde intramural haematoma. In the absence of organ malperfusion, chest pain, or aneurysmal dilatation, the decision was made to opt for medical management. BP was controlled (<130 mmHg systolic) with intravenous b-blockade and nitrate infusions, and subsequently with an oral regimen of an aldosterone-antagonist, calcium-channel and b-blockade (Fig. 2).

Seven months later the patient underwent elective laparoscopic adrenalectomy; histology confirmed the diagnosis of an adrenal adenoma. The patient’s BP has since been tightly controlled on two anti-hypertensives at diminished doses compared with pre-operative requirements. The patient has entered a six-monthly imaging surveillance program, which has shown the aortic diameter remains stable and sub-threshold for intervention at 4.4 cm.

Discussion

The incidence of aortic dissection is approximately 3.0 per 100,000 per year.1 Refractory hypertension causes pressure-induced damage to the intimal-medial layers of the arterial wall, rendering the vessel more vulnerable to dissection. Hyperaldosteronism is rare cause of hypertension and usually asymptomatic but it should be considered, particularly in young, hypokalaemic, or treatment resistant patients. Excess aldosterone causes hypertension primarily via sodium retention and increased circulating volume, and increasing sympathetic activity. At high circulating levels, it has been suggested that aldosterone acting at mineralocorticoid receptors mediates endocardial dysfunction, myocardial fibrosis and cardiac remodelling.4 It may also have a direct effect on matrix metalloproteinases and their inhibitors leading to altered collagen content, altered collagen/elastin ratios and medial necrosis, predisposing the vessel to dissection.3

Accurate diagnosis, using biochemical analysis and cross-sectional imaging (CT or MRI) is vital, as cases of unilateral aldosterone hypersecretion are amenable to surgical management whereas bilateral adrenocortical hyperplasia is not.4,5 Surgery is the treatment of choice for proven aldosterone-secreting adenomas.

Aortic dissection is most commonly seen in the 5th–7th decades following many years of degenerative, hypertensive changes. This patient’s young age adds weight to the
hypothesis that another pathological process, beyond hypertension alone, is contributing.

**Conclusion**

Primary hyperaldosteronism is a rare cause of hypertension but should be considered in patients with resistant hypertension or atypical groups, such as those aged <40 years or advanced vascular disease. Although the triad of hypertension, aldosterone-secreting adenoma and aortic dissection is a rare one, early recognition and definitive management can eliminate the need for a complex vascular intervention and improve outcomes.\(^1,4\) This is especially important for young patients.

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None.

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**References**