Coexisting renal artery stenosis and primary aldosteronism

T. A. Chowdhury and S. S. Lasker

Department of Medicine, Queen Elizabeth Hospital, Edgbaston, Birmingham, UK

Key words: secondary hypertension; primary aldosteronism; renal artery stenosis

Introduction

Onset of marked hypertension at a young age raises the possibility of secondary hypertension. Primary aldosteronism accounts for approximately 0.4% of all cases of hypertension, whilst renal arterial disease accounts for around 2% [1]. We report the case of a young woman presenting with marked hypertension, with signs of end-organ damage, who had coexisting primary aldosteronism and renal artery stenosis, the latter being unmasked when the former had been treated.

Case report

A 22-year-old female had presented to her general practitioner 6 months earlier with a short history of intermittent throbbing headaches and blurred vision. Her general practitioner had noted she was hypertensive on three separate occasions within 6 months, and referred her to the medical clinic for assessment. On examination she was not cushingoid, and was hypertensive with a blood pressure of 195/95 mmHg, equal in both arms. Grade 2 hypertensive changes were seen in her fundi. There were no cardiac murmurs and no radiofemoral delay. Renal bruits could not be heard. Twenty-four-hour blood pressure monitoring confirmed marked sustained hypertension (systolic blood pressure 192–214 mmHg, diastolic blood pressure 91–123 mmHg).

Routine investigations showed normal urea and electrolytes, including a normal serum potassium of 4.0 mmol/l (3.5–5.5 mmol/l). Electrocardiography showed evidence of mild left ventricular hypertrophy on voltage criteria. Chest radiography showed no cardiomegaly or rib notching.

Screening tests for primary aldosteronism and Cushins syndrome were undertaken. The 9 a.m. cortisol after 2 mg dexamethasone the previous night was < 30 nmol/l. Supine plasma renin activity and plasma aldosterone were 0.13 nmol/l (0.39–2.03 nmol/l) and 780 pmol/l (28–445 pmol/l) respectively. Erect plasma renin activity and plasma aldosterone were 0.22 nmol/l (0.76–3.2 nmol/l) and 1450 pmol/l (110–860 pmol/l) respectively. These results were consistent with a diagnosis of primary aldosteronism.

The patient underwent computerized tomography of the abdomen, which showed a 1.5-cm mass within the right adrenal gland, but no other abnormalities. She underwent a laparotomy, where the right adrenal mass was excised. Her recovery was uneventful, although her blood pressure remained elevated at 180/90 mmHg. Histological examination of the mass showed an aldosterone-producing benign adenoma.

At subsequent outpatient review 2 months later, the patient’s blood pressure remained elevated at 176/90, and she remained symptomatic. Repeat supine plasma renin activity and serum aldosterone showed levels of 0.46 nmol/l and 119 pmol/l respectively, and erect values were 0.89 nmol/l and 156 pmol/l respectively. This indicated that her primary aldosteronism was effectively cured. She was thus readmitted for further investigation. Transthoracic echocardiography showed no evidence of coarctation of the aorta. Renal angiography was undertaken, and showed a single tight stenosis of the left renal artery compatible with fibromuscular hyperplasia. She proceeded to angioplasty at the same sitting as the angiography, with good effect.

By discharge, blood pressure had reduced to 160/80 mmHg, and at recent review was 155/72 mmHg. The patient remains asymptomatic and well 6 months after angioplasty.

Discussion

Hypertension secondary to renal or endocrine disease is more frequently recognized as an important cause of hypertension, particularly in youth. Renal arterial disease is most commonly due to fibromuscular hyperplasia in the younger age group, and atherosclerotic disease in older subjects. It is estimated that 2% of hypertensive subjects may have renal arterial disease,
and may be cured of hypertension when the condition is corrected. With the advent of angiotensin-converting enzyme inhibitors as first line treatment for essential hypertension, renal artery stenosis is becoming more frequently recognized when a patient suffers an acute deterioration in renal function on commencing these drugs.

Adrenocortical adenomas are also a well-recognized cause of hypertension. The original report of primary aldosteronism from Conn suggested that hypokalaemia was a prerequisite for the diagnosis of the condition [2]. Subsequent reports, however, suggest that hypokalaemia is only a late feature of the disease, and is not necessary to enable a diagnosis to be made [3]. The diagnosis of the condition rests on the demonstration of a suppressed plasma renin activity in the presence of an elevated plasma aldosterone, or indeed an elevation of the aldosterone:renin ratio [4].

Concomitant primary aldosteronism and renal artery stenosis is rare, but has been previously reported, although the renal artery disease is often atherosclerotic [5–7]. The coexistence of an aldosterone-producing adenoma with fibromuscular hyperplasia of the renal artery has not been previously reported. The possibility of the existence of ‘tertiary hyperaldosteronism’ has been hypothesized, whereby prolonged stimulation of the adrenal gland leading to secondary aldosteronism by excess angiotensin II (as seen in renal artery stenosis), could lead to an autonomously functioning adrenal adenoma—a similar scenario to tertiary hyperparathyroidism [8]. However, on screening a large cohort of patients with primary aldosteronism, Beever and colleagues found evidence of renovascular disease in only 7.4%, suggesting that tertiary hyperaldosteronism is unlikely to be a common phenomenon.

Coexistent renal arterial disease in primary aldosteronism may complicate the diagnosis of both conditions. In our patient, however, the diagnosis of primary aldosteronism was relatively clear on biochemical investigation. Furthermore, visualization of an adrenal adenoma on computerized tomography made the diagnosis clear. The fact that the renal artery stenosis was only unmasked after excision of the aldosterone-producing adenoma suggests that it may have initially contributed less to the patient’s marked hypertension. There remains the possibility that the renal artery stenosis was a sequel of the surgery to remove the adenoma. This however seems unlikely as the stenosis was found on the contralateral kidney, and hence trauma during surgery would not be expected.

Although two causes of secondary hypertension have been treated in this patient, she is not normotensive for her age, and one would have hoped that her hypertension may have settled completely. However, secondary hypertension may become irreversible in some circumstances, particularly when prolonged. Although this patient was only symptomatic for 6 months prior to treatment, she did present with evidence of end-organ damage (hypertensive retinopathy changes and left ventricular hypertrophy), and hence may have had hypertension for some years prior to diagnosis. It is anticipated that if her blood pressure remains moderately elevated in the future, she will eventually require medical treatment for the hypertension.

In summary, we present a young patient with severe hypertension, secondary to an aldosterone-producing adrenal adenoma, and coexisting renal artery stenosis probably due to fibromuscular hyperplasia.

References
2. Conn JW. Primary aldosteronism, a new clinical entity. *J Lab Clin Med* 1955; 45: 3–17

Received for publication: 8.7.97
Accepted: 14.7.97