

Secondary hypertension

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Abstract

Secondary hypertension is rare and the diagnosis may be challenging although, on occasion, there are clinical features indicative of a specific underlying cause. The more commonly encountered causes include renal parenchymal and vascular disease, phaeochromocytoma, endocrine causes, sleep apnoea and drugs.

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Introduction

More than 90% of all patients (some say 95%) with arterial hypertension will have essential hypertension. Secondary hypertension is therefore much less common. The problem for the practitioner is that it is not always easy to suspect the diagnosis of secondary hypertension although, on occasion, there are clinical features indicative of a specific secondary cause.

The decision to investigate for secondary causes of hypertension may be made when:

- The young and the old present with hypertension.
- Blood pressure responds poorly to drug treatment (resistant hypertension).
- Blood pressure begins to rise after being previously well controlled (first checks should be made for non-compliance).
- There is sudden onset of hypertension.

Nevertheless, there is no clear guidance on exactly when to investigate for secondary causes. A brief overview will be given of the more important causes of secondary hypertension.

Renal parenchymal disease

This is the most common cause of secondary hypertension. In this group of conditions, ultrasound of the kidneys is critical for diagnosis. Clinically coexistent hypertension and palpable masses in the abdomen should prompt an abdominal ultrasound to look for polycystic kidney disease. Obstructive uropathy will also be diagnosed by this modality.

Clinically the detection of small kidneys due to end-stage renal parenchymal disease is not possible, but abnormal urine, i.e. proteinuria or haematuria, should be a red flag to perform an ultrasound to assess kidney size. A difficulty is that proteinuria and reduced estimated glomerular filtration rate (eGFR) may also be caused by target organ damage as a result of pre-existent hypertension, while the former may also be the cause of the latter (hypertension, due to renal parenchymal disease); a typical chicken-and-egg scenario.

Renovascular hypertension

In young people, fibromuscular dysplasia, and in the elderly atherosclerosis of renal blood vessels as they originate from the aorta, is the most common cause of renovascular hypertension: both cause stenosis of extra-renal arteries and compromise blood supply to the kidneys.

The only possible clinical clue to this condition is an abrupt onset of hypertension, or increasing difficulty to control blood pressure in the hypertensive. The findings of an abdominal bruit on auscultation, hypokalaemia and progressive decline in renal function would increase suspicion of the condition, but are commonly absent.

Some form of imaging of renal arteries is necessary for diagnosis. Revascularisation decisions are always difficult and need expert advice.

Phaeochromocytoma

This is relatively rare and may be inherited or acquired. The associated hypertension may be stable or paroxysmal, presenting with headache, sweating, pallor and palpitations.

The diagnosis is established by demonstrating an increase in plasma or urinary catecholamines or their metabolites. Very high values are sufficiently diagnostic, but in cases of slight or moderate elevations, further tests are required. Elevated chromogranin A levels will assist in making the diagnosis. After the diagnosis has been made, localisation of the tumour is required.

Primary aldosteronism

This condition is of increasing importance worldwide. Low serum potassium (not caused by a diuretic) is the ideal screening test, but few patients have hypokalaemia early in the disease.

About 30% of cases are caused by a tumour (Conn syndrome) and 70% by adrenal hyperplasia. Carcinoma is a rare cause.

Aldosterone antagonists are important and effective as therapy. Blood pressure is typically resistant to treatment and primary hyperaldosteronism should be considered in all patients with refractory hypertension. The usefulness of aldosterone:renin ratios in diagnosis is still debated.

Cushing syndrome

Hypertension occurs in about 80% of cases. The syndrome is suggested by the typical body configuration (central obesity phenotype). The measurement of 24-hour urinary cortisol excretion is the most practical and reliable diagnostic test. When positive, other confirmatory tests are required.

Rare endocrine causes

These include hyperparathyroidism, acromegaly, congenital hyperplasia, 11-beta-hydroxysteroid dehydrogenase deficiency, hypothyroidism and thyrotoxicosis.

Obstructive sleep apnoea

It is important to consider obstructive sleep apnoea in patients suffering from resistant hypertension, especially when obese. Nevertheless, independent of obesity, hypertension occurs in more than 50% of patients with obstructive sleep apnoea. Hypertensive patients diagnosed as "non-dippers" on 24-hour ambulatory blood pressure monitoring should also be tested for obstructive sleep apnoea.

Weight loss and positive-pressure breathing equipment ameliorate the effects of the syndrome and improve the control of blood pressure considerably.

Coarctation of the aorta

This is the most common congenital cardiovascular cause of hypertension. The diagnosis is made on clinical examination by measuring the blood pressure in the arms and legs. Normally blood pressure levels in the legs is higher than the arms; in coarctation, the leg blood pressure is considerably lower than that measured in the arms. The femoral pulses may be absent.

Drug-induced hypertension

There are many substances and drugs that raise blood pressure. Nonsteroidal anti-inflammatory drugs (both COX-1 and COX-2) are important. Other potential culprits include corticosteroids, anabolic steroids, contraceptives, cocaine, erythropoietin, ciclosporin, and sympathomimetic drugs. Excessive alcohol intake probably remains the most common cause.

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