

Secondary Hypertension: Contemporary Approach to Diagnosis and Management

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ABSTRACT

Background: Secondary hypertension accounts for approximately 5–10% of all cases of elevated blood pressure and arises from an identifiable underlying cause. Recognition is critical, as targeted therapy may restore normotension or substantially reduce the burden of antihypertensive treatment and associated cardiovascular and renal complications.

Methods: This narrative review summarises the current epidemiology, diagnostic workup, and management of the major causes of secondary hypertension, incorporating recent international guidelines and advances in hormonal assays, cross-sectional imaging, and genetic sequencing.

Results: Primary aldosteronism is the most common aetiology, estimated to affect 5–14% of hypertensive individuals. Other important causes include renovascular disease, chronic kidney disease, endocrinopathies (Cushing syndrome, pheochromocytoma, thyroid and parathyroid disorders, acromegaly), obstructive sleep apnoea, monogenic syndromes, vasculitis, coarctation of the aorta, and medication-induced hypertension.

Conclusion: Secondary hypertension encompasses a diverse and potentially reversible spectrum of conditions. Targeted investigation guided by clinical features minimises unnecessary testing and improves diagnostic yield. Future directions include integration of genomic profiling, precision therapeutics, and artificial intelligence-driven screening algorithms.

Keywords: secondary hypertension; primary aldosteronism; renovascular hypertension; pheochromocytoma; monogenic hypertension; obstructive sleep apnoea; endocrine hypertension; chronic kidney disease

INTRODUCTION

Secondary hypertension, present in approximately 5–10% of hypertensive patients, is defined by an identifiable and potentially reversible underlying cause.^{1,2} Early detection is critical, as targeted therapy may restore normotension or substantially simplify pharmacological control, thereby reducing downstream cardiovascular and renal complications.

Clinical pointers suggestive of a secondary aetiology include resistant hypertension, onset before 30 years of age, family history of early-onset hypertension, abrupt worsening of previously stable blood pressure, spontaneous hypokalaemia, adrenal incidentalomas, or features of endocrine or vasculitic disease.^{1,2} Indiscriminate screening in the absence of such pointers risks false-positive results and should be avoided.

This review summarises the epidemiology, diagnostic approach, and management of the major causes of secondary hypertension (Table 1).

Table 1. Common causes of secondary hypertension and initial screening tests.

Category	Common Causes	Initial Screening Tests
Endocrine causes	Primary aldosteronism, Cushing syndrome, pheochromocytoma, thyroid/parathyroid disorders	Plasma aldosterone/renin ratio, dexamethasone suppression test, plasma/urine metanephrines, TSH, calcium/PTH
Renal parenchymal disease	Chronic glomerulonephritis, diabetic nephropathy, polycystic kidney disease	Serum creatinine/eGFR, urinalysis, urine albumin:creatinine ratio, renal ultrasound
Renovascular disease	Atherosclerotic renal artery stenosis, fibromuscular dysplasia	Duplex Doppler ultrasound, CT or MR angiography, plasma renin activity
Sleep-related disorders	Obstructive sleep apnoea	Sleep study (polysomnography)
Drugs and toxins	NSAIDs, oral contraceptives, steroids, cyclosporine, cocaine, liquorice	Medication history, review of substances
Vascular/inflammatory	Coarctation of aorta, vasculitis (e.g. PAN, Takayasu)	Blood pressure in all four limbs, echocardiography, CT/MR angiography, inflammatory markers
Genetic/monogenic	Liddle syndrome, Gordon syndrome, apparent mineralocorticoid excess, glucocorticoid-remediable aldosteronism	Plasma renin and aldosterone, genetic testing

ENDOCRINE CAUSES

Primary Aldosteronism

Primary aldosteronism (PA), also known as Conn's syndrome, is characterised by autonomous aldosterone secretion independent of the renin–angiotensin system, resulting in hypertension and suppressed plasma renin.³ Contrary to historical teaching, hypokalaemia is present in only approximately 30% of cases, and even then potassium levels tend to be near the lower limit of normal.⁴ PA is now recognised as one of the most common

causes of secondary hypertension, with a prevalence of 5–14% in general hypertensive populations⁵ and potentially 20–30% in referral centres among patients with resistant hypertension.⁶

Screening

Screening is recommended for all patients with clinical red flags. The 2025 Endocrine Society guidelines additionally advocate screening all individuals with hypertension, an approach shown to be cost-effective.³ Screening uses the plasma aldosterone-to-renin ratio (ARR), with a positive result indicated by suppressed plasma renin activity (<1 ng/mL/h) and an ARR >20 (>30 being highly predictive). If plasma renin concentration (PRC) is used, an ARR >70 is highly predictive. Specimens should be collected in the morning with the patient seated and well hydrated, potassium repleted, and dietary sodium restriction avoided in the preceding days.

Mineralocorticoid receptor antagonists, beta-blockers, and centrally acting alpha-2 agonists should be withdrawn two to four weeks before testing and substituted with agents such as hydralazine, prazosin, or moxonidine as required. ACE inhibitors and ARBs need not be routinely withdrawn, as suppressed renin whilst on these agents is itself highly suggestive of hyperaldosteronism. A recent trial demonstrated that steroidomics-based screening may offer a drug-independent diagnostic approach for PA in patients in whom withdrawal of antihypertensive therapy is not feasible.⁷

Diagnosis

A positive ARR should be followed by aldosterone suppression testing to confirm autonomous secretion. Four protocols are available: oral sodium loading, intravenous saline infusion, the captopril challenge test, and the fludrocortisone suppression test.^{3,8} Oral sodium loading or intravenous saline are most commonly used; failure to suppress aldosterone confirms the diagnosis. This step may be omitted when the ARR is markedly elevated alongside compelling clinical features.

Lateralisation of aldosterone excess must then be established in surgical candidates. Steps include adrenal CT — interpreted cautiously, as small adenomas may be missed and up to 10% of elderly patients harbour non-functioning incidentalomas⁹ — followed by adrenal vein sampling (AVS), the gold standard for lateralisation, which should be performed in all surgical candidates except those with a large (>1 cm) unilateral adenoma on CT in a young patient with hypokalaemia and markedly elevated ARR. Imaging characteristics of adrenal lesions are summarised in Table 2.

Management

Laparoscopic adrenalectomy is recommended for patients with a unilateral aldosterone-producing adenoma who are suitable surgical candidates. For those unsuitable for surgery or with bilateral hyperplasia, a mineralocorticoid receptor antagonist is indicated. Spironolactone (12.5 mg daily, titrated to 100 mg) is preferred based on availability and cost, though gynaecomastia may occur at higher doses. Eplerenone (25 mg daily) is an alternative, albeit less accessible in Australia. Surgical management is associated with fewer antihypertensive agents at lower doses in the long term.¹⁰

Table 2. Imaging characteristics of adrenal lesions.³⁰

Lesion	CT Findings	MRI Findings	Functional Features
Adrenal adenoma	<10 HU on unenhanced CT; rapid contrast washout	Iso/hypointense on T1; hyperintense on T2; signal loss on opposed-phase imaging	Non-functioning or cortisol/aldosterone secreting
Phaeochromocytoma	Heterogeneous, >10 HU, strong enhancement	Markedly hyperintense on T2 ("light-bulb bright")	Catecholamine-secreting
Adrenal carcinoma	Irregular, >4 cm, necrosis/calcification, delayed washout	Heterogeneous signal	May secrete cortisol or androgens
Metastasis	Bilateral, variable density	Variable T1/T2; no signal drop-out	Non-functioning; history of malignancy

Cushing Syndrome

Cushing syndrome results from prolonged exposure to excess cortisol, most commonly from exogenous glucocorticoid use. Endogenous causes include ACTH-secreting pituitary adenomas (Cushing disease), ectopic ACTH secretion (e.g. from small cell lung carcinoma), and primary adrenal pathology.

The classical phenotype comprises central adiposity, dorsocervical fat pad, moon facies, proximal myopathy, and wide violaceous striae. Hypertension occurs in approximately 75–85% of patients and represents one of its most consistent cardiovascular manifestations.¹¹ Mechanisms include enhanced vascular sensitivity to catecholamines and angiotensin II, up-regulation of mineralocorticoid receptors, and suppression of vasodilatory mediators including nitric oxide. Ectopic ACTH production may additionally cause severe hypokalaemia and hypertension through stimulation of aldosterone production.

Screening

Biochemical screening should be undertaken when exogenous steroid use has been excluded and there is clinical suspicion supported by phenotypic features. To minimise false-positive results, two of the following tests should be performed, tailored to the individual patient: (1) 24-hour urinary free cortisol (interpret with caution in renal impairment; avoid if eGFR <30 mL/min); (2) low-dose (1 mg) overnight dexamethasone suppression test (may be falsely abnormal with elevated cortisol-binding globulin, e.g. in patients on the oral contraceptive pill); and (3) late-night salivary cortisol (may be falsely elevated in shift workers with disrupted circadian rhythm).¹¹

Diagnosis

Positive screening tests should prompt determination of the source of cortisol excess. In approximately 80% of cases, hypercortisolaemia is ACTH-dependent, attributable either to Cushing disease or to ectopic ACTH secretion. The remaining 20% arise from ACTH-independent adrenal pathology.^{1,11}

Serum ACTH measurement guides further investigation. Equivocal or elevated levels (10–20 pg/mL or >20 pg/mL, respectively) prompt high-dose dexamethasone suppression testing; suppression confirms pituitary-driven disease, which is then localised by pituitary MRI. If the adenoma is absent or <6 mm, bilateral inferior petrosal sinus sampling is performed to confirm a central-to-peripheral ACTH gradient. Failure of suppression on high-dose dexamethasone testing indicates

ectopic ACTH secretion, and dedicated imaging to identify the source should follow. Suppressed ACTH (<10 pg/mL) indicates primary adrenal pathology, prompting dedicated adrenal imaging.^{2,11}

Management

Treatment centres on surgical resection of the ACTH- or cortisol-producing lesion. Medical therapy with steroidogenesis inhibitors (ketoconazole, metyrapone, osilodrostat) may be used preoperatively or when surgery is contraindicated. Blood pressure typically improves following biochemical cure, though residual vascular remodelling and metabolic risk may persist.^{1,11}

Phaeochromocytoma

Phaeochromocytoma is a rare catecholamine-secreting neuroendocrine tumour arising predominantly from the adrenal medulla (80–85%) or from extra-adrenal sympathetic paraganglia. Its global incidence is approximately 1.9 new cases per million person-years.¹²

The classical presentation — episodic hypertension, headache, diaphoresis, and tachycardia — is well recognised but not invariable. Approximately 30–40% of cases are associated with hereditary syndromes, including von Hippel–Lindau disease, Multiple Endocrine Neoplasia type 2 (MEN2), Neurofibromatosis type 1, and succinate dehydrogenase subunit syndromes (Table 3).¹³ Genetic testing should be considered in young patients, those with bilateral disease, a positive family history, or disease recurrence after resection.

Table 3. Genetic syndromes associated with phaeochromocytoma.

Syndrome	Gene	Inheritance	Associated Features
MEN 2A / MEN 2B	RET	Autosomal dominant	Medullary thyroid carcinoma, parathyroid hyperplasia (MEN 2A), mucosal neuromas (MEN 2B)
Von Hippel–Lindau (VHL)	VHL	Autosomal dominant	Haemangioblastomas, renal cell carcinoma, pancreatic cysts
Neurofibromatosis Type 1	NF1	Autosomal dominant	Café-au-lait spots, neurofibromas, optic glioma
Paranglioma syndromes (PGL)	SDHD, SDHB, SDHC, SDHAF2	Autosomal dominant	Parangliomas (head, neck, thorax), renal tumours
TMEM127 / MAX mutations	TMEM127, MAX	Autosomal dominant	Isolated or bilateral phaeochromocytomas

Diagnosis

In patients with high clinical suspicion, plasma free metanephrines are the first-line test (sensitivity 96–100%, specificity 85–100%); a result greater than twice the upper limit of normal is considered positive. In patients with a lower index of suspicion, 24-hour urinary fractionated metanephrines and catecholamines offer higher specificity (98–100%).¹⁴ Specimens should be collected supine, in the absence of acute

illness, physical stress, stimulants, or interfering medications (e.g. tricyclic antidepressants, which should be discontinued at least two weeks prior).

Following biochemical confirmation, CT or MRI localises the tumour (both 98–100% sensitive, approximately 70% specific). If non-diagnostic, ⁶⁸Ga-DOTATATE PET or MIBG scintigraphy may identify small or metastatic lesions.¹⁴

Management

Definitive treatment is surgical resection, typically laparoscopic adrenalectomy. Adequate preoperative alpha-adrenergic blockade (phenoxybenzamine or a selective alpha-1 blocker) is essential, followed by beta-blockade if required, to mitigate intraoperative haemodynamic instability.

Other Endocrine Causes

Hypothyroidism

The relationship between hypothyroidism and hypertension is debated.¹⁵ Reduced thyroid hormone levels lead to decreased cardiac output, increased systemic vascular resistance, arterial stiffness, and impaired endothelial function, which may predispose to diastolic hypertension. Whilst early observational studies suggested a blood pressure benefit with thyroid hormone replacement, a subsequent randomised trial in elderly patients with subclinical hypothyroidism demonstrated no effect on blood pressure.¹⁶

Hyperparathyroidism

Hypertension is reported in 40–65% of patients with hyperparathyroidism, though routine screening is not recommended in the absence of hypercalcaemia. Proposed mechanisms include PTH-mediated increases in intracellular vascular smooth muscle calcium, enhanced vasoconstriction, endothelial dysfunction, and altered renal sodium handling. Parathyroidectomy may modestly lower blood pressure in selected patients, though effects are variable.¹⁷

Acromegaly

Hypertension is reported in 18–60% of patients with acromegaly. Excess growth hormone and IGF-1 promote an anti-natriuretic state and increased peripheral vascular resistance. Routine screening for acromegaly is not performed in suspected secondary hypertension in the absence of other clinical manifestations. Successful treatment — surgical or medical — is often associated with partial amelioration of hypertension.¹⁸

RENOVASCULAR HYPERTENSION

Renovascular hypertension results from reduced renal perfusion and consequent overactivation of the renin–angiotensin system (RAS). The two principal aetiologies are atherosclerotic renal artery stenosis and fibromuscular dysplasia (FMD).

Atherosclerotic Renal Artery Stenosis

Atherosclerotic disease is the most common cause of renovascular hypertension in older adults, typically affecting the ostia or proximal segments of the renal arteries and accounting for up to 10–15% of cases of resistant hypertension.¹⁹ It should be suspected in patients with intolerance to ACE inhibitors or ARBs (rise in serum creatinine >30% or precipitous fall in blood pressure), asymmetric kidney sizes, onset of severe hypertension

after age 55, or a unilateral abdominal bruit.²⁰

Diagnostic evaluation includes duplex Doppler ultrasound, CT or MR angiography, or invasive angiography if revascularisation is being considered. Stenosis characteristically involves the proximal 2 cm of the renal artery origin. First-line management is aggressive medical therapy comprising RAAS blockade, statins, antiplatelet agents, and cardiovascular risk factor modification. Randomised trials have not demonstrated a survival benefit from revascularisation over optimal medical therapy; however, selected patients — including those with recurrent flash pulmonary oedema due to bilateral stenosis, rapidly declining renal function, refractory hypertension with a solitary kidney (e.g. transplant renal artery stenosis), or intolerance to medical therapy — may benefit from percutaneous intervention.²⁰

Fibromuscular Dysplasia

FMD is a non-inflammatory, non-atherosclerotic arteriopathy most commonly affecting the bilateral renal arteries and extra-renal medium vessels such as the carotid arteries. The pathognomonic ‘string-of-beads’ appearance on angiography reflects alternating medial fibrosis and aneurysmal dilatation. Unlike atherosclerotic disease, the stenosis in FMD is always distal to the proximal 2 cm of the renal artery origin.

FMD is an important cause of renovascular hypertension in younger women (aged 15–50 years) without conventional atherosclerotic risk factors, with a prevalence of 4–6% in the hypertensive population.²¹ Balloon angioplasty without stenting is the preferred intervention for focal lesions, with normalisation or substantial improvement in blood pressure achievable in the majority of patients. Long-term follow-up is required, as restenosis or contralateral disease develops in approximately 10–20% of cases.^{1,21} Screening for extra-renal vascular involvement — particularly of the internal carotid, superior mesenteric, and iliac arteries — is also advised.

KIDNEY DISEASE

Acute and chronic kidney disease represent one of the most frequent causes of secondary hypertension. Mechanistically, hypertension in CKD results from impaired sodium and water excretion, RAAS activation, and increased sympathetic nervous system activity attributable to reduced nephron mass and ischaemic injury.²⁴ Both glomerular and tubulointerstitial disorders can lead to progressive nephron loss and salt-sensitive hypertension. In turn, hypertension accelerates renal damage, creating a vicious cycle of nephrosclerosis and declining GFR.²⁴

OBSTRUCTIVE SLEEP APNOEA

Obstructive sleep apnoea (OSA) is a major and often underappreciated secondary cause of hypertension, affecting up to 30–50% of hypertensive individuals and present in nearly 70% of those with resistant hypertension. Recurrent upper airway obstruction during sleep causes intermittent hypoxia, hypercapnia, and cortical arousal, triggering sustained sympathetic nervous system activation, oxidative stress, endothelial dysfunction, and systemic inflammation — all of which promote persistent blood pressure elevation. The characteristic pattern is nocturnal or non-dipping hypertension, though daytime pressures rise with progressive disease.

Management has traditionally included continuous positive airway pressure (CPAP) therapy, mandibular advancement devices, and weight loss. A recent landmark trial demonstrated that tirzepatide significantly improved sleep-disordered breathing outcomes and body weight in patients with OSA and obesity, with potential downstream benefits on blood pressure.²⁵

MONOGENIC SYNDROMES AND TUBULOPATHIES

Monogenic forms of hypertension are rare but clinically important, and should be considered in young patients with a strong family history of early-onset hypertension and electrolyte abnormalities. Genetic testing via a 'hypertension and aldosterone disorders panel' is available in Australia under Medicare. Informed consent is required given implications for family planning and the possibility of variants of unknown significance (VUS).

Metabolic Alkalosis and Hypokalaemia

Familial Hyperaldosteronism

Familial hyperaldosteronism (FH) is inherited in an autosomal dominant manner. Four subtypes are recognised: Type I (Glucocorticoid-Remediable Aldosteronism) results from a chimeric CYP11B1/CYP11B2 gene, causing ACTH-mediated aldosterone excess that is suppressible with glucocorticoids. Type II is caused by CLCN2 variants encoding a voltage-gated chloride channel in the zona glomerulosa, resulting in autonomous aldosterone overproduction. Type III is caused by KCNJ5 variants leading to sodium influx, cell depolarisation, and aldosterone secretion. Type IV is caused by CACNA1H variants resulting in increased calcium influx and aldosterone release.²⁶

Apparent Mineralocorticoid Excess (AME)

AME is a rare autosomal recessive disorder caused by loss-of-function mutations in HSD11B2, encoding 11 β -hydroxysteroid dehydrogenase type 2. In the absence of this enzyme, cortisol accumulates in renal tubular cells and activates the mineralocorticoid receptor, producing severe early-onset hypertension, hypokalaemia, metabolic alkalosis, and suppressed renin and aldosterone. An acquired form occurs with chronic liquorice ingestion via inhibition of the same enzyme. Diagnosis is confirmed by elevated urinary cortisol:cortisone metabolite ratios or HSD11B2 mutation testing. Treatment includes amiloride (ENaC blockade), glucocorticoid therapy, and strict avoidance of liquorice.^{26,27}

Liddle Syndrome

Liddle syndrome is an autosomal dominant condition caused by gain-of-function mutations in the ENaC beta or gamma subunit genes (SCNN1B, SCNN1G), resulting in constitutive sodium reabsorption in the distal nephron independent of aldosterone. This produces early-onset, low-renin, low-aldosterone hypertension with hypokalaemic metabolic alkalosis. Mineralocorticoid receptor antagonists are ineffective; treatment relies on direct ENaC inhibitors — amiloride or triamterene.^{26,27}

Metabolic Acidosis and Hyperkalaemia

Gordon Syndrome (Pseudohypoaldosteronism Type II)

Gordon syndrome is an autosomal dominant condition caused by mutations in WNK1, WNK4, CUL3, or KLHL3, leading to excessive NaCl reabsorption in the distal tubule, volume expansion, hypertension, and suppressed renin. Increased sodium reabsorption in the distal tubule reduces sodium delivery to potassium- and hydrogen-secreting cells of the cortical collecting duct, causing hyperkalaemia and metabolic acidosis — the biochemical mirror image of hyperaldosteronism. Gordon syndrome is uniquely responsive to thiazide diuretics, which directly correct both blood pressure and electrolyte abnormalities.²⁷

VASCULAR CAUSES

Vasculitis

Large and medium vessel vasculitis can produce renovascular hypertension by compromising renal perfusion and activating the renin–angiotensin axis, whilst small vessel vasculitis causes hypertension predominantly through glomerular pathology.

Polyarteritis Nodosa

Polyarteritis nodosa (PAN) is a systemic necrotising vasculitis of medium-sized arteries, commonly affecting the renal, mesenteric, and visceral vascular beds. Hypertension occurs in approximately half of affected patients, with arterial wall necrosis and disruption of the elastic lamina leading to aneurysmal dilatation and stenosis. Severe or rapidly progressive hypertension may be a presenting feature, particularly in association with systemic manifestations such as fever, weight loss, peripheral neuropathy, and abdominal pain. Comorbid hepatitis B infection is present in up to 20% of cases and should be treated. Management involves immunosuppression with corticosteroids and cyclophosphamide for severe disease.²²

Takayasu Arteritis

Takayasu arteritis is a large-vessel granulomatous vasculitis affecting the aorta and its major branches, including the renal arteries. It predominantly affects women of Asian origin aged 20–40 years. Hypertension may result from RAS activation due to renal hypoperfusion and from increased afterload associated with aortic inflammation. Disproportionate involvement of the aortic arch vessels may produce erroneously low blood pressure recordings in the upper limbs, masking systemic hypertension — hence blood pressure should be measured in all four limbs. Treatment of active disease involves corticosteroids and immunosuppressants; patients with flow-limiting stenoses causing hypertension or end-organ ischaemia may require revascularisation.²³

Coarctation of the Aorta

Coarctation of the aorta is a congenital narrowing typically at the aortic isthmus just distal to the left subclavian artery, producing a proximal–distal pressure gradient. Systemic arterial pressure above the constriction is chronically elevated, whilst distal perfusion is reduced. This activates the RAAS, promotes arterial stiffness and endothelial dysfunction, and causes vascular remodelling. Even following surgical or percutaneous repair, 20–70% of patients develop persistent or recurrent hypertension due to sustained vascular changes and altered baroreceptor physiology.²⁸

MEDICATION-INDUCED HYPERTENSION

Several commonly prescribed or over-the-counter medications can elevate blood pressure through sodium retention, sympathomimetic stimulation, or interference with antihypertensive efficacy. The most frequently implicated agents are NSAIDs, oestrogen-containing oral contraceptives, corticosteroids, antidepressants (particularly SNRIs), decongestants, calcineurin inhibitors, erythropoiesis-stimulating agents, and recreational drugs such as amphetamines and cocaine. Dietary licorice may also contribute. The reported prevalence of potentially blood pressure-raising medication use in hypertensive adults is 1–9%.²⁹ Withdrawal or substitution of the offending agent may normalise blood pressure, though this is not always clinically feasible, and optimisation of antihypertensive therapy remains necessary.

CONCLUSION

Secondary hypertension encompasses a diverse spectrum of potentially reversible conditions accounting for a clinically significant minority of hypertensive disease. Primary aldosteronism is particularly under-diagnosed and warrants active consideration in any patient meeting screening criteria. Identifying a secondary cause not only guides specific and effective management but also informs cardiovascular risk stratification and long-term prognosis. Advances in hormonal assays, high-resolution cross-sectional imaging, and readily accessible genetic testing have substantially refined diagnostic algorithms and facilitated earlier detection.

Future directions lie in the integration of genomic profiling, precision therapeutics, and artificial intelligence-driven screening algorithms to further refine detection and personalise treatment for patients with secondary hypertension.

DECLARATIONS

Conflict of Interest

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Author Contributions

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