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HYPERTENSIVE CRISIS IN ENDOCRINE AND RENAL DISORDERS: PATHOPHYSIOLOGY, CLINICAL MANIFESTATIONS, AND EVIDENCE-BASED MANAGEMENT

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Abstract

Hypertensive crisis represents a critical medical emergency characterized by severe blood pressure elevation (systolic ≥180 mmHg and/or diastolic ≥120 mmHg) with potential for acute target-organ damage. While essential hypertension accounts for approximately 95% of cases, secondary causes—particularly endocrine and renal disorders—contribute significantly to hypertensive emergencies and are potentially curable with timely intervention. Endocrine causes including pheochromocytoma, Cushing's syndrome, and primary aldosteronism account for nearly 3% of secondary hypertension cases, whereas renovascular and renoparenchymal diseases represent major non-endocrine secondary etiologies. This comprehensive narrative review examines the pathophysiological mechanisms linking endocrine and renal disorders to hypertensive crisis, delineates clinical manifestations and diagnostic approaches, and synthesizes current evidence-based management strategies. Understanding the interplay between hormonal dysregulation, renin-angiotensin-aldosterone system activation, and renal dysfunction is essential for clinicians to recognize secondary causes, initiate targeted therapy, and prevent life-threatening complications including stroke, myocardial infarction, and acute renal failure.

Keywords: Hypertensive crisis, endocrine hypertension, renal hypertension, pheochromocytoma, Cushing's syndrome, primary aldosteronism, chronic kidney disease, hypertensive emergency, secondary hypertension, target organ damage, renin-angiotensin-aldosterone system

1. Introduction

Hypertension affects approximately 1.28 billion adults worldwide, representing one of the most significant global health challenges of the 21st century. Among these individuals, hypertensive crisis—encompassing both hypertensive urgency and hypertensive emergency—

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poses immediate threats to patient survival and long-term organ function. The 2025 AHA/ACC Hypertension Guideline has strengthened recommendations for special populations, particularly those with diabetes and chronic kidney disease (CKD), emphasizing systolic blood pressure targets below 130 mmHg when tolerable.

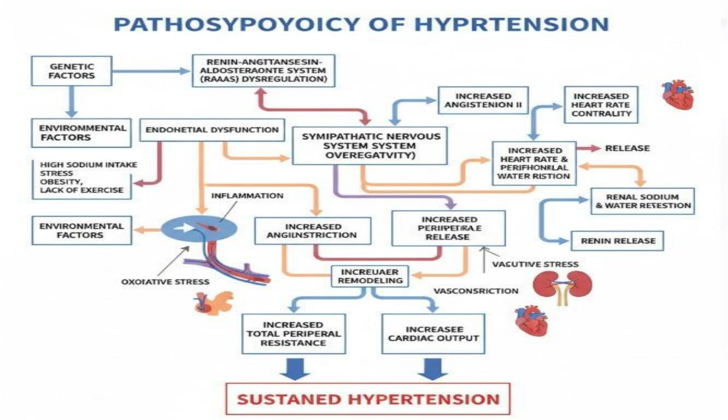
Hypertensive emergency is formally defined as a rapid and significant elevation of blood pressure with systolic pressure greater than 180 mmHg and/or diastolic pressure exceeding 120 mmHg, accompanied by evidence of acute target-organ damage affecting the brain, heart, kidneys, or vasculature. Endocrine and metabolic conditions account for less than 5% of all hypertensive emergencies; however, when present, they can precipitate severe morbidity and mortality. Prompt recognition remains paramount to initiate targeted therapy and avoid catastrophic complications such as stroke, acute coronary syndromes, and irreversible renal injury.

The kidneys and endocrine system maintain intricate physiological relationships governing blood pressure homeostasis. The renin-angiotensin-aldosterone system (RAAS), sympathetic nervous system, and various hormonal axes—including the hypothalamic-pituitary-adrenal axis and thyroid axis—collectively modulate vascular tone, sodium handling, and cardiac output. Dysfunction within any of these systems can precipitate severe, refractory hypertension that progresses to crisis levels. This review provides a comprehensive examination of hypertensive crisis in the context of endocrine and renal disorders, integrating current pathophysiological understanding with contemporary clinical management frameworks.

2. Pathophysiology of Hypertensive Crisis

2.1 General Mechanisms

The pathophysiology of hypertensive crisis involves a complex interplay between humoral vasoconstrictors, endothelial dysfunction, and impaired autoregulation. Abrupt rises in vascular resistance cause endothelial injury, disrupting the delicate balance between vasodilation and vasoconstriction. This vascular ischemic state further increases systemic vascular resistance, leading to pressure natriuresis and subsequent RAAS activation. The resulting vicious cycle perpetuates vascular injury across end organs including the brain, heart, kidneys, and retina.



A central concept in crisis management is autoregulation of blood flow, which ensures constant perfusion across varying arterial pressures. In normotensive individuals, autoregulation functions across mean arterial pressures (MAP) of approximately 50–150 mmHg. Chronic

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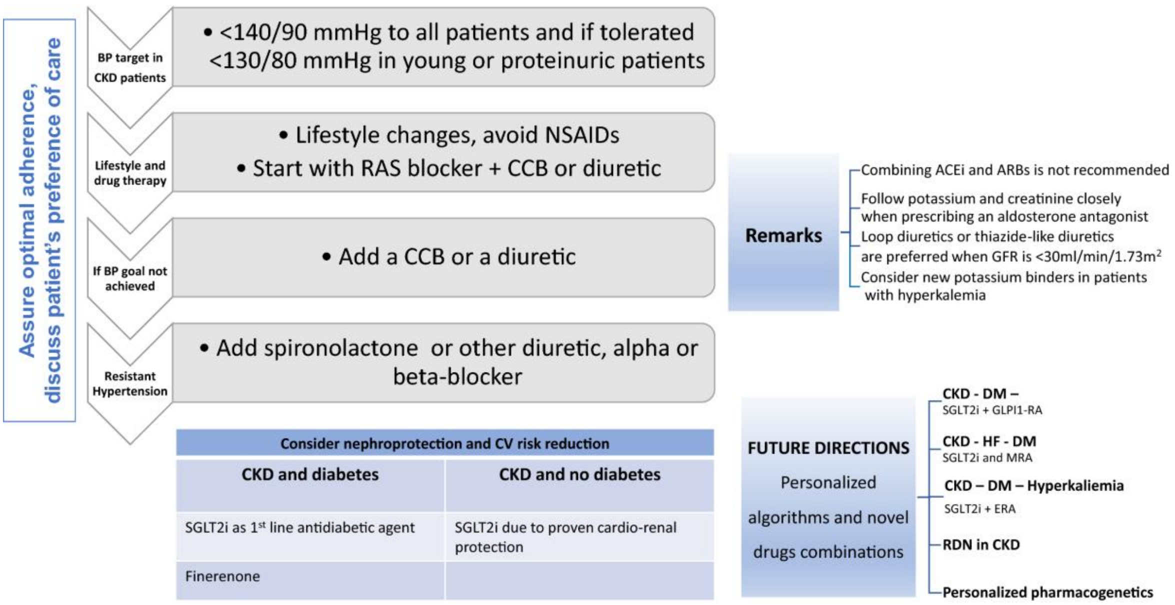
hypertension shifts this curve rightward, meaning organs cannot compensate for sudden pressure drops below their new autoregulatory threshold. In malignant hypertension, autoregulation itself becomes impaired, rendering patients vulnerable to ischemic injury from both excessive hypertension and overly aggressive treatment.

2.2 Endocrine Mechanisms

Endocrine hypertension arises from hormonal derangements that directly influence vascular tone, sodium retention, and cardiac contractility. Pheochromocytomas and paragangliomas secrete catecholamines (norepinephrine, epinephrine, and dopamine), producing intense α-adrenergic-mediated vasoconstriction and β-adrenergic-mediated tachycardia. Cushing's syndrome elevates circulating cortisol, which activates mineralocorticoid receptors, enhances vascular reactivity to catecholamines, and promotes sodium retention. Primary aldosteronism, now recognized as potentially affecting up to 20% of hypertensive patients, causes volume expansion through sodium retention and potassium wasting, with direct vascular remodeling effects independent of blood pressure elevation.

2.3 Renal Mechanisms

Renal parenchymal disease and renovascular hypertension represent major secondary causes of hypertensive crisis. In chronic kidney disease, multiple factors contribute to hypertension including RAAS upregulation, increased sympathetic tone, endothelial dysfunction, increased arterial stiffness, and enhanced salt sensitivity. Renovascular disease—whether from atherosclerosis (older individuals) or fibromuscular dysplasia (young women)—activates the RAAS through renal hypoperfusion, generating angiotensin II-mediated vasoconstriction and aldosterone-driven volume expansion. The bidirectional relationship between hypertension and kidney disease creates a self-perpetuating cycle where each condition worsens the other.



3. Endocrine Causes of Hypertensive Crisis

3.1 Pheochromocytoma and Paraganglioma

Pheochromocytomas are catecholamine-secreting neuroendocrine tumors arising from chromaffin cells of the adrenal medulla, while paragangliomas originate from extra-adrenal

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sympathetic ganglia. These tumors represent the quintessential endocrine cause of hypertensive crisis, with blood pressure patterns ranging from sustained hypertension with intermittent spikes to normotensive states with sudden, dramatic elevations reaching crisis levels.

The clinical triad of headache, excessive sweating, and palpitations typifies pheochromocytoma, though presentations vary considerably. Orthostatic hypotension occurs in more than 50% of patients due to volume contraction and impaired baroreceptor function—a paradoxical finding that should raise diagnostic suspicion when occurring against a background of hypertension. Hypertensive crises may be triggered by tumor manipulation during physical examination, exercise, postural changes, micturition, or even food intake. Crisis symptoms include dizziness, flushing, visual disturbances, panic, nausea, vomiting, and occasionally epileptic auras.

Diagnostic evaluation relies on biochemical confirmation through measurement of plasma free metanephrines or 24-hour urinary fractionated metanephrines, which offer superior sensitivity and specificity compared to catecholamine measurements alone. Imaging with CT or MRI localizes the tumor, while functional imaging (MIBG scintigraphy or PET-CT with gallium-68 DOTATATE) identifies metastatic disease. Genetic testing is recommended for all patients given the high prevalence of hereditary syndromes including MEN2, von Hippel-Lindau disease, neurofibromatosis type 1, and succinate dehydrogenase mutations.

3.2 Cushing's Syndrome

Cushing's syndrome results from chronic exposure to excess glucocorticoids, whether from pituitary adrenocorticotropic hormone (ACTH) overproduction (Cushing's disease), ectopic ACTH secretion, or adrenal cortisol-secreting adenomas. Hypertension occurs in approximately 80% of patients and frequently proves resistant to conventional antihypertensive therapy. The hypertensive mechanisms involve cortisol-mediated mineralocorticoid receptor activation (particularly when 11β-hydroxysteroid dehydrogenase type 2 is overwhelmed), enhanced vascular reactivity to catecholamines, and increased hepatic angiotensinogen production.

Cushing's syndrome-related hypertensive emergencies may present with characteristic features including central obesity, moon facies, purple striae, proximal muscle weakness, and glucose intolerance. However, in ectopic ACTH production—such as from small cell lung carcinoma or, rarely, ACTH-secreting pheochromocytomas—the presentation may be dominated by severe hypokalemia and rapid-onset hypertension without classic stigmata. Diagnosis involves 24-hour urinary free cortisol measurement, late-night salivary cortisol, and the 1-mg overnight dexamethasone suppression test. ACTH levels and imaging distinguish pituitary, ectopic, and adrenal etiologies.

3.3 Primary Aldosteronism

Primary aldosteronism (Conn's syndrome), characterized by autonomous aldosterone secretion with suppressed renin, has emerged as the most common identifiable cause of secondary hypertension, potentially affecting 5–20% of hypertensive patients. Aldosterone-producing adenomas and bilateral adrenal hyperplasia represent the predominant pathologies. The condition promotes severe hypertension through sodium retention and volume expansion,

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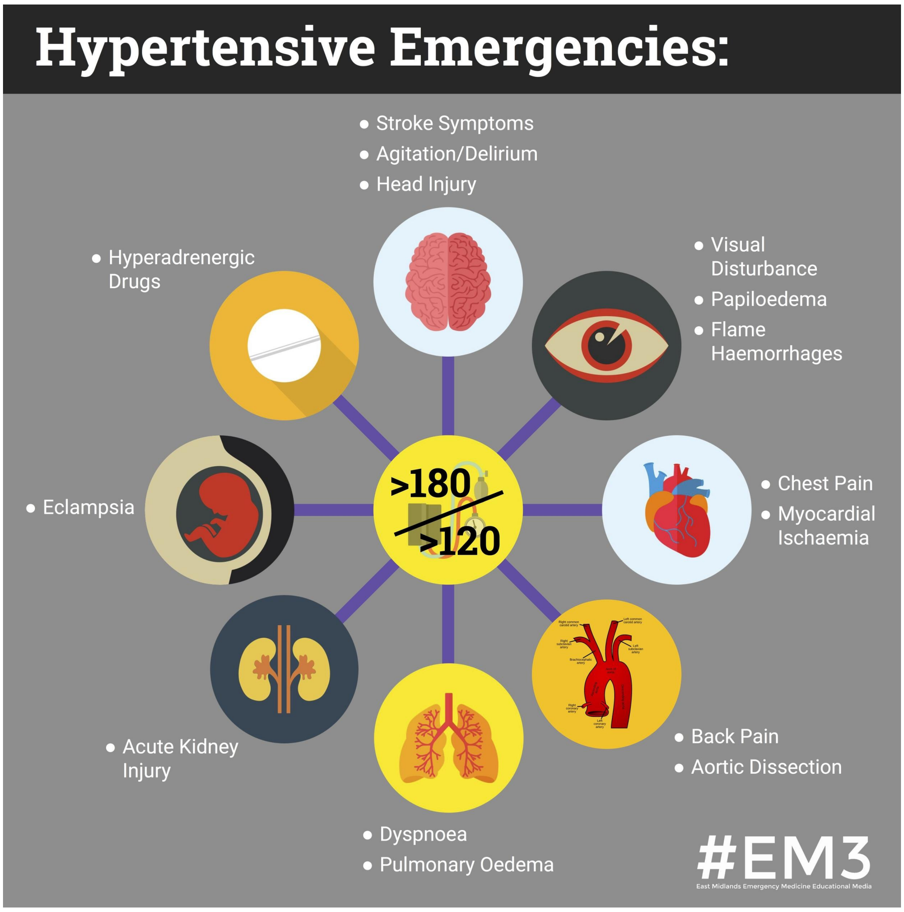
with additional direct vascular remodeling effects mediated by mineralocorticoid receptor activation in endothelial and vascular smooth muscle cells.

Patients with primary aldosteronism frequently present with resistant hypertension, hypokalemia (though increasingly recognized in normokalemic variants), and metabolic alkalosis. The aldosterone-to-renin ratio (ARR) serves as the primary screening test, with confirmatory testing using saline suppression or captopril challenge. Adrenal CT and adrenal vein sampling distinguish unilateral adenomas (amenable to surgical cure) from bilateral hyperplasia (managed medically with mineralocorticoid receptor antagonists). Recognition of primary aldosteronism is particularly important because targeted treatment—surgical or medical—can achieve cure or dramatic improvement in blood pressure control.



4. Renal Causes of Hypertensive Crisis

4.1 Renovascular Hypertension

Renovascular hypertension arises from renal artery stenosis, reducing renal perfusion and triggering compensatory RAAS activation. Atherosclerotic renal artery stenosis predominantly affects older individuals with diffuse vascular disease, while fibromuscular dysplasia—characterized by medial fibroplasia with the classic "string of beads" angiographic appearance—occurs more frequently in young women. The ischemic kidney generates excessive renin, converting angiotensinogen to angiotensin I and subsequently angiotensin II, a potent vasoconstrictor that also stimulates aldosterone secretion.

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Clinical clues to renovascular hypertension include onset of hypertension before age 30 or after age 55, abrupt deterioration of previously controlled hypertension, resistant hypertension requiring three or more medications, recurrent flash pulmonary edema, unexplained azotemia, or asymmetric kidney size on imaging. Doppler ultrasonography with renal artery resistive indices, CT angiography, magnetic resonance angiography, and invasive angiography establish the diagnosis. Revascularization through angioplasty with stenting (for atherosclerotic disease) or balloon angioplasty (for fibromuscular dysplasia) may cure or significantly improve hypertension, though medical management with RAAS blockade remains appropriate for many patients.

4.2 Renoparenchymal Disease

Chronic kidney disease (CKD) represents a major cause of secondary hypertension and a frequent precipitant of hypertensive crisis. The pathophysiology involves multiple interconnected mechanisms: RAAS activation from reduced nephron mass, increased sympathetic nervous system activity through afferent renal nerve signaling, endothelial dysfunction with reduced nitric oxide bioavailability, increased arterial stiffness, enhanced salt sensitivity, and volume overload from impaired natriuresis. The 2025 AHA/ACC guidelines recommend stronger use of renin-angiotensin system inhibitors in patients with diabetes and CKD, particularly when albuminuria exceeds 30 mg/g or eGFR falls below 60 mL/min/1.73 m².

Glomerular diseases—including IgA nephropathy, membranous nephropathy, focal segmental glomerulosclerosis, and lupus nephritis—frequently present with severe hypertension due to sodium retention, RAAS activation, and impaired renal autoregulation. Polycystic kidney disease, the most common inherited renal disorder, causes hypertension through compression of normal renal parenchyma by expanding cysts, ischemia-mediated RAAS activation, and sympathetic overactivity. Scleroderma renal crisis represents a particularly devastating form of renoparenchymal hypertensive emergency, characterized by abrupt onset of severe hypertension, rapidly progressive renal failure, and microangiopathic hemolytic anemia.

5. Clinical Manifestations and Target-Organ Damage

Hypertensive crisis produces diverse clinical manifestations depending on the affected end organs. The British and Irish Hypertension Society (BIHS) position document categorizes target-organ damage into cerebrovascular, cardiac, renal, and ophthalmic manifestations. Endocrine and renal etiologies may produce additional syndrome-specific features that aid diagnostic localization.

Target Organ	Manifestations	Endocrine/Renal Context
Cerebrovascular	Hypertensive encephalopathy, intracerebral hemorrhage, subarachnoid hemorrhage, ischemic stroke	Pheochromocytoma crisis may mimic stroke; CKD increases hemorrhagic risk
Cardiac	Acute heart failure, acute coronary syndrome, aortic dissection, arrhythmias	Cushing's syndrome accelerates atherosclerosis; aldosterone promotes

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		cardiac fibrosis
Renal	Acute kidney injury, proteinuria, hematuria, scleroderma renal crisis	Renal disease is both cause and consequence; bilateral RAS may worsen with ACEi/ARB
Ophthalmic	Papilledema, flame hemorrhages, exudates, cotton-wool spots	Malignant hypertension hallmark; indicates chronic severe BP elevation
Metabolic	Hypokalemia, metabolic alkalosis, hyperglycemia	Primary aldosteronism (hypokalemia); Cushing's syndrome (hyperglycemia)
Vascular	Aortic dissection, peripheral ischemia	Pheochromocytoma catecholamine surge causes vasospasm

6. Diagnostic Approach

The diagnostic evaluation of hypertensive crisis requires simultaneous assessment of blood pressure severity, target-organ damage, and underlying etiology. Laboratory studies should include comprehensive metabolic panels, complete blood count with peripheral smear (for microangiopathic hemolytic anemia), urinalysis with albumin-to-creatinine ratio, cardiac biomarkers, and thyroid function tests. The 2025 AHA/ACC guidelines emphasize that testing for secondary causes may be deferred until the patient is medically stabilized, though initial screening should not be overlooked.

6.1 Endocrine Screening

When endocrine hypertension is suspected, targeted biochemical testing should proceed systematically. Plasma free metanephrines or 24-hour urinary fractionated metanephrines screen for pheochromocytoma, with sensitivity exceeding 97% for plasma metanephrines. The aldosterone-to-renin ratio, measured under standardized conditions (morning, after 2 hours upright, with potassium repletion), screens for primary aldosteronism. Late-night salivary cortisol, 24-hour urinary free cortisol, and the 1-mg overnight dexamethasone suppression test evaluate for Cushing's syndrome. These tests should ideally be performed before initiating medications that may confound interpretation, though in crisis situations treatment takes precedence.

6.2 Renal Evaluation

Renal assessment in hypertensive crisis includes serum creatinine, estimated GFR, electrolytes, bicarbonate, and urinalysis. Renal imaging—ultrasonography, CT, or MRI—evaluates kidney size, cortical thickness, and structural abnormalities. Doppler ultrasound assesses renal artery flow velocities and resistive indices. CT or MR angiography visualizes renal artery anatomy when renovascular disease is suspected. In patients with acute kidney injury complicating hypertensive crisis, nephrology consultation should occur early to guide management and assess for underlying glomerular disease.

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7. Management Strategies

Management of hypertensive crisis in endocrine and renal disorders follows general principles of controlled blood pressure reduction while incorporating etiology-specific interventions. The fundamental goal is to reduce mean arterial pressure by no more than 20–25% within the first 6–24 hours, maintaining diastolic pressure above 100 mmHg (except in acute aortic syndromes) to preserve cerebral and coronary perfusion. Rapid or excessive reduction risks ischemic complications including cortical blindness, stroke, and myocardial infarction.

7.1 General Pharmacological Principles

Intravenous antihypertensive therapy is indicated for hypertensive emergencies with acute target-organ damage. Labetalol (combined α and β blocker) serves as first-line therapy for many presentations, particularly hypertensive encephalopathy and aortic dissection. Nicardipine, a dihydropyridine calcium channel blocker, offers titratability and safety in renal dysfunction. Clevidipine, an ultra-short-acting calcium channel blocker, provides rapid control with quick offset. Nitroprusside, though potent, is generally reserved for refractory cases given risks of cyanide toxicity and coronary steal. RAAS-blocking agents (ACE inhibitors and ARBs) are generally avoided in the acute phase of malignant hypertension due to risk of precipitous pressure drops.

7.2 Endocrine-Specific Management

Pheochromocytoma crisis demands α-adrenergic blockade as the cornerstone of therapy. Phenoxybenzamine (oral) or phentolamine (intravenous) reverses catecholamine-mediated vasoconstriction. Adequate fluid resuscitation is essential to correct intravascular volume depletion from pressure natriuresis. β-blockade should never precede α-blockade, as unopposed α-adrenergic stimulation can precipitate catastrophic hypertension. Once α-blockade is established, β-blockers may control tachycardia. Definitive treatment requires surgical resection after adequate medical preparation (typically 7–14 days of α-blockade).

Cushing's syndrome management targets the underlying cortisol excess. Pituitary adenomas may be resected transsphenoidally; adrenal adenomas require adrenalectomy. Medical options including ketoconazole, metyrapone, and mifepristone control cortisol production while awaiting definitive treatment. Primary aldosteronism due to unilateral adenoma is cured by laparoscopic adrenalectomy. Bilateral hyperplasia is managed with mineralocorticoid receptor antagonists (spironolactone or eplerenone) combined with conventional antihypertensives.

7.3 Renal-Specific Management

Renovascular hypertension management balances blood pressure control with preservation of renal function. RAAS blockade effectively controls hypertension but risks acute kidney injury in bilateral stenosis or solitary kidney scenarios. Close monitoring of creatinine after ACE inhibitor or ARB initiation is mandatory. Revascularization is indicated for flash pulmonary edema, resistant hypertension, or progressive renal failure. In CKD-related hypertensive crisis, loop diuretics address volume overload, while calcium channel blockers and β-blockers provide additional control. The 2025 guidelines support SGLT-2 inhibitors and mineralocorticoid receptor antagonists for cardiovascular and renal protection, even when blood pressure effects are modest. Sodium restriction below 2 grams daily and moderate physical activity (≥30 minutes, 5–7 days weekly) represent foundational lifestyle interventions.

Condition	First-Line Therapy	Key Considerations	Definitive Treatment
Pheochromocytoma Crisis	IV Phentolamine or Phenoxybenzamine	α-blockade before β-blockade; fluid resuscitation	Surgical resection after stabilization
Cushing's Syndrome	Ketoconazole/Metyrapone (medical); surgery	Control cortisol excess; manage hyperglycemia	Transsphenoidal surgery or adrenalectomy
Primary Aldosteronism	Spironolactone/Eplerenone	Monitor potassium; avoid ACEi/ARB initially in crisis	Adrenalectomy (unilateral); MRA (bilateral)
Renovascular HTN	CCB, β-blocker; cautious ACEi/ARB	Monitor creatinine; avoid in bilateral stenosis	Angioplasty ± stenting
CKD Hypertension	Loop diuretics, CCB, β-blocker	Sodium <2g/day; SGLT2i for renal protection	Dialysis if ESRD; transplant evaluation

8. Conclusion

Hypertensive crisis in the context of endocrine and renal disorders represents a complex interplay of hormonal dysregulation, neurohumoral activation, and target-organ vulnerability. While endocrine causes account for a minority of hypertensive emergencies, their potential for cure through targeted intervention makes recognition imperative. Pheochromocytoma, Cushing's syndrome, and primary aldosteronism each produce distinct pathophysiological signatures that guide both acute management and long-term treatment strategies. Similarly, renovascular and renoparenchymal diseases demand careful diagnostic evaluation and individualized therapeutic approaches that balance blood pressure control with organ perfusion.

The 2025 AHA/ACC Hypertension Guideline reinforces the importance of aggressive blood pressure targets in special populations, particularly those with CKD and diabetes, while emphasizing the need for personalized treatment. Contemporary management frameworks from the BIHS and other international bodies stress the dangers of overly rapid blood pressure reduction and advocate for controlled, stepwise approaches guided by autoregulatory principles. Future directions include expanded screening for primary aldosteronism, novel therapeutic agents such as endothelin receptor antagonists, and refined revascularization strategies for renovascular disease. Ultimately, successful management of hypertensive crisis in endocrine and renal disorders requires interdisciplinary collaboration among emergency physicians, endocrinologists, nephrologists, cardiologists, and surgeons to optimize patient outcomes and prevent recurrence.

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