Beyond the Usual Suspects: Hypercortisolism as a Key Cause of Resistant Hypertension

Jan B. Basile, MD

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FACULTY

Jan B. Basile, MD
Professor of Medicine
Division of Cardiology
Medical University of South Carolina
Ralph H. Johnson VA Medical Center
Charleston, SC

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INTRODUCTION

Hypertension (HTN) is one of the most common disorders in the United States, affecting approximately 50% of adults.[1] Despite multiple medications for managing blood pressure, resistant HTN occurs in 10% to 20% of patients treated for HTN.[2] According to the American Heart Association (AHA), resistant HTN is defined as above-goal elevated blood pressure despite concurrent use of 3 antihypertensive drug classes, commonly including a long-acting calcium channel blocker, a blocker of the renin-angiotensin system, and a diuretic, at maximally tolerated doses.[2] Resistant HTN also includes patients who achieve blood pressure targets on ≥4 antihypertensive medications.[2]

Hypercortisolism can result in HTN and other cardiometabolic comorbidities. Primary care clinicians (PCCs) usually manage patients with HTN and are often the first clinicians to evaluate the clinical picture that might trigger suspicion of hypercortisolism. Identifying patients with HTN who may have hypercortisolism is critical for improving outcomes.

Key takeaways

- Hypercortisolism has detrimental systemic effects that can result in HTN and other cardiometabolic comorbidities, increasing the risk of mortality.
- Hypercortisolism may act as a potential driver for HTN due to a variety of pathophysiologic mechanisms affecting the blood pressure regulatory system.
- Data from multiple studies, including the recent MOMENTUM study, show the prevalence of hypercortisolism in patients with resistant HTN to be as high as 27.3%.
- Screening for hypercortisolism is currently underutilized and should be considered in at-risk populations with resistant HTN who have physical characteristics or comorbidities that may indicate an increased likelihood of hypercortisolism.

BURDEN OF ENDOGENOUS HYPERCORTISOLISM IN HTN

Hypercortisolism is a disease spectrum characterized by excess cortisol and is associated with increased cardiometabolic comorbidities and mortality.[3] Endogenous hypercortisolism can be classified into 2 main categories[4,5]:

- ***ACTH-dependent hypercortisolism:*** Includes excess adrenocorticotropic hormone (ACTH) secretion by pituitary tumors (Cushing disease) and nonpituitary tumors (ectopic ACTH secretion).
- ***ACTH-independent hypercortisolism:*** Includes autonomous cortisol secretion by one or both adrenal glands.

Exogenous hypercortisolism refers to the intake of excess cortisol from external sources. This is most commonly from prescribed steroid medication, though sometimes cortisol is taken surreptitiously for other reasons. While Cushing syndrome (increased cortisol due to any cause) may be endogenous or exogenous, this review primarily aims to describe the role of endogenous hypercortisolism in patients with HTN, and we refer to endogenous hypercortisolism as hypercortisolism.

No matter how severe the clinical presentation or the degree of excess cortisol, hypercortisolism results in detrimental systemic effects, leading to various cardiometabolic comorbidities and increased mortality (**TABLE 1**).[3,6] Certain cardiometabolic risks of hypercortisolism, such as type 2 diabetes (T2D), have been established by prior studies such as CATALYST, which found a hypercortisolism prevalence of 24% in patients with T2D.[7-9] HTN is a prominent cardiometabolic risk of hypercortisolism; 89.3% of patients with hypercortisolism had HTN in the CATALYST population.[9] The risk of HTN in hypercortisolism is substantial, ranging from 40% to 90%.[3] Additionally, surgical resolution of hypercortisolism improves blood pressure control in about 30% to 70% of patients, indicating that hypercortisolism is often an underlying cause of HTN.[10,11]

TABLE 1. Increased cardiometabolic risk and mortality across the hypercortisolism spectrum.[3]

	Cardiometabolic risk, %* Lower → Higher		
	NFAI	**ACS**	**Overt CS**
Cardiovascular disease	3	10-20	20-30
Osteoporosis	20-40	40-60	40-50
HTN	40-75	60-80	50-90
T2D	10-25	20-40	20-50
Metabolic syndrome	70-80	70-90	80-90
Mortality	—	HR 12 (compared to NFAI)	HR 2-4 (compared to general population)
Serum cortisol post 1-mg DST	**<1.8 µg/dL**	**≥1.8 µg/dL**	

Abbreviations: ACS, autonomous cortisol secretion; CS, Cushing syndrome; DST, dexamethasone-suppression test; HR, hazard ratio; NFAI, nonfunctioning adrenal incidentalomas; T2D, type 2 diabetes.

*Percentages indicate the proportion of the populations with hypercortisolism who have the cardiometabolic conditions listed.

THE UNDERLYING PATHOPHYSIOLOGY OF HTN SECONDARY TO HYPERCORTISOLISM

The relationship between hypercortisolism and HTN is complex and thought to be multifactorial.[12,13] There are several proposed mechanisms by which excess cortisol disrupts blood pressure regulatory systems, including effects on the renin-angiotensin-aldosterone system (RAAS), mineralocorticoid receptor activity, vasoregulatory factors, and the sympathetic nervous system (**FIGURE 1**).[14-21]

- Excess endogenous cortisol causes increased hepatic synthesis of angiotensinogen and angiotensin II receptors type 1a, leading to increased vasoconstriction.[13]
- An estimated 5% to 21% of adrenal tumors cosecrete cortisol and aldosterone; increases in aldosterone lead to higher fluid volume and HTN.[13,22]
- Aldosterone and glucocorticoids bind to mineralocorticoid receptors; in hypercortisolism, excess cortisol leads to receptor activation, which increases sodium reabsorption, potassium secretion, and plasma volume, causing HTN.[13]
- Hypercortisolism affects vasoregulatory factors by glucocorticoid-mediated impairment of the nitric oxide pathway and disruption of the synthesis of vasodilators (kallikrein-kinin system), causing increased vasoconstriction and higher levels of endothelin-1 and erythropoietin.[13]
- Hypercortisolism adversely affects the sympathetic nervous system as excess cortisol is associated with enhanced pressor sensitivity to noradrenaline and beta-adrenergic agonists, resulting in increased blood pressure.[13]

Thus, hypercortisolism may act as a potential driver for HTN due to a variety of pathophysiologic mechanisms.

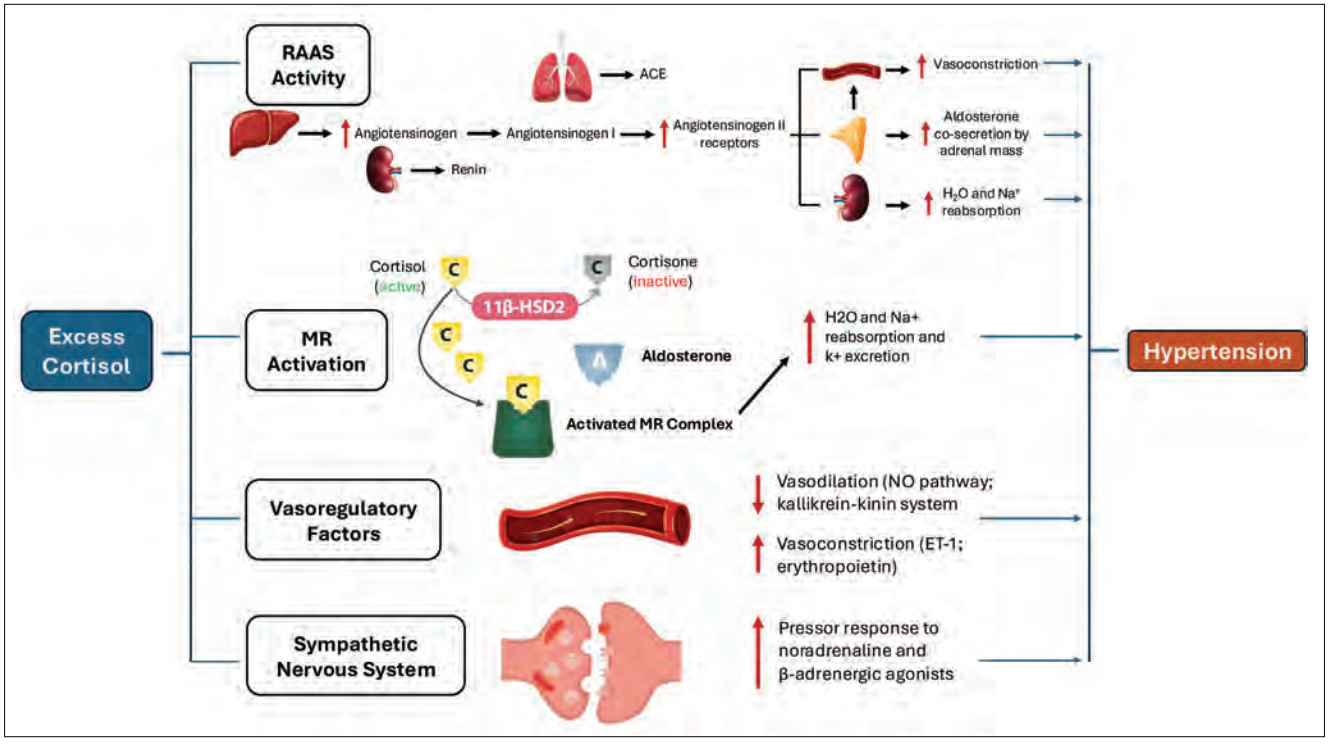
PREVALENCE OF HYPERCORTISOLISM IN PATIENTS WITH HTN

The prevalence of hypercortisolism in patients with HTN ranges widely depending on the population studied, with higher rates reported in select at-risk populations (**TABLE 2**).[4,23-31] A hypercortisolism prevalence of 0.1% to 7.5% has been reported in unselected patient populations with HTN, compared with a 7.5% to 18.3% prevalence of hypercortisolism in selected populations with HTN.[23-31] Selected population characteristics include resistant HTN, onset at <40 years, and individuals with adrenal lesions.[26-28]

MOMENTUM was a prospective study designed to provide an estimate of hypercortisolism prevalence and its associated clinical characteristics in a US population of adults with resistant HTN.[32] The study included 1000 adults with resistant HTN, defined by the AHA criteria noted previously, using a systolic blood pressure target of <130 mmHg. Patients with investigator-determined white-coat HTN, nonadherence to BP medications, and individuals whose DST results could be difficult to interpret were excluded.[32]

BP was measured by an automated device, which takes 3 measurements separated by 1 minute and provides the mean result. Patients with confirmed resistant HTN were tested for hypercortisolism at a second visit, using the 1-mg DST, and those with a post-DST cortisol level >1.8 µg/dL and dexamethasone ≥140 ng/dL were classified as having hypercortisolism.[32] Results indicate findings from the MOMENTUM study showed 27.3% prevalence of hypercortisolism in patients

FIGURE 1. Pathophysiology of cortisol-mediated HTN.[14-21]



Abbreviations: ACE, angiotensin-converting enzyme; ET-1, endothelin-1; H^2O, water; K, potassium; MR, mineralocorticoid receptor; Na, sodium; NO, nitric oxide. Red arrows indicate changes with elevated cortisol.
Images designed by Freepik.

with resistant HTN. Results also showed adrenal nodules in 24.3% of patients with hypercortisolism. In addition, the prevalence of hypercortisolism was >30% in patients with diabetes and cardiac disorders (e.g., atrial fibrilation, coronary artery disease, heart failure), and >40% in patients with kidney disease. According to the MOMENTUM findings, screening for hypercortisolism may be warranted in individuals with resistant HTN.[33]

SCREENING FOR HYPERCORTISOLISM IN PATIENTS WITH HTN

Although the links between hypercortisolism and HTN are known, clinicians are challenged to identify patient phenotypes and to determine those recommended by guidelines for hypercortisolism screening.[4] Additionally, hypercortisolism screening in at-risk patients with HTN is low, leading to missed diagnoses and suboptimal patient outcomes.[34]

Identifying patients with HTN who should be screened for hypercortisolism involves assessing characteristics that indicate a higher risk of hypercortisolism, such as age of onset of HTN, severity of HTN, HTN phenotype (dipping, nondipping, etc.), radiologic features (adrenal lesions), and clinical features of hypercortisolism.[12] Combining these characteristics with the use of a sensitive test for hypercortisolism—the 1-mg DST (described in more detail below) is recommended as first-line—can help clinicians more effectively identify patients with hypercortisolism.

HTN phenotypes in patients with HTN

The prevalence of HTN is approximately equal among men and women and across etiologies of hypercortisolism.[35,36] However, a nondipping circadian rhythm blood pressure profile is associated with hypercortisolism and may represent another clinical characteristic indicating a higher risk of hypercortisolism among patients with HTN.[37,38] Dipping refers to a decline in nighttime blood pressure of 10% to 20% compared to daytime levels, and various dipping patterns are associated with differing degrees of cardiovascular risk.[37] Nighttime blood pressure dipping patterns include the following[37]:

1. Reverse dipping (increase in nighttime blood pressure)
2. Nondipping (≤10% decline in nighttime blood pressure)
3. Dipping (>10% and ≤20% decline in nighttime blood pressure)
4. Extreme dipping (>20% decline in nighttime blood pressure)

TABLE 2. Prevalence of hypercortisolism in patients with HTN.

Study	Study design	Sample size	HTN population	Screening test(s)	Cutoff	Prevalence
Non-select patient populations						
Rudnick et al (1977)[23]	Retrospective, single-center	665	16-29 years: BP ≥140/90 mmHg 30-69 years: BP ≥160/100 mmHg ≥70 years: BP ≥170/100 mmHg	Not defined	Not defined	**0.2%**
Danielson et al (1981)[24]	Retrospective, single-center	1000	<40 years: BP ≥160/95 mmHg 40-60 years: BP ≥170/105 mmHg >60 years: BP ≥180/110 mmHg	Serum cortisol	Not defined	**0.1%**
Sinclair et al (1987)[25]	Prospective, cross-sectional, single-center	3783	Nonmalignant HTN not further defined Mean BP for men was 181/111 mmHg Mean BP for women was 185/109 mmHg	Not defined	Not defined	**0.1%**
Anderson et al (1994)[30]	Cross-sectional (1-day blood pressure study)	4429	Patients with HTN referred to a HTN referral center	Serum cortisol	Not defined	**0.5%**
Omura et al (2004)[29]	Prospective, cross-sectional, single-center	1020	Adults with HTN (BP >140/90 mmHg) with suspected endocrinologic diseases	1-mg DST	3.0 µg/dL	**1.0%**
Musolino et al (2025)[31]	Prospective, cross-sectional, single-center	106	Patients aged 30-70 years with HTN (BP >140/90 mmHg or the use of any antihypertensive treatment), without diabetes, and without obesity who were referred to a hypertension clinic	1-mg DST	1.8 µg/dL	**7.5%**
Select patient populations						
Martins et al (2012)[26]	Prospective, cross-sectional, single-center	430	Resistant HTN (BP ≥140/90 mmHg despite at least 3 antihypertensive drugs at full dosages, including a diuretic)	1-mg DST + LNSC	DST: 50 nmol/L LNSC: 3.6 nmol/L	**8.0%**
Trifanescu et al (2013)[27]	Retrospective, single-center	80	≤40 years of age at HTN onset (BP range 145-300/80-170 mmHg)	1-mg DST	1.8 µg/dL	**7.5%**
Aoe et al (2020)[28]	Retrospective, single-center	104	HTN (BP ≥140/90 mmHg) + adrenal masses	Possible subclinical CS 1-mg DST	DST: 1.8 µg/dL	**18.3%**

Abbreviations: BP, blood pressure; LNSC, late-night salivary cortisol.

These profiles have various levels of cardiovascular risk, with nondipping profiles associated with elevated risk.[37,38] About 40% to 56% of patients with hypercortisolism have a nondipping blood pressure.[39,40] Furthermore, the nondipping profile may be more prevalent in patients with adrenal hypercortisolism, among hypercortisolism etiologies.[39,40] Of note, identifying dipping blood pressure patterns typically requires 24-hour ambulatory blood pressure monitoring, which may not be feasible for all patients with HTN.

Screening for and diagnosing hypercortisolism in patients with HTN

In addition to a nondipping HTN phenotype, other patient characteristics indicate an increased likelihood for hypercortisolism, and such patients should be screened.[12]

- ***Age:*** <40 years of age with Grade 2 HTN or HTN in childhood
- ***Onset:*** acute worsening in patients with previously stable HTN or diabetes
- ***Severity:*** resistant HTN
- ***Radiologic findings:*** adrenal incidentalomas and adrenal lesions (these patients should also be screened for pheochromocytomas and aldosteronomas)[41,42]
- ***Clinical features of hypercortisolism:*** easy bruising, facial plethora, striae; proximal myopathy or muscle weakness; osteoporosis at young age; unusual features for age or multiple, progressive features

There are several tests available for identifying hypercortisolism, including the 1-mg DST, the 24-hour urinary

free cortisol (UFC), and the LNSC tests. The 1-mg DST is the most sensitive test and is the recommended first-line screening method.[43] The post-DST serum cortisol threshold for hypercortisolism is 1.8 µg/dL, and along with adequate dexamethasone plasma levels (>140 ng/dL) to prove adherence to the 1 mg dose, this is associated with a 100% sensitivity.[44] While the 24-hour UFC and LNSC tests are available, they are less sensitive for hypercortisolism screening, and the 24-hour UFC is not as practical for patients.[44]

A diagnosis of hypercortisolism is typically established by biochemical testing in patients with a clinical picture suggesting hypercortisolism.[12,43] Diagnostic flowcharts for hypercortisolism may be helpful for clinicians to implement a standardized process for detecting and diagnosing the disease in patients with HTN.[12,43] If PCCs suspect hypercortisolism based on clinical presentation and testing results (**FIGURE 2**), they should engage the multidisciplinary health care team for further workup and treatment, potentially including a referral to endocrinology and cardiology.[45] While PCCs may refer for further workup and diagnosis, this is a diagnosis that can be made by a PCC, and PCCs can properly diagnose and manage hypercortisolism to achieve timely treatment and optimal patient outcomes.

CASE STUDY

A 52-year-old man with a 15-year history of HTN is being managed by his primary care team in collaboration with a cardiologist. He currently takes losartan, hydrochlorothiazide, and amlodipine at maximum doses. His blood pressure today is 158/96 mmHg. It has been increasing over the past 2 years, and his cardiologist has noted a nondipping nocturnal blood pressure pattern. His PCC decides to screen him for hypercortisolism using the 1-mg DST.

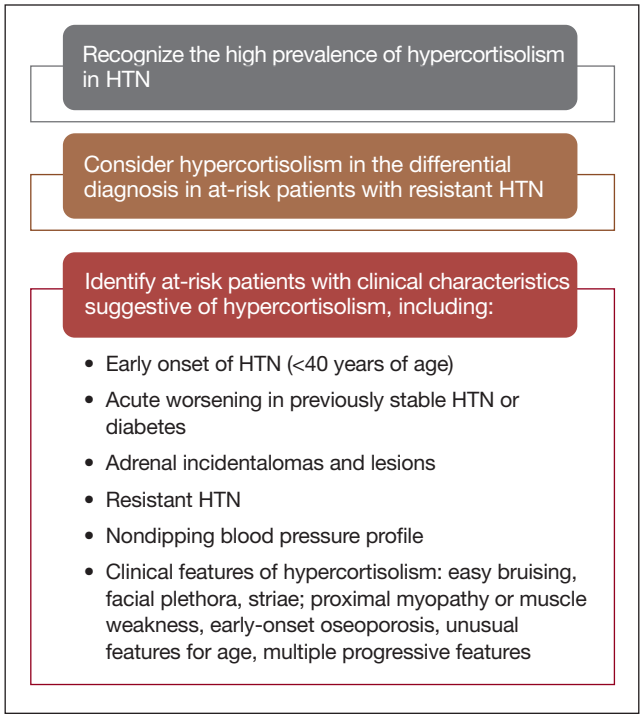
Laboratory evaluations

- Morning cortisol: 17 µg/dL (normal 10-25 µg/dL)[46]
- Serum cortisol after 1-mg DST: 3.8 µg/dL (normal is <1.8 µg/dL)[47]
- Serum dexamethasone after 1-mg DST: 417.2 ng/dL (expected is >140 ng/dL for adequate serum cortisol suppression)[48]
- 24-hour UFC: 47 µg/24 hr (normal range varies depending on specific test; example normal range is <11-53 µg/24 hr)[49]

Clinical assessment

This patient with resistant HTN has a strong likelihood of hypercortisolism, as evidenced by the results of the 1-mg DST and characteristics such as a nondipping blood pressure pattern and early onset of HTN (<40 years of age).

FIGURE 2. Key takeaways and action items for PCCs regarding resistant HTN and hypercortisolism.



Clinical learning

This case highlights the need to screen for hypercortisolism in patients with HTN and clinical characteristics that raise suspicion for cortisol-mediated elevations in blood pressure. While the patient may not receive a full diagnosis of hypercortisolism in primary care, the PCC can initiate a referral process, sending the 1-mg DST results to an endocrinologist to speed the process of a definitive diagnosis.

The PCC can also implement measures to mitigate cardiovascular risk, such as optimizing antihypertensive therapy and addressing other cardiometabolic factors. The PCC might also consider hypercortisolism-directed therapy, as addressing the underlying hypercortisolism is likely to improve the patient's blood pressure and cardiovascular risk.

SUMMARY

Hypercortisolism has detrimental systemic effects that can result in HTN and other cardiometabolic comorbidities, increasing the risk of mortality. Hypercortisolism may act as a potential driver for HTN due to a variety of pathophysiologic mechanisms. Data from multiple studies, including MOMENTUM, suggest a prevalence of hypercortisolism in up to 27.3% patients with resistant HTN. Screening should be considered in patients with resistant HTN and features suggesting an increased likelihood of hypercortisolism. ●

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