Management of Hypertensive Urgency *in an Urgent Care Setting*

Urgent message: Effective management of patients presenting to urgent care with acute high blood pressure starts with differentiating between hypertensive *emergency* and hypertensive *urgency* and ends with appropriate treatment and counseling.

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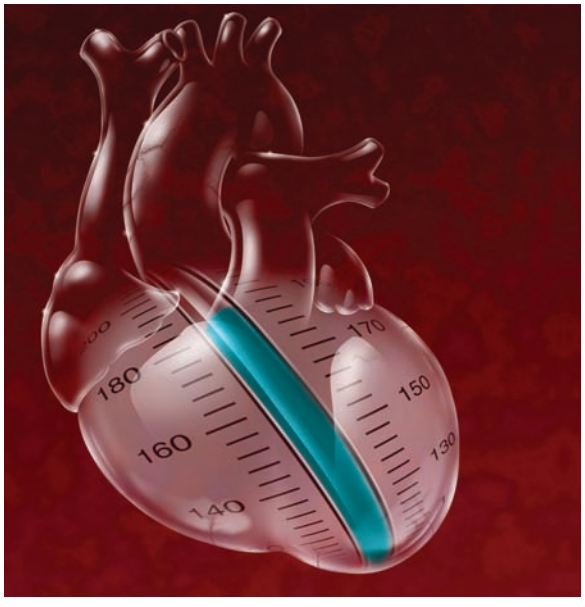
Introduction

Urgent care physicians routinely encounter patients with high blood pressure, but management—particularly for those patients with precarious elevations—remains controversial. Alternative options involve the use of various drug-therapy modalities in the urgent care setting with close observation, or initiation of oral medication and releasing the patient to home with specific instructions.

The consequences of inappropriate treatment can be disastrous, and include myocardial infarction, stroke, and death.



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Classification of Hypertension

Hypertension can be classified in various ways. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) classifies hypertension as shown in **Table 1**. Four categories of blood pressures are described, the most significant being Stage 2, defined as pressures >160/100 mmHg. While the JNC 7 does not define a blood pressure limit for hypertensive urgency or emergency, the report classifies "severe elevation" in blood pressure as >180/120 mm HG.

The World Health Organization (WHO), International Society of Hypertension (IHS), and European Society of Hypertension (ESH) all classify hypertension as shown in **Table 2**. In this system, there are six blood pressure categories, with the highest being Stage 3 at >180/110 mmHg.

Historically, systolic blood pressure (SBP) >179 and diastolic blood pressure (DBP) >109 has broadly been considered to be a "hypertensive crisis."[1] These pressures are further sub-classified as either hypertensive emer-

gency or hypertensive urgency.

Hypertensive emergency exists if there are signs of acute end-organ damage such as encephalopathy, myocardial infarction, unstable angina, pulmonary edema, eclampsia, stroke, head trauma, life-threatening arterial bleeding, and aortic dissection. As there is no absolute pressure measurement to define hypertensive emergency, it is identified by the physical signs of acute end-organ damage. Consequently, patients with a low baseline pressure can present with "normal" or mildly elevated pressure and be considered to have a true hypertensive emergency.

Patients with markedly elevated blood pressure but who lack these signs are determined to be in *hypertensive urgency*.[1] Some clinicians classify hypertensive urgency as "elevated blood pressure (diastolic pressure usually >120 mm Hg) that is not associated with new or progressive end-organ damage".[2]

Table 1: JNC-7 Classification of Hypertension

Category	SBP/DBP (mm Hg)
Optimal	<120/80
Prehypertensive	121–139 /80–89
Stage 1 hypertension	140–159/ 90–99
Stage 2 hypertension	>160/>100

Table 2: WHO, ISH, & ESH Classification of Hypertension

Category	SBP/DBP (mm Hg)
Optimal	<120/80
Normal	120-129/80-84
High normal	130-139/85-89
Stage 1 hypertension	140–159/90–99
Stage 2 hypertension	160-179/100-109
Stage 3 hypertension	>180/110

In hypertensive urgency, there is a risk of imminent end-organ damage, but such damage has not yet occurred. Particularly susceptible patients often have pre-existing conditions, e.g., renal insufficiency, congestive heart failure, coronary artery disease, CNS disorders, or retinal changes.

One to two percent of all hypertensive patients may present with hypertensive emergency or crisis at some point of their lives.[1]

Other terminologies used in these instances include:

- *Acute hypertensive episode*, which is defined as:
  - Stage 3 hypertension
  - systolic pressure 180 mmHg
  - *and* diastolic pressure 110 mmHg

with no signs or symptoms of evolving or impending target-organ damage.

- *Transient hypertension*, which is the presence of high blood pressure in association with other conditions such as anxiety, alcohol-withdrawal, sudden medication cessation, and toxic levels of some substances. In this case, treatment is aimed at the underlying cause.
- *White-coat hypertension*, or anxiety-related high blood pressure readings seen only in a physician's office, with otherwise normal blood pressure. This is a surprisingly common finding, especially in newly diagnosed hypertensive individuals. They actually exhibit normal pressures in their regular environment.

The goal in hypertensive *emergency* is to rapidly and carefully control the blood pressure to prevent fatal and irreversible end-organ damage. Action is usually taken in minutes up to a few hours as per the clinical situation, and intravenous medicines are usually employed. The aim may not be to reduce the blood pressure into the normal range in certain clinical scenarios such as stroke.

In hypertensive *urgency*, blood pressure can be controlled safely over period of hours or days in the outpatient setting.

Etiology

The etiology of hypertensive urgency is not well understood. Most such patients have pre-existent hypertension,[3] and non-adherence with antihypertensive medications near the time of the episode is seen in about 50% of them.[4] Illicit drug usage is also reported to be a risk factor for the development of hypertensive emergency.[5] Other causes of both urgency and emergency are shown in **Table 3**.

Pathophysiology

During the hypertensive episode, there is an abrupt increase in the systemic vascular resistance due to humoral vasoconstriction. This may be the triggering event.[6]

Increased blood pressure causes endothelial damage by increasing the endothelial permeability and local activation of the clotting cascade (platelet and fibrin deposition), resulting in fibrinoid necrosis and intimal proliferation. The endothelium is then unable to compensate or auto-regulate for changes in blood pressure. A vicious cycle ensues with further increases in resistance and endothelial damage.

High blood pressure also increases the stretch on the vessel wall which activates the renin-angiotensin system. This plays an important part in severely elevated blood pressures.

The combined process of endothelial damage, loss of auto-regulation, activated renin-angiotensin system, decrease in vasodilators (nitric oxide, prostacycline), and sustained blood pressure elevation can lead to tissue ischemia and end-organ damage. Major organ systems involved include the central nervous, cardiovascular, renal, and gravid uterus.[7, 8]

Table 3: Etiologic Causes of Hypertensive Urgency/Emergency

Essential Hypertension	
Renal	• Renal artery stenosis • Glomerulonephritis
Vascular	• Vasculitis – hemolytic-uremic syndrome – thrombotic thrombocytopenia purpura
Pregnancy-related	• Preeclampsia • Eclampsia
Pharmacologic	• Sympathomimetics • Clonidine withdrawal • Beta-blocker withdrawal • Cocaine • Amphetamines
Endocrine	• Cushing's syndrome • Conn's syndrome • Pheochromocytoma • Renin-secreting adenomas • Thyrotoxicosis
Neurologic	• Central nervous system trauma • Intracranial mass
Autoimmune	• Scleroderma renal crisis

Single-organ involvement is found in approximately 83% of patients presenting with hypertensive emergencies. Dual-organ involvement is found in 14% of cases, and multi-organ involvement (>3 organ systems) is found in approximately 3% of patients presenting with a hypertensive emergency.[9]

Clinical Presentation

A proper history and physical examination help a physician to differentiate between hypertensive urgency and emergency. A focused history should be taken to rule out end-organ damage, the signs and symptoms of which are shown in **Table 4**.

The history should include any previous history of high blood pressure, antihypertensive medications used and adherence to medication regimens, over-the-counter and illicit drug use (cocaine, amphetamines, decongestants, stimulants, oral contraceptives, and NSAIDs), and the presence of previous end-organ damage (e.g. renal, cardiac, or cerebrovascular).

Common symptoms related to hypertensive emergencies are chest pain (27%), dyspnea (22%), and neurologic deficits (21%).[10] Non-specific symptoms like a headache may be present in hypertensive urgency.

The physical exam should begin with measuring the blood pressure in both arms, using an appropriately sized cuff. Smaller cuffs can falsely elevate blood pressure readings in obese patients, and vice versa. The physical exam should also include a supine and standing blood pressure, as well as a measurement in the neck to assess for signs of elevated jugular venous pressure.

Next, pulses should be assessed in all extremities, and auscultation performed on the lungs (for signs of pulmonary edema), the renal arteries (for bruits), and the heart (for murmurs or gallops).

A focused neurologic and fundoscopic assessment should be done to rule out a cerebrovascular accident. Lateralizing signs are uncommon in hypertensive encephalopathy and are more suggestive of a stroke. Other studies which may be employed to help rule out a hypertensive emergency include electrocardiogram, chest x-ray, urinalysis, complete blood count, evaluation of electrolytes, and serum tests for renal function.

In a patient with severely elevated blood pressure, symptoms suggestive of acute end-organ damage confirm the diagnosis of hypertensive emergency, and the treatment plan should include immediate transfer to the

Table 4: Signs and Symptoms of End-organ damage

End-organ damage	Signs and symptoms
Hypertensive encephalopathy	• Signs of cerebral edema – insidious onset headaches – nausea – vomiting – altered mental status – confusion – drowsiness – seizures – occasional focal deficits – coma • Retinal hemorrhage or exudates • Signs of acute renal failure – oliguria – hematuria – proteinuria
Intracranial hemorrhage/stroke syndrome	• May occur with routine physical activity, especially during intense emotional activity or exertion • Headache and vomiting may lead to decrease level of consciousness • Typically, there is gradual progressive worsening of symptoms and increasing neurologic deficits, depending upon site of bleed
Acute left ventricular failure with pulmonary edema	• Cough, dyspnea and fatigue rapidly becoming severe • Chest discomfort or pain may be apparent • Tachypnea, tachycardia, S3 and/or S4 sounds, crackles at the pulmonary bases can be present • Signs of concomitant right-sided failure, including jugular venous distension and pedal edema, may be present
Acute coronary syndrome	• Typical or atypical chest pain (atypical chest pain especially in diabetic patients and inpatients with known cardiac or non-cardiac atherosclerotic disease)
Acute myocardial infarction (AMI)	• Typical or atypical chest pain • Electrocardiogram changes consistent with AMI
Dissecting aortic aneurysm	• Abrupt onset of thoracic or abdominal pain • Mediastinal or aortic widening on chest x-ray • Absence of proximal extremity or carotid pulse • Blood pressure difference of more than 20 mmHg between the right and left arm
Worsening renal failure	• Azotemia • Proteinuria • Oliguria • Hematuria
Eclampsia	• Pregnant patient with nausea, vomiting, or seizures

hospital for further management.

Patients with hypertensive urgency, on the other hand, can be treated in the urgent care setting.

Treatment

The goal of treatment in hypertensive urgency is to slowly reduce the blood pressure over a period of 24 hours using oral antihypertensive agents. This is usually done on an outpatient basis unless patient follow-up is unpredictable.

As non-adherence is the major cause of hypertensive urgencies, restarting the previously established regimen is usually sufficient. Treatment may be restarted with a lower dose and gradually increased as tolerated over a period of several days.

The mean arterial blood pressure should not be reduced by more than 25% in the first 24 hours.[10] Rapid or excessive reductions in blood pressure can have deleterious effects, including hypotension. This is more commonly seen in high-risk patients like the elderly, or patients with severe peripheral vascular disease, or severe atherosclerotic, cardiac, or intracranial disease.[10]

We should stress the importance of lowering blood pressure gradually to acceptable measurements; there is no evidence suggesting that immediately decreasing blood pressure to levels below "normal" reduces risk in the hypertensive patient.

Close follow-up, usually within 24 hours, is recommended. If there are severe comorbid conditions or safety issues at home, the patient can be observed in an inpatient setting for a day. A reduction in blood pressure to 160/110 mmHg is all that is required in the first 24 hours.

Essential information for oral antihypertensive agents commonly used for the treatment of hypertensive urgency is provided in **Table 5**.

Nifedipine is a dihydropyridine-derived calcium channel blocker that has

Table 5: Oral Antihypertensive Medications used in Hypertensive Urgency

Medication	Classification	Onset/Duration of action	Adverse effects	Dosing schedule	Special considerations
Clonidine	Centrally acting α-2-adrenergic agonist	Onset: 30–60 minutes Duration: 6–8 hours	• Dry mouth • Drowsiness • Constipation • Bradycardia • Orthostatic • Hypotension • Rebound • Hypertension with abrupt discontinuation	0.1 to 0.2 mg; additional doses of 0.1 mg given every hour until diastolic is <115 mmHg or a maximum dose of 0.7 mg has been given	• Safe for elderly or renal failure patients • Beta-blockers may worsen withdrawal symptoms • Contraindicated with sinus bradycardia, sick sinus syndrome, 1st-degree heart block or obtundation
Captopril	Angiotensin-converting enzyme (ACE) inhibitor	Onset: 15–30 minutes Duration: 4-6 hours	Skin rash, cough, taste alterations, hyperkalemia, angioedema, renal failure (in patients with bilateral renal artery stenosis)	25 mg; may be repeated every 30 minutes as needed	• Captopril is the shortest-acting ACE-inhibitor • Blood pressure will not decrease significantly if no response is observed within 30 to 60 minutes
Losartan	Angiotensin II-receptor (ARB) antagonist	Peak: 1-3 hours	Generally well-tolerated with incidence of adverse effects similar to placebo	50 mg; higher doses have not been found to produce more significant blood pressure reductions	Extensive first-pass metabolism may cause bioavailability to double in patients with hepatic impairment
Nicardipine (regular release)	2nd-generation dihydropyridine calcium channel blocker	Onset: 0.5-2 hours Duration: 8 hours	Hypotension, heart palpitations, reflex tachycardia, headache, flushing, dizziness	30 mg	• Extensive first-pass metabolism • Do not use in acute heart failure or coronary ischemia
Labetolol	Nonselective α-1, β-adrenergic receptor blocker	Onset: 20 min–2 hours Duration: dose dependent	• Nausea • Vomiting • Dizziness • Heart block • Headache • Fatigue • Broncho-constriction	200-400 mg; repeat every 3 hours as needed	Do not uses in patients with • heart failure • asthma/COPD • bradycardia • 1st-degree heart block

been used extensively in the past for the treatment of hypertensive urgency. However, nifedipine has never been approved by the FDA for short-term use in hypertension. More recently, the risks of serious adverse reactions like severe hypotension, acute coronary events and ischemic stroke have led the U.S. National Heart, Lung, and Blood Institute to issue a warning that this agent should not be used in the treatment of hypertension, angina, and myocardial infarction.

Conclusion

Initial recognition of an absence of end-organ damage is crucial in differentiating hypertensive urgency from hypertensive emergency, and establishing a treatment plan. Judicious use of oral antihypertensive agents in the outpatient clinical setting can safely lower blood pressure over a period of several days, leading to improved outcomes. ■

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