Hypertensive Emergencies: Are You Prepared?


The untreated hypertensive emergency carries a one-year mortality of more than 90%. This review provides keys to the management of critical states of cardiopulmonary, neurologic, and renal dysfunction due to hypertension.

By Eric Y. Baden, MD, and Bruce D. Adams, MD

Hypertension is a disorder that virtually all physicians encounter routinely, regardless of their practice setting. About 25% of all patients seen in emergency departments, for example, have elevated blood pressure. Hypertension is well known for being a “silent killer” because of its long asymptomatic phase and chronic adverse effects on a person’s cardiovascular health. Under certain circumstances, however, an elevated blood pressure can represent an immediately life-threatening condition in the form of a hypertensive emergency. With a one-year mortality of more than 90% if left untreated, a hypertensive emergency is an ominous syndrome associated with impending morbidity or even death if not properly recognized and treated.

In this article, we will present three patients with symptoms and signs of a hypertensive emergency. We will then discuss the condition in terms of its pathophysiology and its effects on the cardiopulmonary, neurologic, and renal systems. Lastly, we will review appropriate diagnostic testing and management strategies.

THREE PATIENT PRESENTATIONS

The following three cases represent scenarios that physicians may encounter in patients presenting with elevated blood pressure.

Patient #1. A 70-year-old woman presents with tearing chest pain that started suddenly 30 minutes earlier and radiates to her back. She has no other complaints. Her medical history is significant for hyperlipidemia and hypertension, for which she was taking a statin and two blood pressure pills. However, she says she stopped taking all three pills a week ago because she “felt fine.”

Physical examination reveals a thin woman clutching her chest in moderate distress. Her vital signs are: pulse, 100; blood pressure, 210/120 mm Hg; respiratory rate, 22; temperature, 98.6°F. The cardiac exam reveals a II/IV blowing diastolic rumble heard at the base but no gallops or rubs. She has a decreased right radial pulse compared with her other three extremities and bibasilar inspiratory crackles.

The rest of her exam is normal. An ECG shows left ventricular hypertrophy, and a chest x-ray shows a widened mediastinum.

Patient #2. A 45-year-old woman presents with sudden onset of headache and confusion. The rest of her history, unfortunately, is limited by her altered mental status. Physical examination reveals a confused woman in mild distress from the headache. Her vital signs are: pulse, 100; blood pressure, 210/110 mm Hg; respiratory rate, 22; temperature, 98.6°F. The neurologic exam is significant for left-sided sensory neglect and agnosia. The remainder of the exam is normal.

A stat unenhanced computed tomography (CT) scan is obtained (see image). It shows an intraparenchymal hemorrhage from an arteriovenous malformation.
Cerebrovascular event. Hypertensive patient #2, who presented with headache and confusion, underwent CT scanning that revealed intraparenchymal hemorrhage from an arteriovenous malformation.

Patient #3. A 55-year-old man presents with progressive chest pain and dyspnea that started an hour ago. Twelve months earlier, he was diagnosed with diffuse scleroderma; he has no other medical or surgical history. Physical examination reveals a fully oriented man in severe respiratory distress. His vital signs are: pulse, 100; blood pressure, 210/110 mm Hg; respiratory rate, 40; temperature, 98.6°F. Auscultation detects extensive rales. He has the classic cutaneous findings of scleroderma—taut skin, sclerodactyly, and Raynaud’s phenomenon. An ECG shows left ventricular strain, and a chest x-ray reveals severe pulmonary edema.

His laboratory values are: blood urea nitrogen, 40 mg/dl; creatinine, 2.5 mg/dl; and serum renin plasma activity, 24.5 ng/ml/hr (normal = 0.77 to 4.6 ng/ml/hr). Cardiac enzymes are normal.

DISTINCTION IN TERMINOLOGY

We will revisit these patients later in this article, but before we move on to a discussion of the
Pathophysiology in hypertensive emergency, it is important to point out a key distinction in terminology. Along the continuum of elevated blood pressure lie two syndromes—hypertensive emergency and hypertensive urgency—that are defined by the presence or absence, respectively, of end organ damage. A hypertensive emergency is a syndrome of symptoms and signs that reflects acute end organ damage directly resulting from a severely elevated blood pressure. In contrast, hypertensive urgency is an elevation in blood pressure without evidence of end organ dysfunction; however, the term implies the imminent development of end organ damage if the patient’s blood pressure is not lowered within days to weeks.

While the absolute blood pressure reading is not always relevant, it is generally accepted that a hypertensive emergency occurs at a higher blood pressure than a hypertensive urgency. However, the critical point is that the degree of hypertension is not as significant as the patient’s presentation.

A normotensive individual’s arteries dilate and constrict in response to changes in blood pressure, an autoregulatory process that maintains tissue perfusion at a relatively constant level. Patients with chronic hypertension develop arteriolar hypertrophy, shifting their autoregulatory curve to the right. The blood pressure level at which autoregulatory mechanisms fail depends on the chronicity of the patient’s hypertension and the extent of structural changes present. It is this concept that reinforces the importance of a patient’s clinical presentation rather than an absolute blood pressure reading when making the distinction between a hypertensive emergency and hypertensive urgency.

The table below defines these and other terms that are used to describe various syndromes resulting from elevated blood pressure.

<table>
<thead>
<tr>
<th>Terminology Used for Hypertension in Emergency and Acute Settings</th>
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<tbody>
<tr>
<td>hypertensive emergency</td>
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<tr>
<td>hypertensive urgency</td>
</tr>
<tr>
<td>hypertensive crisis</td>
</tr>
<tr>
<td>malignant hypertension</td>
</tr>
<tr>
<td>accelerated hypertension</td>
</tr>
<tr>
<td>malignant nephrosclerosis</td>
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As noted, true hypertensive emergencies involve acute progressive end organ damage from severely elevated blood pressure. The dysfunction can occur in the cardiopulmonary, neurologic, or renal system (see box below).
Cardiopulmonary
- aortic dissection
- cardiac ischemia
- pulmonary ischemia

Neurologic
- hypertensive encephalopathy
- stroke syndrome
  - hemorrhagic (intraparenchymal or subarachnoid)
  - ischemic
- eclampsia

Renal
- acute renal failure
- scleroderma renal crisis

CARDIOPULMONARY HYPERTENSIVE EMERGENCIES

Cardiac ischemia from a hypertensive emergency results from increased afterload and ventricular wall stress. The chest pain that ensues is similar to the anginal chest pain of an acute coronary syndrome (ACS). Both types of pain are caused by a relatively inadequate oxygen supply to the myocardium and can be explained in terms of supply and demand. The chest pain from a hypertensive emergency results from a sudden increase in myocardial oxygen demand. Conversely, the cardiac ischemia from an ACS results from a sudden decrease in supply when an atherosclerotic plaque ruptures, an acute thrombus forms, and forward blood flow is impaired.

The distinction between these two pathologies is important because treatment priorities differ. Treatment of cardiac ischemia from a hypertensive emergency is focused on decreasing oxygen demand by lowering blood pressure. This is best accomplished with nitroglycerin, administered sublingually or as an infusion, or with a beta blocker, such as metoprolol, labetalol, or esmolol. Treatment of an ACS, on the other hand, is intended to increase oxygen supply through antiplatelet therapy, heparinization, and fibrinolytic medications and mechanical revascularization. Often, the distinction between these strategies is blurred and both treatment approaches are taken simultaneously.

Pulmonary edema represents an acute failure of the heart to overcome an elevated blood pressure. Longstanding hypertension produces left ventricular hypertrophy; any abrupt elevation in blood pressure can create an oxygen supply-demand mismatch. The ischemic left ventricle stiffens, leading to an inability to relax (diastolic dysfunction) and an inability to receive blood. This backflow increases the hydrostatic forces within the pulmonary system, resulting in pulmonary edema. Next, the sensation of breathlessness develops, causing anxiety and catecholamine release. This sympathomimetic drive further increases oxygen demand, creating a vicious cycle.

Understanding this process is important since treatment focuses not only on lowering blood pressure but also on blunting the catecholamine surge. The antihypertensive agent of choice is the angiotensin-converting enzyme inhibitor enalaprilat, which acts as both an afterload and preload reducer. A cheaper and effective alternative is a nitroglycerin infusion.

About 90% of cases of aortic dissection are associated with hypertension. An elevated blood pressure not only contributes to its etiology but also to its progression. The process begins as a tear in the intima that extends into the vessel wall before reentering the lumen, creating a “double-barreled” aorta. Patient #1 above represents the classic presentation. Associated symptoms and physical examination and radiographic findings depend on where along the aorta the dissection occurs and the other arteries involved.

The variety of clinical presentations makes aortic dissection a challenging diagnosis. Chest radiography is an initial screening tool with a sensitivity of 80% to 90%, but definitive diagnosis requires transesophageal echocardiography, CT angiography, magnetic resonance imaging (MRI), or aortography. The precise diagnostic test is controversial. Each method has its inherent
advantages and disadvantages. The test of choice depends on institutional capabilities, physician preference, and patient stability.

For patient #1, CT angiography was used to diagnose an ascending aortic dissection involving the aortic valve (causing the diastolic murmur of aortic regurgitation) and the brachiocephalic and subclavian arteries (causing the decreased radial pulse). It is the preferred study for the hemodynamically stable patient since it is readily available at most hospitals and can rule in or rule out other diagnoses; it also has a sensitivity approaching 98%. Transthoracic echocardiography can be performed at the bedside for the hemodynamically unstable patient. Aortography is still considered the gold standard, despite having a sensitivity of only 77% to 88%.

While the treatment for an ascending aortic dissection is ultimately surgical, medical management is critical in limiting the severity and may be all that is required for descending dissections. When confronted with a potential aortic dissection, decreasing systolic blood pressure to less than 120 mm Hg with a vasodilator is necessary in order to minimize aortic shear stress and extension of the dissection. To prevent a reflex tachycardia, a beta blocker needs to precede the vasodilator. A combination of the beta blocker esmolol followed by nitroprusside or labetalol, a combination alpha and beta blocker, are the preferred treatments.

Patient #1 had her heart rate controlled with an esmolol infusion, followed by nitroprusside to control her blood pressure. She was then taken immediately to the operating room for repair of the aortic dissection.

NEUROLOGIC HYPERTENSIVE EMERGENCIES

Hypertensive encephalopathy is a diagnosis of exclusion that results from progressively worsening cerebral edema. It is characterized by the insidious onset of headache, nausea, and vomiting, followed by nonlocalized symptoms such as irritability and confusion. If left untreated, it progresses to seizures, coma, and ultimately death. Papilledema is usually present, along with significant retinopathy (cotton wool spots). Focal deficits can occur but usually do not localize to a single anatomic lesion.

These neurologic symptoms differ from the abrupt onset of focal neurologic symptoms typically seen with a stroke, such as the hemorrhagic stroke suffered by patient #2 presented above. Both hemorrhagic and ischemic strokes present with focal neurologic deficits that localize to a single lesion, corroborated by an unenhanced CT or MRI scan (or magnetic resonance angiography). Subarachnoid hemorrhage is a type of hemorrhagic stroke that presents with the sudden onset of a severe headache, typically described by the patient as "the worst headache of my life" or "like a thunderclap." It is diagnosed by classic findings on head CT or lumbar puncture and confirmed by cerebral angiography.

Distinguishing between the various neurologic hypertensive emergencies is important because appropriate management of these conditions is somewhat different. Sodium nitroprusside is still the drug of choice for encephalopathy and can be used in the other conditions. Labetalol is a good alternative. Nimodipine, a calcium channel blocker, is a useful treatment adjunct for subarachnoid hemorrhage. It is thought to decrease the incidence and severity of reflex cerebral vasospasm, thus preventing conversion of the hemorrhagic stroke to an ischemic one.

Target blood pressure also depends on the presumptive diagnosis. It is lower for encephalopathy than for an acute stroke in progress.

Eclampsia is an obstetric hypertensive emergency marked by seizure activity or coma in the absence of an alternative disease process. It can occur at any time from the second trimester to the first 48 hours after delivery. Treatment includes reduction of blood pressure, prevention or control of seizures, and early obstetric consultation since definitive treatment requires delivery of the fetus.

The antihypertensive agent of choice for eclampsia is hydralazine, a direct vasodilator that is a class B drug and therefore safe during pregnancy. Nicardipine and fenoldopam are alternatives. Labetalol can be used but some sources label it as pregnancy category D during the second and third trimesters. Nitroprusside should be avoided because of the potential for cyanide toxicity. It should be considered only after all other agents have failed.
RENAL HYPERTENSIVE EMERGENCIES

Renal function and blood pressure directly affect one another. A severely elevated blood pressure may lead to acute renal failure or may exacerbate chronic renal failure, which can lead to hypertension. Elevated blood pressure impairs the kidneys’ autoregulatory properties, resulting in decreased renal perfusion. The renin-angiotensin-aldosterone cascade is triggered, leading to even further vasoconstriction, sodium retention, and increased blood pressure and further impairment of autoregulation. This vicious cycle continues until arteriolar necrosis occurs and renal failure results.

Patients present clinically with decreased urine output, increased blood urea nitrogen and creatinine levels, proteinuria, or the finding of red blood cells or red blood cell casts in the urine. Worsening renal function in the setting of hypertension is a hypertensive emergency requiring an immediate reduction in blood pressure. Fenoldapam, a dopamine agonist is a reasonable treatment option, as are nicardipine and labetalol. Diuretics are a useful adjunct to help manage fluid balance.

Scleroderma renal crisis is a rare condition that deserves special mention because of its unique treatment. Initial blood pressure control can be achieved with traditional medications targeted at presenting symptoms, such as the pulmonary edema seen in patient #3 above. However, definitive treatment is intravenous (IV) enalaprilat.

EVALUATION PROCESS

An alphabet-based mnemonic can assist in the assessment of any patient with severely elevated blood pressure and the determination as to whether a hypertensive emergency is present (see algorithm). The recommended evaluation process centers on obtaining accurate bilateral blood pressures, a focused history, a careful physical examination emphasizing neurologic, pulmonary, and cardiovascular findings, and objective studies such as a chest x-ray, ECG, urinalysis, renal function testing, and CT of the head. If possible, test results should be compared with the patient’s medical records to determine which, if any, of the abnormalities are acute.
It is important to consider other life-threatening causes of hypertension besides a hypertensive emergency. While not exhaustive, the box below lists other such diagnoses that require initial stabilization.
Patients with a true hypertensive emergency are best treated in an intensive care unit (ICU) with IV drug therapy and close observation. Immediate transport should be arranged for patients diagnosed in a clinic or community setting. The treatment goal is to restore blood pressure to a range in which autoregulatory forces can be re-established. This can be accomplished by a reduction in blood pressure of no more than 25% within the first hour. Further acute reductions can cause end organ ischemia. If the reduced blood pressure is well tolerated and the patient remains clinically stable, gradual reductions toward a normal blood pressure can be implemented over the next 24 to 48 hours by an intensivist in an ICU setting.

There are exceptions to this approach. No clear evidence exists from clinical trials to support immediate antihypertensive treatment in evolving strokes. Furthermore, it is theorized that the hypertensive state is necessary to maintain perfusion and salvage the edematous area adjacent to the infarcted brain, the so-called ischemic penumbra. If the patient’s blood pressure is extremely high (mean arterial pressure above 130 mm Hg or systolic blood pressure above 220 mm Hg), a reasonable approach would be to gradually lower the blood pressure while doing serial neurologic exams. Clinical improvement warrants continued reductions toward the aforementioned goal of 25%. However, any deterioration in the patient’s condition mandates immediate discontinuation of antihypertensive treatment.

Conversely, however, patients with aortic dissection require a rapid decrease in blood pressure. The target systolic blood pressure for this emergent condition of less than 120 mm Hg, if tolerated, will likely violate the 25% rule.

The drug regimen used to treat a hypertensive emergency often depends on physician preference, the specific emergency that is present, and patient comorbidities. The table below provides an overview of the commonly used medications.
**USE OF AN INTRA-ARTERIAL CATHETER**

An intra-arterial catheter is useful when treating a true hypertensive emergency. By calculating the area under the blood pressure curve, it can provide the most accurate determination of mean arterial pressure. It also provides real-time fluctuations in mean arterial pressure with medical interventions and infusion adjustments and can be helpful with fast-acting medications such as nitroprusside. However, treatment should not be delayed to place an intra-arterial catheter. Initial management can safely proceed in the emergency department using traditional noninvasive blood pressure monitoring.

A final note: The term “urgency” can lead to overly aggressive management of patients with severe, uncomplicated hypertension. There is no evidence to suggest that aggressively lowering blood pressure in the emergency department is associated with improved outcomes. Such patients are best managed in consultation with their outpatient provider. They may benefit from adjustments in their antihypertensive therapy or resumption of medications if noncompliance was the problem, or they may not need any changes at all in their treatment plan. The most important part of the discharge plan is to ensure a follow-up visit within the next few days.

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**Intravenous Drugs Used to Treat Hypertensive Emergencies**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of action</th>
<th>Dose</th>
<th>Onset of action</th>
<th>Duration of action</th>
<th>Adverse effects</th>
<th>Indications</th>
<th>Cautions/contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>nitroprusside</td>
<td>peripheral vasodilator</td>
<td>0.25-10 µg/kg/min</td>
<td>immediate</td>
<td>1-10 min</td>
<td>nausea, vomiting, thiocyanate and cyanide toxicity</td>
<td>most hypertensive emergencies</td>
<td>caution with renal failure; avoid during pregnancy</td>
</tr>
<tr>
<td>labetalol</td>
<td>alpha, beta-1, and beta-2 blocker</td>
<td>20-80 mg bolus q 10 min; maximum 300 mg total dose</td>
<td>2-5 min</td>
<td>2-4 hr</td>
<td>bronchoconstriction, heart block, hypotension, bradycardia</td>
<td>most hypertensive emergencies</td>
<td>avoid in pulmonary edema from acute heart failure; avoid during pregnancy</td>
</tr>
<tr>
<td>esmolol</td>
<td>beta-1 selective blocker</td>
<td>500 µg/kg loading dose, then 50-200 µg/kg/min</td>
<td>2-10 min</td>
<td>10-30 min</td>
<td>bronchoconstriction, heart block, hypotension, bradycardia</td>
<td>aortic dissection; follow with afterload reduction</td>
<td>caution with severe bronchoconstriction and advanced heart block</td>
</tr>
<tr>
<td>nicardipine</td>
<td>calcium channel blocker</td>
<td>5-15 mg/hr</td>
<td>5-10 min</td>
<td>1-6 hr</td>
<td>reflex tachycardia, hypotension</td>
<td>most hypertensive emergencies</td>
<td>caution with ACS; avoid in pulmonary edema from acute heart failure</td>
</tr>
<tr>
<td>fenoldopam</td>
<td>dopamine agonist</td>
<td>0.1-0.3 µg/kg/min</td>
<td>&lt;5 min</td>
<td>30-60 min</td>
<td>reflex tachycardia</td>
<td>most hypertensive emergencies</td>
<td>caution if glaucoma is present</td>
</tr>
<tr>
<td>nitroglycerine</td>
<td>smooth muscle relaxer</td>
<td>5-100 µg/min</td>
<td>2-5 min</td>
<td>5-10 min</td>
<td>methemoglobinemia, tachyphylaxis</td>
<td>coronary ischemia</td>
<td>avoid if erectile dysfunction medication was used within prior 24 hours</td>
</tr>
<tr>
<td>enalaprilat</td>
<td>ACE inhibitor</td>
<td>1.25-5 mg q 4-6 hr</td>
<td>15-30 min</td>
<td>6-12 hr</td>
<td>hyperkalemia, renal insufficiency, dry cough</td>
<td>acute pulmonary edema, scleroderma, renal crisis</td>
<td>avoid during pregnancy</td>
</tr>
<tr>
<td>hydralazine</td>
<td>direct vasodilator</td>
<td>10-20 ng</td>
<td>10-20 min</td>
<td>1-4 hr</td>
<td>reflex tachycardia, coronary steal syndrome</td>
<td>severe preeclampsia, eclampsia</td>
<td>caution if coronary artery disease is present</td>
</tr>
</tbody>
</table>

ACS = acute coronary syndrome
Suggested Reading


