CLINICAL PRACTICE

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Acute Severe Hypertension

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This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.*

A 58-year-old woman with known hypertension comes to the emergency department and reports headaches and blurred vision for the past 3 days. Her prescribed medications include amlodipine, hydrochlorothiazide, and lisinopril, but she acknowledges spotty adherence and has not taken any of the drugs in approximately 3 weeks. On examination, she is anxious but comfortable. The average of multiple seated blood-pressure measurements is 242/134 mm Hg, and the heart rate is 68 beats per minute. Funduscopy shows arteriolar narrowing, bilateral flame hemorrhages, cotton-wool spots, and papilledema; auscultation reveals a fourth heart sound. The remainder of the examination is normal. The electrocardiogram shows left ventricular hypertrophy. Other laboratory tests and chest radiography are normal. Emergency computed tomography of the head shows heterogeneous hypoattenuation of subcortical white matter in the posterior parieto-occipital regions bilaterally but no hemorrhage or infarction. How would you further evaluate and treat this patient?

THE CLINICAL PROBLEM

Blood-pressure elevations above 180/110 to 120 mm Hg can result in acute injury to the heart, brain, and the microvasculature.[1-3] If acute hypertension-mediated target-organ damage is present, the condition is labeled "hypertensive emergency" and demands immediate and aggressive treatment to limit progressive injury (Fig. 1). There is less agreement on terminology and management in the absence of acute target-organ damage (which I will refer to here as "hypertensive urgency"), although this condition is two to three times more common than hypertensive emergency.[4,5] Acute severe hypertension, at times with acute target-organ damage,[6] may also manifest perioperatively; the present review focuses on the occurrence of acute severe hypertension outside the perioperative setting. Similarly, the recommendations in this article do not apply to hypertension during pregnancy.

Both absolute blood-pressure level and the pace of rise determine the risk of acute hypertension-mediated target-organ damage. Many patients with chronic hypertension have severe blood-pressure elevations for months or years without apparent effects, whereas sudden increases that are more modest (e.g., to a level of 160/100 mm Hg in a previously normotensive patient) can cause severe injury, particularly to the cerebral vasculature (as in eclampsia, pheochromocytoma, drug-induced acute hypertension, or acute glomerulonephritis).

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KEY CLINICAL POINTS

ACUTE SEVERE HYPERTENSION

- Acute severe hypertension that is accompanied by acute target-organ injury (hypertensive emergency) is associated with substantial morbidity and in-hospital mortality, thus requiring immediate treatment in an intensive care unit.
- Acute severe hypertension without acute target-organ damage (hypertensive urgency) is not associated with adverse short-term outcomes and can be managed in the ambulatory setting.
- Nonadherence to previously prescribed antihypertensive medications is the most common factor leading to acute severe hypertension.
- Chronic hypertension shifts the cerebral blood flow autoregulation curve to the right (i.e., to higher blood-pressure levels), which confers a predisposition to cerebral hypoperfusion at relatively high (normal) blood-pressure levels. This principle guides the pace of blood-pressure reduction in acute severe hypertension.
- Hypertensive emergencies are managed with intravenous medications guided by the type of target-organ damage.
- Hypertensive urgencies should be managed with oral medications and arrangements for prompt follow-up.

Acute severe hypertension accounts for an estimated 4.6% of all visits to emergency departments and is a frequent reason for hospitalizations in the United States.[5] It is more common in persons who are older than 60 years of age, black, or uninsured or underinsured or who live in lower-income areas.[5,7] Large claims-based data sets in the United States indicate that hospital admissions for hypertensive emergencies have steadily increased during the past 20 years,[5,7-9] but in-hospital mortality has improved over time and currently ranges between 0.2% and 11%.[8-10]

Even in the absence of acute target-organ damage, episodes of severe hypertension have long-term implications. In a study involving 2435 patients with a previous transient ischemic attack, an isolated systolic blood pressure above 180 mm Hg (without symptoms) was associated with an increase in stroke risk during 3 years of follow-up by a factor of 5, as compared with no episodes of systolic blood pressure above 140 mm Hg, regardless of usual blood pressures.[11] Similarly, a prospective cohort study showed that patients who had an admission with hypertensive urgency had a 50% higher risk of fatal or nonfatal cardiovascular events than controls, despite similar blood-pressure levels during follow-up.[12]

In contrast to these long-term implications, hypertensive urgencies do not appear to be associated with adverse short-term outcomes.[13-16] Although rates of admission to the hospital are relatively high (up to 11% during the 30 days after initial presentation[10,15]), studies have not shown increased risks of adverse outcomes in the days to several months after patients were sent home from the office or emergency department.[13,15,16] A recent analysis of 58,535 ambulatory office encounters with patients who had a systolic blood pressure of 180 mm Hg or higher, a diastolic blood pressure of 110 mm Hg or higher, or both (mean, 182.5/96.4 mm Hg) showed a similar incidence of cardiovascular events at 6 months (0.9%) among patients who were hospitalized and among propensity-matched patients who were discharged after the encounter.[15]

STRATEGIES AND EVIDENCE

Figure 1 outlines a structured approach to the management of acute severe hypertension. The key elements include accurate measurement of blood-pressure levels; careful evaluation for potential precipitants, symptoms, and evidence of target-organ damage; and treatment decisions based on the presence of symptoms or acute target-organ damage.

BLOOD-PRESSURE MEASUREMENT

Blood pressure must be measured in both arms and the thigh using appropriate technique and validated devices[17] (Table S1 in the Supplementary Appendix, available with the full text of this article at NEJM.org). Most hospitals use automated devices that rely on oscillometric measurements. Two large registry studies comparing oscillometric and intraarterial measurements in critical care[18] or surgical[19] patients showed that oscillometric devices consistently underestimate blood-pressure levels by as much as 50/30 mm Hg when recorded intraarterial levels are above

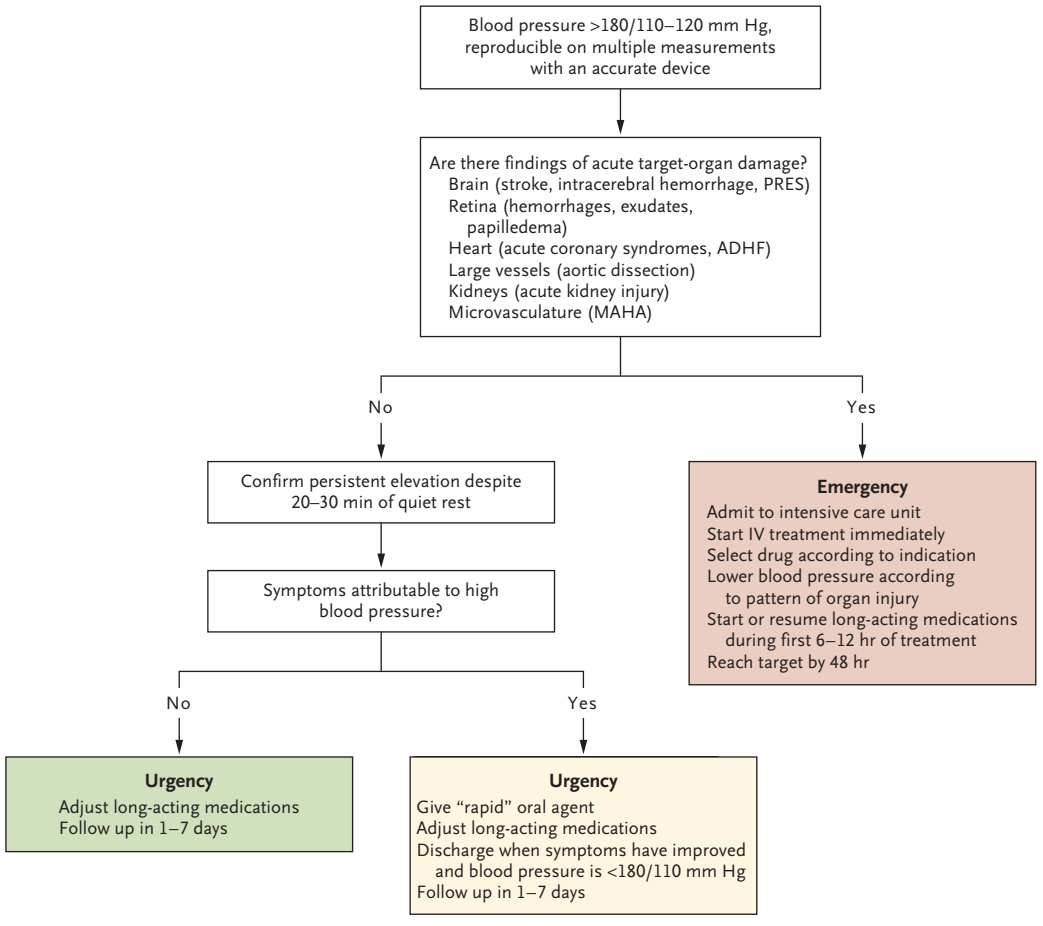

Figure 1. Evaluation and Management of Acute Severe Hypertension.

A focused evaluation of the targets of hypertension-mediated organ injury should be performed in every patient. With respect to the brain, inquire about focal motor or sensory deficits and speech or visual changes. Perform a neurologic examination, looking for motor deficits and gait, speech, and visual abnormalities. If there are positive findings, perform imaging with computed tomography (CT) or magnetic resonance imaging. With respect to the retina, perform funduscopy or retinal fundus photography, looking for evidence of microvascular injury (hemorrhages or exudates) or cerebral edema (papilledema). With respect to the heart, ask about chest pain, dyspnea, orthopnea, paroxysmal nocturnal dyspnea, palpitations, and edema. Look for signs of heart failure on examination (elevated jugular venous pressure, bibasilar rales, third heart sound, or edema). Perform chest radiography, electrocardiography (for ischemic changes), and troponin measurement in most patients. Acute coronary syndromes include unstable angina and myocardial infarction. With respect to large vessels (aorta), ask about chest or back pain. Obtain blood-pressure measurements in both arms and thigh, looking for asymmetry. If suspicion is aroused, obtain CT of the chest and abdomen with contrast or transesophageal echocardiography. With respect to the kidneys, measure the serum creatinine level to rule out acute kidney injury. Urinalysis may show proteinuria or hematuria as a sign of microvascular injury. With respect to the microvasculature, obtain a complete blood count, looking for anemia and thrombocytopenia suggestive of microangiopathy. ADHF denotes acute decompensated heart failure, IV intravenous, MAHA microangiopathic hemolytic anemia, and PRES posterior reversible encephalopathy syndrome. Adapted from Whelton et al.[1]

180/100 mm Hg. Auscultatory measurements that use aneroid or mercury devices also have substantial discordance from intraarterial measurements in high blood-pressure ranges,[20,21] even when meticulous technique is applied. Because of the potential underestimation of the severity of hypertension, the use of oscillometric (and auscultatory) devices should be discouraged

when evidence of target-organ damage is present and intravenous agents are required. In such cases, placement of an arterial catheter is indicated. In the absence of target-organ damage, noninvasive oscillometric or auscultatory devices are typically used to guide treatment, despite their limitations.

DETERMINATION OF PRECIPITATING FACTORS

Most patients presenting with acute severe hypertension are already known to be hypertensive and have received treatment.[15,22] Nonadherence to prescribed antihypertensive medications is the most common precipitating factor. In a large ambulatory database, three quarters of the patients who were evaluated for a systolic blood pressure of 180 mm Hg or higher or a diastolic blood pressure of 110 mm Hg or higher had a diagnosis of hypertension, and more than half had already been prescribed two or more antihypertensive agents.[15] In a prospective study involving patients with hypertension, nonadherence to medication was the strongest predictor of a hypertensive crisis.[23] These data underscore the importance of interventions that improve treatment adherence[24] (e.g., the use of patient monitoring of blood pressure, social-support opportunities,[25] and partnerships with health coaches, nurses, or pharmacists[25-27]), although studies are lacking to show that these interventions reduce the risk of acute severe hypertension.

Other common precipitating factors for acute severe hypertension include dietary sodium indiscretion; use of prescribed, over-the-counter, or illicit drugs (e.g., cocaine, amphetamines, sympathomimetic agents, nonsteroidal antiinflammatory drugs, and high-dose glucocorticoids); anxiety or panic; and acute stroke or heart failure, which can be both cause and consequence of severe hypertension. Patients with acute glomerulonephritis, preeclampsia, pheochromocytoma, or scleroderma renal crisis may present with acute severe hypertension. Among hospitalized patients, mobilization of infused intravenous fluids, withholding of antihypertensive medications, pain, and urinary retention are considered common precipitants. For patients presenting without a clear precipitant or who meet criteria for treatment-resistant hypertension during follow-up,[28] further testing should be considered for secondary causes of hypertension, such as renovascular disease, primary aldosteronism, glucocorticoid excess, pheochromocytoma, and, in younger patients, coarctation of the aorta.

EVALUATION OF ACUTE TARGET-ORGAN DAMAGE

A key part of the initial evaluation is the assessment of symptoms, signs, and diagnostic tests suggestive of acute target-organ damage (Fig. 1), including injury to the brain, heart, large vessels (aorta in particular), kidneys, and the microvasculature (including the retina). Diffuse microvascular injury (also known as "malignant hypertension") manifests as high-grade retinopathy, acute kidney injury, or microangiopathic hemolytic anemia and thrombocytopenia. These features may occur together or in isolation.

In the absence of symptoms to guide the evaluation, there are limited data on the yield of diagnostic tests. In a prospective study involving 167 patients in the emergency department with a triage diastolic blood pressure of 100 mm Hg or higher (mean, 194/112 mm Hg), routine metabolic panels revealed acute kidney injury requiring admission in 7% of patients.[29] In retrospective studies, the results of most diagnostic tests that are obtained in patients without evidence of acute target-organ damage have been normal or simply reflective of long-term exposure to hypertension.[14,15,30] Still, it is common practice to obtain a basic metabolic panel to assess renal function and electrolyte levels, a complete blood count to screen for microangiopathy, a urinalysis to identify proteinuria or hematuria, and an electrocardiogram and troponin levels to rule out asymptomatic myocardial injury.

Patients without target-organ damage are usually asymptomatic.[22,31,32] Symptoms, when present, may include headache, atypical chest pain, dyspnea, dizziness, lightheadedness, and epistaxis.

TREATMENT

AUTOREGULATION OF CEREBRAL BLOOD FLOW

Autoregulation of organ blood flow refers to physiological adaptations that allow organ perfusion to remain relatively constant across a wide blood-pressure range (Fig. 2). In the context of acute severe hypertension, flow autoregulation is most important; this autoregulation is best studied in the brain,[33] although the same principles are applicable to most end organs. In chronic severe hypertension, cerebral blood flow is maintained at similar levels as in normal per-

sons, but its autoregulatory curve is shifted to the right. This shift allows patients to tolerate higher blood-pressure levels without cerebral edema[33,34] but confers a predisposition to cerebral hypoperfusion at substantively higher blood-pressure levels than in normotensive persons, although these curves are neither consistent nor predictable at the individual level.[34,35] Limited data suggest that treatment of severe hypertension for several months may improve autoregulation to a modest extent,[34] whereas patients with mild-to-moderate hypertension (<180/110 mm Hg) recover autoregulatory responses within weeks after the initiation of effective therapy.[36]

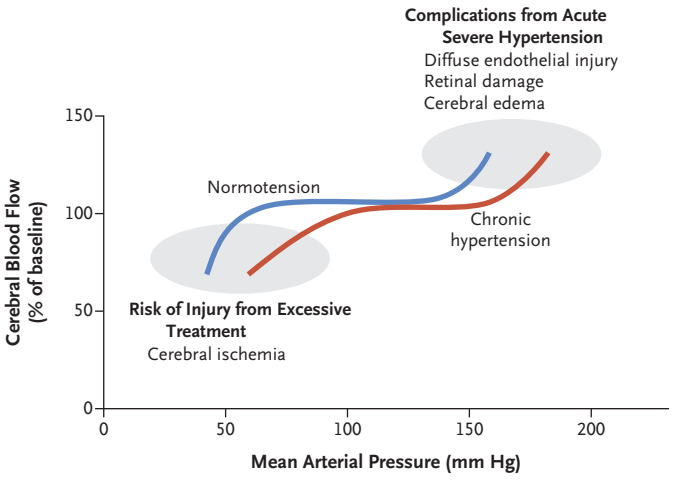

Figure 2. Autoregulation of Cerebral Blood Flow and Implications for the Treatment of Hypertensive Emergencies.

Cerebral blood flow is relatively stable across a wide blood-pressure range through changes in cerebrovascular resistance. On the high end of the curve, increased cerebrovascular resistance prevents pressure-induced increases in blood flow. Once the upper limit of autoregulation is crossed, small changes in blood pressure produce substantial increases in blood flow that result in vasogenic cerebral edema and its complications. On the low end of the curve, decreased cerebrovascular resistance allows flow to be maintained despite progressively lower blood pressure. Once the autoregulatory limit is reached, small decreases in blood pressure produce substantial impairment in cerebral perfusion. In clinical experiments, the lower limit of autoregulation occurs at blood-pressure levels approximately 25% lower than baseline. Symptoms of cerebral hypoperfusion develop when cerebral blood flow falls by more than approximately 30%. There is wide individual variability in autoregulatory limits and thresholds at both ends of the curve. The autoregulatory curve is shifted to the right in uncontrolled hypertension. Treatment of hypertension for weeks to months may improve or correct the autoregulatory abnormalities, especially in patients without long-standing severe hypertension.

CHOICE OF TREATMENT

There are relatively few trials comparing different agents for hypertensive emergency and hypertensive urgency.[37-39] Treatment is largely determined by an understanding of the pathophysiological features, the presence and type of target-organ injury, the availability and costs of medications, and physician experience with given agents.[10,40] There is considerable variability in practice regarding the choice of medications.[10,40]

HYPERTENSIVE EMERGENCIES

All patients should be admitted to an intensive care unit and treated with intravenous antihypertensive drugs on the basis of the clinical scenario (Tables 1 and 2 and Table S2). In the United States, labetalol, nitroglycerin, nicardipine, hydralazine, and nitroprusside are the most commonly used agents.[10] Of these medications, hydralazine has unpredictable effects, often leads to excessive blood-pressure lowering,[41] and should generally be avoided as a first option.[1,3] Studies comparing labetalol and nicardipine have shown faster achievement of blood-pressure control and less variability in blood pressure (allowing blood pressure to stay closer to target) with nicardipine but no significant differences in adverse events or mortality.[39,42,43] In one trial comparing clevidipine with nicardipine, clevidipine resulted in less variability than nicardipine.[38]

In the absence of studies comparing different rates of blood-pressure reduction, management is guided by autoregulatory principles; guidelines recommend that blood pressure be decreased by no more than 20 to 25% during the first hour and then to 160/100 to 110 mm Hg during the ensuing 2 to 6 hours.[1] Excessive blood-pressure reduction (resulting in systolic blood pressure below 100 to 120 mm Hg) may occur in up to 10% of patients[10,40] and is associated with an increased risk of death.[44,45] If excessive blood-pressure reduction occurs, prompt discontinuation of intravenous drugs and, in some cases, temporary use of vasopressors, intravenous fluids, or both is indicated. Resumption or initiation of long-acting antihypertensive drugs should take place alongside intravenous therapy to provide a smoother transition, shorten the need for intravenous drugs and intensive care, and minimize the risk of rebound hypertension, which is also associated with increased mortality.[44] The appropriate timing for starting or restarting oral drugs is uncertain; because the risk of hypoten-

Table 1. Treatment Strategies in Hypertensive Emergencies.*

Acute Target-Organ Damage	Timing for Acute BP Reduction†	Preferred Intravenous Drugs‡
Diffuse microvascular injury ("malignant hypertension")§	Decrease BP by 20–25% during first hr and to 160/100 mm Hg by 2–6 hr	Labetalol, nicardipine, nitroprusside
Hypertensive encephalopathy	Decrease BP by 20–25% during first hr and to 160/100 mm Hg by 2–6 hr	Labetalol, nicardipine, nitroprusside; avoid hydralazine
Acute intracerebral hemorrhage	If systolic BP is 150–220 mm Hg, decrease systolic BP to 140–150 mm Hg within 1 hr, particularly in patients without known hypertension and those with underlying vascular abnormalities, such as aneurysms or arteriovenous malformations. In patients with large hematoma volume and evidence of increased intracranial pressure, BP management should be more liberal (keep systolic BP <180 mm Hg). Lowering systolic BP below 140 mm Hg may be harmful.	Labetalol, nicardipine, clevidipine, nitroprusside; avoid hydralazine
Acute ischemic stroke	If thrombolytic therapy is indicated, decrease BP to <185/110 mm Hg before giving thrombolytic agents and maintain BP <180/105 mm Hg for the first 24 hr. If thrombolytic therapy is not indicated and there is no acute target-organ damage other than stroke, the strategy depends on BP. If BP is <220/120 mm Hg, no intervention is indicated for the first 48–72 hr. If BP is ≥220/120 mm Hg or there is other acute target-organ damage, such as heart failure or myocardial infarction, decrease BP by 15% within 1 hr.	Labetalol, nicardipine, clevidipine, nitroprusside; avoid hydralazine
Acute coronary syndromes	Decrease systolic BP to <140 mm Hg within 1 hr; keep diastolic BP >60 mm Hg	Nitroglycerin, labetalol, esmolol, metoprolol; avoid hydralazine
Acute heart failure	Decrease systolic BP to <140 mm Hg within 1 hr	Nitroglycerin, nitroprusside; loop diuretics needed in most cases; enalaprilat or hydralazine may be useful; avoid beta-blockers
Aortic dissection	Decrease both systolic BP to <120 mm Hg and heart rate to <60 beats/min within 20 min	Esmolol (or labetalol) plus one of nicardipine, clevidipine, nitroprusside, or nitroglycerin; both a beta-blocker (unless bradycardia is already present) and a vasodilator should be used

* BP denotes blood pressure.
† Initiation, reinstatement, or adjustments of long-acting oral antihypertensive medications should take place during the first 6 to 12 hours. Oral drugs are chosen on the basis of conventional guidelines. Intravenous medications can be tapered on the basis of observed blood-pressure levels after the addition of oral agents.
‡ Nitroprusside continues to be a recommended agent by most guidelines. Given its potential toxicity, it should be avoided as a first choice when other options are available.
§ Diffuse microvascular injury is identified as high-grade retinopathy (hemorrhages, exudates, or papilledema), acute kidney injury, or microangiopathic hemolytic anemia or thrombocytopenia, present alone or in combination.

Table 2. Most Commonly Used Intravenous Drugs for the Treatment of Hypertensive Emergencies.*

Drug	Class	Dose	Onset and Offset of Action	Cautions
Nicardipine	Dihydropyridine calcium-channel blocker, vasodilator	5–15 mg/hr IV drip; adjust by 2.5 mg/hr every 5–15 min	Rapid onset (2–5 min); duration of action, 1–4 hr; adjust dose more slowly (every 15 min) in patients with impaired renal or liver function	Contraindicated in patients with acute coronary ischemia (because of reflex tachycardia)
Clevidipine	Dihydropyridine calcium-channel blocker, vasodilator	1–16 mg/hr IV drip; double dose every 90 sec, more slowly as BP approaches goal	Rapid onset (2–4 min) and offset (5–15 min) of action	Contraindicated in patients with acute coronary ischemia (because of reflex tachycardia) and in patients with allergy to soy or eggs; may increase triglyceride levels (it is a lipid emulsion)
Labetalol	Combined nonselective beta-blocker and alpha-1 blocker (IV beta-to-alpha blocking ratio, approx. 7:1)	10–20 mg IV bolus followed by drip; may repeat at same dose or double dose after 10 min before starting drip; 0.5–10 mg/min IV drip; adjust drip every 15 min; for intermittent boluses, 10–80 mg IV every 10 min (maximum cumulative dose, 300 mg)	Rapid onset (5 min) but prolonged duration of action (3–6 hr, sometimes longer at higher doses)	Contraindicated in patients with heart failure, bradycardia or heart block, asthma or severe airway reactivity; caution in cocaine overdose
Esmolol	Selective beta-1 blocker	500–1,000 μg/kg bolus, followed by 100–300 μg/kg/min IV drip; adjust drip by 50 μg/kg/min every 5 min	Rapid onset (2–10 min) and offset (10–30 min) of action	Contraindicated in patients with heart failure, bradycardia or heart block, asthma, cocaine overdose
Metoprolol	Selective beta-1 blocker	2.5–5 mg IV every 5 min (maximum total dose, 15 mg), then every 4–6 hr	Onset, 15 min; peak, 30–60 min; duration of action, 4–6 hr; not a very potent antihypertensive	Contraindicated in patients with heart failure, bradycardia or heart block, asthma, cocaine overdose
Nitroglycerin	Nitrate donor, mixed venous and arteriolar dilator with predominant venous effects	10–400 μg/min IV drip; adjust by 10–20 μg/min every 5–15 min	Rapid onset and offset (5–10 min) of action; with prolonged use, a higher dose may be needed for same effect	Contraindicated in patients with right ventricular infarction
Nitroprusside	Direct vasodilator with predominant arteriolar effects	0.25–10 μg/kg/min IV drip; adjust by 0.5 μg/kg/min every 5 min	Rapid onset and offset (1–2 min) of action	Contraindicated in pregnancy (risk of fetal cyanide toxicity); use with impaired renal function may lead to thiocyanate toxicity, and use in liver disease may lead to cyanide intoxication, which may occur with higher doses or prolonged infusions; may cause methemoglobinemia (dose-dependent), increased intracranial pressure
Hydralazine	Direct arterial vasodilator	5–20 mg IV every 15–20 min, then every 3–4 hr (maximum dose, 20 mg per dose)	Onset and duration of action variable, depending on acetylator status; usual onset within 20 min and peak within 1 hr; duration of action, 3–6 hr; often demands a beta-blocker or other negative chronotropic agent to control reflex tachycardia	Use should be avoided in most cases; may worsen ischemia (owing to reflex tachycardia and possible coronary steal); unpredictable BP responses, often excessive

* This table is restricted to agents available in the United States. Detailed information is provided in Table S2 in the Supplementary Appendix. IV denotes intravenous.

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sion is greatest in the first 6 hours of intravenous therapy, a reasonable approach is to start oral agents 6 to 12 hours after starting intravenous therapy.[44] Long-acting drugs are chosen on the basis of standard guidelines for chronic hypertension management.[1,2] After initial stabilization, experience indicates that most patients can tolerate normalization of blood pressure within 48 to 72 hours, although some require longer periods owing to dizziness, fatigue, or mental slowness.

The recommended pace and intensity of blood-pressure reduction vary depending on the presence of certain conditions, particularly aortic dissection, eclampsia, pheochromocytoma crisis, and intracerebral hemorrhage, all of which demand more aggressive approaches to limit ongoing injury.[1,3,46] Ischemic stroke[1,3,47] requires more conservative management to avoid peri-infarction hypoperfusion and worse stroke outcomes. Consensus recommendations are based on very limited data and in some cases are not uniform across guidelines.[1,3,48,49]

HYPERTENSIVE URGENCIES

Most patients without acute target-organ damage can be cared for as outpatients.[13-16] Treatment with guideline-concordant long-acting medications[1,2] should be started, reinstated, or adjusted, and follow-up should be scheduled within 1 to 7 days.

In a study involving more than 500 patients presenting to an emergency department with severe hypertension, blood pressure fell to less than 180/110 mm Hg after 30 minutes of quiet rest (before medication administration) in approximately one third of the patients.[13] If quiet rest or control of anxiety or other precipitating factors is insufficient, an oral antihypertensive agent may be given. Intravenous medications are discouraged in this context.

For patients with symptoms that are presumed to relate to hypertension but are not indicative of target-organ damage (e.g., headache, atypical chest pain, or epistaxis), it is reasonable to choose an oral agent with a faster onset of action, such as clonidine (0.1 to 0.3 mg), labetalol (200 to 400 mg), captopril (25 to 50 mg), prazosin (5 to 10 mg), or nitroglycerin 2% topical ointment (1 to 2 in.). Nifedipine (given orally or sublingually) should be avoided owing to unpredictable blood-pressure reduction, possibly resulting in cardiovascular events.[50] Medications can be administered every 30 minutes until the target blood pressure is achieved. A systematic review of comparative trials and cohort studies suggested similar acute blood-pressure reductions with different agents.[51] Clinical experience and descriptions of the acute effects of clonidine and labetalol suggest that they may be associated with less abrupt blood-pressure changes than other agents.[52-54] Patients are generally discharged once symptoms have improved, which often coincides with a decrease in blood pressure to a level below 160 to 180/100 to 110 mm Hg.

AREAS OF UNCERTAINTY

Large randomized trials are lacking to identify the most effective treatment for hypertensive urgencies and emergencies generally and for specific underlying conditions. For previously untreated patients who present to the emergency department, there is controversy regarding whether antihypertensive medication should be prescribed at discharge.[55] The American College of Emergency Physicians currently recommends initiation of therapy in the emergency department only for selected patients who are likely to have poor follow-up and recommends referral without initiation of treatment in the rest. Although there is reasonable concern about inappropriate treatment of normotensive patients, withholding treatment may represent a missed opportunity to minimize risk.

GUIDELINES

Recommendations for the management of acute severe hypertension are included in major U.S. and European hypertension guidelines.[1-3] There are variations in terminology and specific blood-pressure thresholds, but all the guidelines acknowledge the critical role of acute target-organ damage and adopt blood-pressure thresholds of 180/110 to 120 mm Hg to define urgencies and emergencies (Table S3). There is general agreement on the pace of blood-pressure reduction and the need for the use of intravenous drugs in an intensive care environment for the management of hypertensive emergencies. The approach proposed in this article is generally consistent with these guidelines.

CONCLUSIONS AND RECOMMENDATIONS

The patient described in the vignette has acute severe hypertension complicated by posterior reversible encephalopathy syndrome, a hypertensive emergency precipitated by nonadherence to antihypertensive therapy. She should be admitted to the intensive care unit and immediately begin treatment with continuous intravenous antihypertensive therapy guided by invasive intraarterial blood-pressure monitoring. Nicardipine (or clevidipine) and labetalol are the preferred agents in this context. Given her relative bradycardia, nicardipine would be my choice. Although data are lacking to guide the appropriate pace of blood-pressure reduction, I would lower her blood pressure by approximately 20 to 25% in the first hour and would aim for a blood pressure of approximately 160/100 mm Hg by 6 hours. If she has a good response and relative hypotension does not develop, I would restart amlodipine and lisinopril at that point. Because stepped additions may reduce the risk of excessive blood-pressure reduction, I would restart her diuretic the following day if needed. I would wean the nicardipine over a period of 18 to 36 hours, although the timing should be guided by close blood-pressure monitoring. I would discharge her once her symptoms improve and hypertension is controlled for at least 24 hours without the use of intravenous therapy, with arrangements for a follow-up office appointment within 1 week. I would not pursue an evaluation for secondary hypertension unless her blood pressure remained uncontrolled at follow-up.

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