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Hypertensive Urgency and Emergency

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A 52-year-old man presented to the emergency department complaining of worsening occipital headache and confusion. He reported experiencing numbness and weakness involving the right side of his body as well as blurry vision over the past 12 hours. His past medical history was pertinent for hypertension, bilateral renal artery stenosis, cocaine abuse, and hyperlipidemia. On arrival, his blood pressure was 213/134 mm Hg. On physical examination, he was confused. Papilledema was seen on fundoscopic examination. He had mild motor weakness (4/5) in the right upper extremity. Laboratory studies revealed the following: serum potassium, 3.1 mEq/L; blood urea nitrogen, 36 mg/dL; and serum creatinine, 2.5 mg/dL (baseline creatinine, 1.5 mg/dL). Electrocardiogram revealed left ventricular hypertrophy by voltage criteria and nonspecific ST-T wave abnormalities in the lateral leads. Computed tomography scan of the head without contrast revealed diffuse bilateral white matter changes consistent with hypertensive encephalopathy. The patient was admitted to the intensive care unit and started on intravenous nitroprusside. Blood pressure decreased to 190/100 mm Hg over the first 3 hours and neurologic symptoms resolved within 5 hours. He was switched to his usual oral regimen on the third day of hospital admission and was discharged home on the fifth day with controlled blood pressure.

Hypertension is an extremely common disorder in modern Western societies, with an age- and sex-adjusted prevalence of approximately 28% in North America.[1] Physicians in clinical practice are likely to encounter patients with hypertensive urgency and emergency. Although improved management of chronic hypertension has decreased the lifetime incidence of hypertensive crisis to less than 1%, patients presenting with severe hypertension represent up to 25% of all patients presenting to urban emergency departments.[2] One-year and 5-year mortality following untreated hypertensive emergency are 70% to 90% and 100%, respectively.[3] With adequate blood pressure control, these mortality rates decrease to 25% and 50%, respectively.[3] This article reviews the approach to appropriately diagnosing and managing hypertensive urgency and emergency.

DEFINITION

Hypertensive emergency (crisis) is characterized by a severe elevation in blood pressure (> 180/120 mm Hg) complicated by evidence of impending or progressive target organ dysfunction.[4] Examples of target organ dysfunction include coronary ischemia, disordered cerebral function, cerebrovascular events, pulmonary edema, and renal failure. *Hypertensive urgency*, on the other hand, is a severe elevation in blood pressure without progressive target organ dysfunction.[4] Notably, these definitions do not specify absolute blood pressure levels as hypertensive urgency or emergency may occur with a modest increase in blood pressure in previously normotensive persons (eg, during pregnancy or with acute cocaine intoxication).

ETIOLOGY AND PATHOPHYSIOLOGY

Severe elevations in blood pressure may develop de novo or may complicate underlying essential or secondary hypertension (**Table 1**). In white patients, 20% to 30% of cases of hypertensive urgency or emergency are secondary to uncontrolled essential hypertension, while in black patients, the percentage is as high as 80%.[5]

The initiating factor in hypertensive emergency and urgency is poorly understood. A rapid rise in blood pressure associated with increased systemic vascular resistance is thought to be the triggering event.[6] The rate of change in blood pressure is directly related to the likelihood that

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TAKE HOME POINTS

- Distinguishing between hypertensive emergency (associated with acute target organ damage) and urgency (no target organ damage) is crucial to appropriate management.
- Diagnosis of hypertensive emergency requires a thorough history (evidence of target organ damage, illicit drug use, and medication compliance) as well as a complete physical examination, basic laboratory data, and electrocardiogram to assess for the presence of target organ damage and determine its severity.
- In general, hypertensive urgency is managed using oral antihypertensive drugs in outpatient or same-day observational settings, while hypertensive emergency is managed in an intensive care unit or other monitored settings with parenteral drugs.
- The initial goal in hypertensive urgency is a reduction in mean arterial pressure by no more than 25% within the first 24 hours using conventional oral therapy; in hypertensive emergency, mean arterial pressure should be reduced approximately 10% during the first hour and an additional 15% within the next 2 to 3 hours.
- Various medications are available for the treatment of hypertensive emergency; specific target organ involvement and underlying patient comorbidities dictate appropriate therapy.

an acute hypertensive syndrome will develop, with rapid increases over a short period of time increasing the likelihood of a syndrome.[7] The endothelium plays a central role in blood pressure homeostasis, mainly by modulating vascular tone via secretion of substances such as nitric oxide and prostacyclin.[8] Stretch of the vessel wall during significant blood pressure elevation causes activation of the renin–angiotensin system, which also appears to be an important factor in the development of severely elevated blood pressure. When there is a sustained or severe elevation in blood pressure, the compensatory endothelial vasodilatory response is turned off, leading to endothelial decompensation, which results in a further rise in blood pressure and endothelial damage. This process leads to a self-sustaining cycle, resulting in a progressive increase in resistance and further endothelial dysfunction.[9] The **Figure** outlines the underlying pathophysiology of hypertensive emergency.

Table 1. Causes of Hypertensive Emergency

Essential hypertension
Renal disease
Parenchymal disease
Chronic pyelonephritis
Primary glomerulonephritis
Vascular/glomerular disease
Systemic lupus erythematous
Systemic sclerosis
Renal vasculitides (microscopic polyarteritis nodosa, Wegener's granulomatosis)
Tubulointerstitial nephritis
Renovascular disease
Renal artery stenosis
Fibromuscular dysplasia
Atherosclerotic renovascular disease
Macroscopic polyarteritis nodosa
Drugs
Abrupt withdrawal of a centrally acting α_2-adrenergic agonist (clonidine, methyldopa)
Phencyclidine, cocaine or other sympathomimetic drug intoxication
Interaction with monoamine oxidase inhibitors (tranylcypromine, phenelzine, and selegiline)
Pregnancy
Eclampsia/severe pre-eclampsia
Endocrine
Pheochromocytoma
Primary aldosteronism
Glucocorticoid excess
Renin-secreting tumors
Central nervous system disorders
CVA infarction/hemorrhage
Head injury

Adapted with permission from Kitiyakara C, Guzman NJ. Malignant hypertension and hypertensive emergencies. J Am Soc Nephrol 1998;9:135.

CVA = cerebrovascular accident.

DIAGNOSIS

Distinguishing between hypertensive emergency (associated with acute target organ damage) and urgency (no target organ damage) is a crucial step in appropriate management of these conditions as management differs between them. The history and physical examination are extremely important in determining the severity of an acute hypertensive crisis syndrome and guiding further management. Laboratory data and

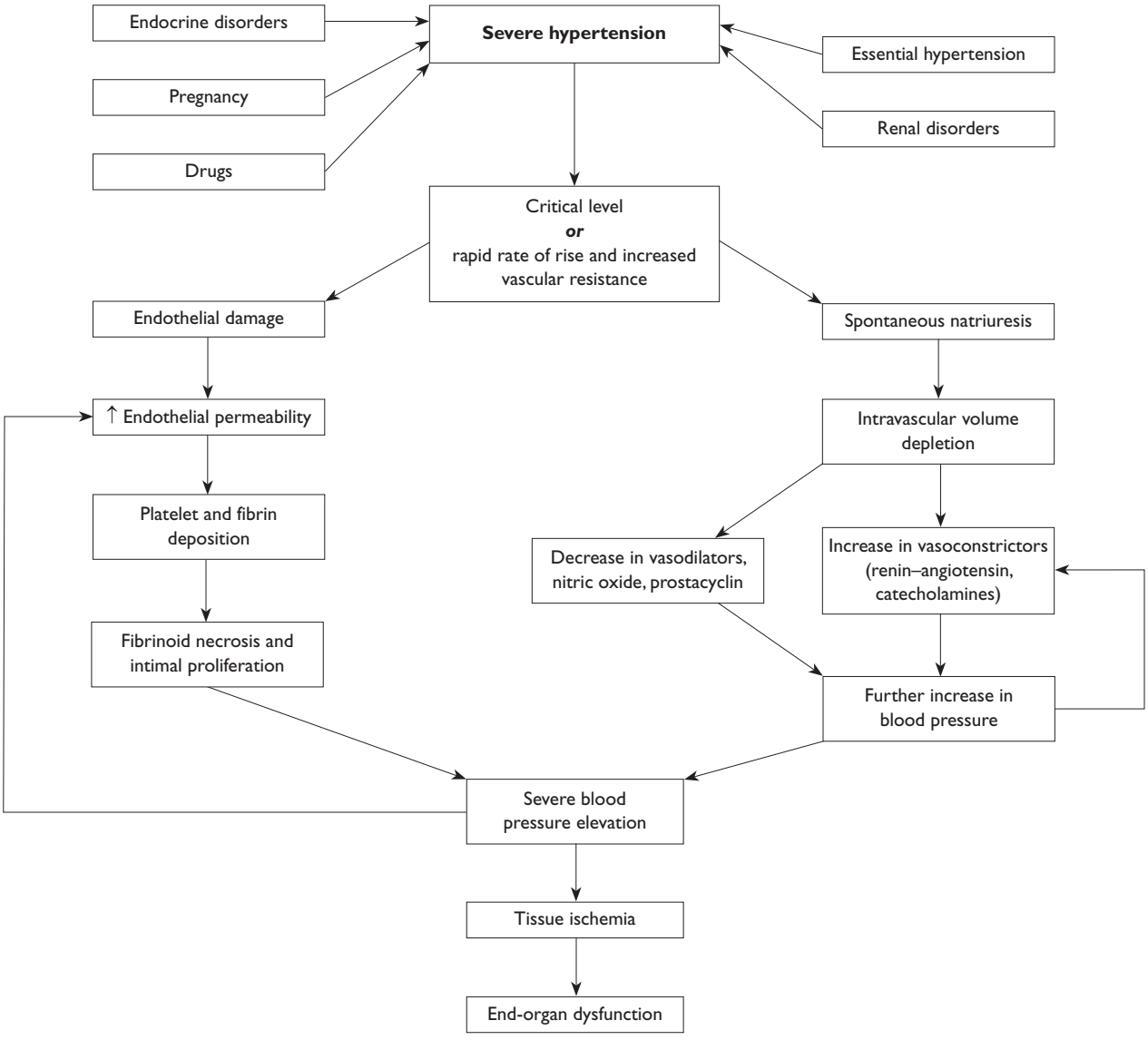

Figure. Pathophysiology of hypertensive emergencies. (Adapted with permission from Kitiyakara C, Guzman NJ. Malignant hypertension and hypertensive emergencies. J Am Soc Nephrol 1998;9:135.)

other diagnostic tests such as electrocardiogram and chest radiograph can provide important information regarding possible end-organ damage.

History

The history should include information regarding when the patient was diagnosed with hypertension; baseline blood pressure; the presence of previous end-organ damage, in particular renal and cerebrovascular damage; details regarding antihypertensive therapy and compliance with the regimen; intake of over-the-counter medications (sympathomimetics, nonsteroidal anti-inflammatory drugs, certain herbal products); and illicit drug use (cocaine, methamphetamine, ephedra). Patients should be asked specifically whether they abruptly stopped taking β blockers or central sympatholytic agents as abrupt cessation of these medications may lead to rebound hypertension. In addition, patients should be asked about symptoms suggestive of end-organ compromise, including chest pain (myocardial ischemia/infarction, aortic dissection), shortness of breath (acute pulmonary edema secondary to left ventricular failure), back pain (aortic dissection), and neurologic symptoms such as headache and blurry vision.

Neurologic symptoms may be due to intracerebral or subarachnoid hemorrhage or hypertensive encephalopathy. Hypertensive encephalopathy is an acute organic brain syndrome or delirium related to cerebral edema believed to result from loss of cerebral vascular autoregulatory function in the setting of severely elevated blood pressure. It is characterized by headache, nausea, and vomiting initially, followed by altered mental status and/or seizure if hypertension is not treated.

Physical Examination

Blood pressure should be measured in both arms as a significant discrepancy between arms (> 20 mm Hg in systolic blood pressure) is suggestive of aortic dissection. In addition, blood pressure should be measured in both the supine and standing positions to assess volume status as patients presenting with hypertensive emergency may be intravascularly volume depleted due to pressure natriuresis. Head and neck examination must include a complete fundoscopic examination, as grades III (flame-shaped hemorrhages, fluffy, white cotton wool spots, and yellow-white exudates) and IV (papilledema with blurring of the disk margins accompanied by hemorrhages and exudates) retinopathy are the hallmarks of hypertensive emergency.

Cardiovascular examination includes auscultation for new murmurs. A diastolic murmur consistent with aortic insufficiency may support the diagnosis of aortic dissection. Mitral regurgitation may develop secondary to ischemic rupture of a papillary muscle. Signs suggestive of heart failure (elevated jugular venous pressure, S_3 gallop) also should be sought. The presence of rales on pulmonary examination suggests vascular congestion and pulmonary edema. Evidence of atherosclerotic disease in any vascular bed, especially in smokers, should heighten suspicion for renovascular hypertension due to critical renal artery stenosis. A systolic/diastolic abdominal bruit suggests renovascular disease as the underlying cause of hypertension.

A careful neurologic examination should always be completed. The presence of focal neurologic signs indicate ischemic or hemorrhagic stroke. Delirium or a flapping tremor is suggestive of hypertensive encephalopathy. Hypertensive encephalopathy is a diagnosis of exclusion; other causes that must be ruled out include stroke, subarachnoid hemorrhage, and mass lesions.

Laboratory Testing

Baseline investigations include a complete blood count with peripheral smear for the presence of schistocytes, which may suggest microangiopathic hemolytic anemia. Serum electrolytes, blood urea nitrogen, and serum creatinine concentrations should be measured to evaluate for renal impairment. Hypokalemic metabolic alkalosis may be seen as a result of intravascular volume depletion and secondary hyperaldosteronism. Comparison of the measured serum creatinine value with baseline values should be done to evaluate for the presence of acute and/or chronic kidney disease.

Other Diagnostic Tests

An electrocardiogram should be obtained in all patients with hypertensive crisis as it may reveal evidence of myocardial ischemia or infarction in the acute setting as well as evidence of left ventricular hypertrophy due to chronic hypertension. A chest radiograph should be obtained to evaluate for pulmonary vascular congestion as well as a widened mediastinum, which suggests aortic dissection. Urinalysis and urine sediment examination to evaluate for hematuria and/or cellular casts also should be done. A computed tomography scan of the head without contrast should be performed in any patient with neurologic symptoms, which include change in mental status or focal neurologic signs suggestive of a cerebrovascular accident or hemorrhage.

MANAGEMENT

An important issue in the management of patients with hypertensive urgency or emergency is how quickly and to what degree to lower the blood pressure; however, there are no high-quality prospective studies that address these questions. Management should be tailored to the individual patient based not only on absolute blood pressure number, but also on the presence or absence of end-organ damage.[6]

Hypertensive Urgency

General principles. In general, hypertensive urgency can be managed using oral antihypertensive agents in an outpatient or same-day observational setting, although this approach may not be appropriate when patient follow-up is difficult or unpredictable. Treatment is initiated with very low doses of oral agents using incremental doses as needed and avoiding large starting doses that may result in excessive blood pressure reduction. Avoiding excessive reduction is especially important in patients who are at highest risk for hypotensive complications, such as the elderly, patients with severe peripheral vascular disease, and those with known severe atherosclerotic cardiovascular and intracranial disease. The initial goal is to reduce blood pressure to 160/110 mm Hg over several hours to days using conventional oral therapy.[10] Mean arterial pressure should be reduced by no more than 25% within the first 24 hours

using conventional oral therapy. Antihypertensive agents used to treat hypertensive urgency are described below.

Specific agents. Captopril is an angiotensin-converting enzyme (ACE) inhibitor with an onset of action beginning within 15 to 30 minutes and a maximum drop in blood pressure occurring between 30 and 90 minutes. Captopril is given as a 25-mg oral dose initially, followed by incremental doses of 50 to 100 mg 90 to 120 minutes later. Significant adverse effects include cough, hypotension, hyperkalemia, angioedema, and renal failure (especially in patients with bilateral renal artery stenosis, in whom it should be avoided).

The calcium channel blocker nicardipine is one of the few therapies used in the setting of hypertensive urgency that has been evaluated in a randomized controlled trial and has demonstrated statistical superiority over placebo. In a study that randomly assigned 53 patients with urgent hypertension to nicardipine or placebo, effective blood pressure control was observed in 65% of the treatment group compared with 22% of the placebo group ($P = 0.002$).[11] The usual oral dose is 30 mg, which can be repeated every 8 hours until the target blood pressure is achieved. Onset of action is ½ to 2 hours. Common adverse reactions include palpitations, flushing, headache, and dizziness.[11]

Labetalol has mixed α_1- and β-adrenergic blocking properties and an onset of action within 1 to 2 hours. A wide dose range has been studied in different populations, making generalization difficult. In a randomized study of 36 patients, groups receiving 100, 200, or 300 mg orally had significant decreases in both systolic and diastolic blood pressure.[12] In general, the starting dose is 200 mg orally, which can be repeated every 3 to 4 hours.[12] Common side effects include nausea and dizziness.

Clonidine is a central sympatholytic (α_2-adrenergic receptor agonist) agent with an onset of action within 15 to 30 minutes and a peak effect within 2 to 4 hours. A typical oral regimen is a 0.1 to 0.2 mg loading dose followed by 0.05 to 0.1 mg every hour until target blood pressure is achieved, up to a maximum dose of 0.7 mg. Common side effects include sedation, dry mouth, and orthostatic hypotension.

Nifedipine is a calcium channel blocker with a peak effect within 10 to 20 minutes. Short-acting nifedipine is not approved by the US Food and Drug Administration for treating hypertension, and major concerns have been raised over its safety in the treatment of hypertensive urgency due to reports of unpredictable drops in blood pressure and associated risk of stroke.[13,14] In 1995, an ad hoc panel convened by the National Heart, Lung, and Blood Institute to review evidence regarding the safety of calcium channel blockers concluded that "short-acting nifedipine should be used with great caution (if at all), especially at higher doses, in the treatment of hypertension."[15]

Hypertensive Emergency

General principles. Treatment of hypertensive emergency is tailored to each individual case based on the extent of end-organ damage as well as other comorbid conditions (**Table 2** and **Table 3**). Blood pressure management in this setting requires the use of parenteral drugs, as precise and rapid control of blood pressure is critical. These patients should always be managed in an intensive care unit or other settings that allow continuous monitoring of blood pressure. The ideal rate of blood pressure lowering is unclear, but reducing the mean arterial pressure by 10% during the first hour and an additional 15% within the next 2 to 3 hours has been recommended.[16] More rapid reduction in blood pressure may result in cardiac or cerebrovascular hypoperfusion.

Pressure natriuresis may cause volume depletion in patients with hypertensive emergency, and administering vasodilator medications to these patients can lead to precipitous drops in blood pressure. Patients with volume depletion should receive intravenous (IV) saline to restore intravascular volume and shut off the renin-angiotensin-aldosterone system. Management of specific emergencies is discussed in the following sections.

Neurologic emergency. Common neurologic emergencies in the setting of hypertensive crisis include hypertensive encephalopathy, intracerebral hemorrhage, and acute ischemic stroke. Severe hypertension is very common in the setting of acute stroke, and there is controversy surrounding the goal blood pressure. In intracerebral hemorrhage, there is typically disruption of the cerebral autoregulation of blood flow in the area of the bleed, and blood flow and oxygen delivery are dependent on systemic perfusion pressure. The American Heart Association recommends treating hypertension in the setting of an intracerebral bleed only when blood pressure is more than 180/105 mm Hg.[17] Mean arterial pressure should be maintained below 130 mm Hg.[17]

In patients with ischemic stroke, perfusion pressure distal to the obstructed vessel is low, and compensatory vasodilatation of these blood vessels occurs to maintain adequate blood flow. A higher systemic pressure is required to maintain perfusion in these dilated blood vessels. Most patients, irrespective of pre-ischemic blood pressure control, experience a sustained rise in

Table 2. Parenteral Drugs Used for Treatment of Hypertensive Emergencies

Agent	Mechanism of Action	Dose	Onset	Duration of Action	Adverse Effects/Precautions
Sodium nitroprusside	Nitric oxide compound, direct arterial and venous vasodilator	0.25–10 µg/kg/min IV infusion	Immediate	2–3 min after infusion	Nausea, vomiting, Thiocyanate and cyanide intoxication Increased intracranial pressure Methemoglobinemia Delivery sets must be light resistant
Fenoldopam mesylate	Dopamine-1 receptor agonist	0.1–0.3 µg/kg/min IV infusion	< 5 min	30 min	Headache, flushing, tachycardia Local phlebitis Mild tolerance after prolonged infusion May reduce serum potassium ECG changes: nonspecific T-wave changes/ventricular extra systoles
Nitroglycerin	Nitric oxide compound; direct arterial and venodilator (mainly venous)	5–100 µg/min IV infusion	2–5 min	5–10 min	Headache, tachycardia, flushing Methemoglobinemia Requires special delivery system due to drug binding to tubing
Enalaprilat	ACE inhibitor	0.625–2.5 mg every 6 hr IV	Within 30 min	12–24 hr	Acute renal failure in patients with bilateral renal artery stenosis Prolonged half-life
Hydralazine	Direct vasodilation of arterioles with little effect on veins	5–20 mg IV bolus or 10–40 mg IM; repeat every 4–6 hr	10 min IV 20–30 min IM	1–4 hr IV	Tachycardia, flushing, headache Sodium and water retention Increased intracranial pressure Aggravation of angina
Nicardipine	Calcium channel blocker	5–15 mg/hr IV infusion	1–5 min	15–30 min, but may exceed 4 hr after prolonged infusion	Tachycardia, headache, flushing Local phlebitis Aggravation of angina
Esmolol	β-Adrenergic blocker	500 µg/kg bolus injection IV or 50–100 µg/kg/min by infusion. May repeat bolus after 5 min or increase infusion rate to 300 µg/kg/min	1–2 min	10–30 min	Hypotension, nausea Asthma First-degree atrioventricular block Heart failure
Labetalol	α-, β-Adrenergic blocker	20–80 mg IV bolus every 10 min; 0.5–2.0 mg/min IV infusion	5–10 min	3–6 hr	Bronchoconstriction Heart block Vomiting, scalp tingling Heart failure exacerbation
Phentolamine	α-Adrenergic receptor blocker	5–15 mg IV bolus	1–2 min	10–30 min	Tachycardia, flushing, headache

Adapted with permission from Vidt D. Hypertensive crises: emergencies and urgencies. The Cleveland Clinic disease management project. 12 Jan 2006. Available at www.clevelandclinicmeded.com/diseasemanagement/nephrology/crises/crises.htm. Accessed 2 Feb 2007; *and* National High Blood Pressure Education Program. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Bethesda (MD): Dept. of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute; 2004. NIH Publication No. 04–5230.

ACE = angiotensin-converting enzyme; ECG = electrocardiogram; IM = intramuscular; IV = intravenous.

blood pressure during cerebral ischemia, including transient ischemic attack. Therefore, in patients with ischemic stroke, blood pressure should be carefully observed for the first 1 to 2 hours to determine if it will spontaneously decrease. Only a persistently mean arterial pressure over 130 mm Hg or a systolic blood pressure over 220 mm Hg should be carefully treated. In this setting, mean arterial pressure should be lowered by 15% to 20%.[18]

Hypertensive encephalopathy is a severe end-organ manifestation of the hypertensive process. Gradual lowering of the blood pressure frequently leads to rapid improvement of neurologic symptoms. If patients do not improve within 6 to 12 hours, evaluation

for other causes of the encephalopathic process should be undertaken.

Cardiac emergency. Major cardiac emergencies in the setting of hypertensive crisis include acute myocardial ischemia or infarction, pulmonary edema, and aortic dissection. Patients presenting with a hypertensive emergency involving myocardial ischemia or infarction are typically treated with nitroglycerin as older studies have shown that it reduces myocardial oxygen consumption and increases flow beyond the stenotic coronary area.[19] In the absence of significant heart failure, β-blockers (eg, labetalol, esmolol) also should be used to control blood pressure.

In the setting of acute aortic dissection, an IV β-blocker (eg, labetalol or esmolol) should be given first, followed by a vasodilating agent, classically IV nitroprusside. These agents are used to lower the systolic blood pressure to a goal of less than 120 mm Hg within 20 minutes. The order of administration is critical as giving vasodilators alone can lead to increased shear stress in the vessel wall as a result of increased dP/dt with vasodilatation as well as subsequent reflex tachycardia, increasing the risk of further dissection. Pharmacologic therapy is usually a temporary bridge to more definitive surgical treatment of dissection.

Typical treatment of pulmonary edema includes IV administration of diuretics followed by IV administration of an ACE inhibitor (usually enalaprilat) and nitroglycerin. Sodium nitroprusside may be used if these agents do not adequately control blood pressure.

Hyperadrenergic states. Patients with catecholamine excess in settings such as pheochromocytoma, cocaine or amphetamine overdose, monoamine oxidase inhibitor–induced hypertension, or clonidine withdrawal syndrome can present with hypertensive crisis syndrome. In pheochromocytoma, initial blood pressure control can be achieved with sodium nitroprusside (arterial vasodilator) or with IV phentolamine (ganglion-blocking agent).[20] β-Blockers may be added for improved blood pressure control but should never be used alone until α blockade is achieved as paradoxical hypertension may occur. Hypertension due to clonidine withdrawal is best treated initially with resumption of clonidine followed by the addition of other drugs described above. Benzodiazepines have become one of the first-line agents in the setting of cocaine intoxication. They reduce the heart rate and blood pressure through their anxiolytic effects and are therefore recommended in patients with cocaine-associated ischemia who are hypertensive, tachycardic, or anxious.

Kidney failure. Acute kidney injury can be a cause or a consequence of hypertensive emergency. Acute kidney injury can present with proteinuria, microscopic hematuria, oliguria, and/or anuria. Optimal treatment is controversial. Although IV nitroprusside is widely used, it can cause cyanide or thiocyanate toxicity. Parenteral fenoldopam mesylate (a dopamine-1 receptor agonist) has shown promising results and enhanced safety. Use of fenoldopam avoids potential cyanide or thiocyanate toxicity associated with infusion of nitroprusside in the setting of renal failure and also improves renal function as measured by creatinine clearance.[16]

Table 3. Drug of Choice in Treatment of Specific Types of Hypertensive Emergencies

Type of Emergency	Drug of Choice	Second-line Drugs
Neurologic		
Hypertensive encephalopathy	Nitroprusside	Labetalol or nicardipine
Subarachnoid hemorrhage	Nimodipine	Labetalol or nicardipine
CVA	Labetalol	Nitroprusside, enalaprilat
Renal		
Acute kidney injury	Nicardipine	Fenoldopam
Cardiac		
Aortic dissection	β-Blocker + nitroprusside	Labetalol, trimethaphan
Pulmonary edema	Nitroglycerin	Nitroprusside ± ACE inhibitor
Cardiac ischemia	Nitroglycerin ± β-blocker	Nitroprusside, labetalol
Adrenergic crisis		
Pheochromocytoma	Nitroprusside + β-blocker	Phentolamine
Cocaine		
Eclampsia	Methyldopa Magnesium sulfate (do not use with calcium channel blocker)	Hydralazine

Adapted with permission from Varon J, Marik PE. The diagnosis and management of hypertensive crises. Chest 2000;118:221.

ACE = angiotensin-converting enzyme; CVA = cerebrovascular accident.

CONCLUSION

Hypertensive urgency and emergency are associated with significant morbidity and mortality. Prompt recognition and early treatment is crucial in preventing or halting progressive target organ damage. Frequent monitoring that is typically only feasible in the intensive care unit is necessary to achieve appropriate

therapeutic endpoints. Treatment must be tailored to each patient, based on the presence of specific target organ damage and underlying comorbidities. The benefits of treating severe hypertension must be weighed against the risk of excessive blood pressure lowering. There are no high-quality prospective studies that address how quickly and to what degree blood pressure should be lowered. Extensive counseling should be provided to patients upon discharge, especially if noncompliance with medications contributed to the hypertensive crisis syndrome. **HP**

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