Managing Hypertensive Emergencies in the Urgent Care Setting

Urgent message: Health-care practitioners frequently see patients in urgent care centers who have elevated blood pressure. It is vital that they be able to identify hypertensive emergencies to immediately start lowering such patients' blood pressure and then transfer them to an emergency department, to avoid hypertensive damage to the brain, heart, and kidneys.

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Introduction

Elevated blood pressure (BP) is very common in the urgent care setting, oftentimes from pain or from chronic hypertension (HTN). HTN is generally defined as a sustained BP >140/90 mm Hg. It affects approximately 40% of patients presenting to U.S. emergency departments (EDs) each year.[1] Furthermore, it is very important to identify a very small subset of patients who meet the criteria for hypertensive emergency. That is the focus here: identifying and treating patients who have a true hypertensive emergency, as defined by a BP acutely elevated BP from baseline (no specific cutoff value but usually >180/120 mm Hg) plus *evidence of end-organ damage*. These patients need an appropriate organ-system work-up and should be referred immediately to an ED. Even though hypertensive emergencies make up only 0.2% of ED patient visits, it is extremely important not to miss this true emergency. HTN-related brain damage, either from hypertensive encephalopathy, hemorrhagic or ischemic stroke, or head trauma, make up approximately 40% to 50% of U.S. cases each year. Car-

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diac damage from severe HTN accounts for approximately 30% to 40% of U.S. cases each year and manifests as heart failure, cardiac ischemia, and acute pulmonary edema. Kidney damage (acute kidney injury, nephrosclerosis, or tubular necrosis) constitute about 10% to 15% of hypertensive emergencies.[2,3] Aortic dissection and eclampsia are less-prevalent causes of HTN syndromes that need aggressive emergency management.

Clinical Scenarios

Given that the brain, heart, and kidneys are the primary organs damaged in hypertensive emergencies, what follows are three clinical scenarios (one per organ system) that are based on actual patients presenting to an urgent care center or ED. The focus should be on identifying or ruling out life-threatening conditions. It is important to concentrate on red flags, or the lack thereof, that indicate end-organ damage. With this perspective, this article explores key points from the history of present illness, physical examination, diagnostic work-up, medical management, and appropriate disposition.

- **Case 1—painkiller for a headache:** A 79-year-old man with past medical history of HTN presents to an urgent care center with a 2-day history of headache. The patient states that currently he is "just fine" and only has a mild headache. He says he just needs a painkiller for his headache and would like to go home. He has been brought in by his daughter because she noted that earlier today during their phone conversation, he had slowed speech and decreased attentiveness. She states he is usually pretty good about taking his medications and is probably fine but wants him "checked out." The patient says that sometimes he "just gets tired" during the day and that his daughter is overreacting. The patient's BP is 217/103 mm Hg (mean arterial pressure [MAP], 141 mm Hg), and he states it has been running a little high lately, but the daughter says, "Not that high."
- **Case 2—just a prescription refill:** A 68-year-old obese woman with past medical history of "only mild heart failure" says that she could not get an appointment to see her primary-care provider this week and she is "here just for a medication refill." She emphasizes that she only needs a simple refill prescription because she has been out of her Lasix (furosemide) for the past 4 days. You note that she is speaking in 5- to 6-word sentences, and she does say that she was quite winded from walking in from the parking lot. She eventually reveals that her shortness of breath has been worsening and that her legs are more swollen than usual. Her BP is 179/148 mm Hg.
- **Case 3—worn out:** A 52-year-old man reports being light-headed after working in his yard all day on a warm, sunny day. The patient says he has no history of illness but reports that he has not seen a physician for years. He says his wife made him come in because he did not look well. He says that he is "really worn out" from his labors today and that he probably "overdid it" and should have drunk more water. He recalls urinating when he woke up this morning but thinks that he may have urinated only one other time today. He is currently thirsty. His BP is 211/151 mm Hg.

These scenarios serve here as references to elucidate appropriate treatment of patients with severely elevated BP in the urgent care setting.

Red Flag Signs and Symptoms

Whenever a patient's BP is remarkably elevated above baseline (again, there is no official cutoff, but the one common in clinical settings is >180/120 mm Hg), then our focus is to search for any signs or symptoms of end-organ damage. If either parameter of their BP is >180/120 mm Hg and the patient is completely asymptomatic, then we call this *severe asymptomatic HTN*. These patients can undergo a work-up, be given a diagnosis, and treated as outpatients in accordance with the Eighth Joint National Committee (JNC 8) guideline released in 2014 on treating high BP in adults.[4] However, the focus here is on what constitutes significant end-organ damage that would necessitate transferring the patient from an urgent care center to an ED for admission and further management of their hypertensive emergency.

Hypertensive Brain Damage

Most argue that a moderate to severe headache alone with markedly elevated BP without any other neurologic signs or symptoms is *insufficient* to meet criteria for a hypertensive emergency. Moreover, the same can be said for epistaxis or dizziness. However, if the patient is exhibiting *any neurologic dysfunction* (i.e., difficulty with vision, hearing, balance, coordination, speech, agitation, delirium, altered mental status, or focal neurologic findings) related to their surges in elevated BP, then a hypertensive emergency should be suspected. These symptoms are caused by cerebral edema from an enor-

mous amount of pressure in the capillary beds, causing swelling of the brain that can even lead to seizures and coma. The patient in case 1 ("painkiller for a headache") initially did not exhibit any of the red flags, but his history of difficulty with speech and decreased attentiveness during a recent phone conversation *should absolutely not be dismissed* as caused by tiredness. Patients may minimize their own symptoms, but if urgent care providers also give in to this minimization, it could be disastrous for both patients and providers.

Hypertensive Heart Damage

Acutely elevated systemic BP places an increased strain on the heart to pump and overcome the patient's increased systemic vascular resistance. This increased workload on the heart can cause or exacerbate heart failure and may lead to cardiac ischemia. Symptoms can include chest pain, chest tightness, shortness of breath, dyspnea on exertion, increased peripheral edema, tachycardia, respiratory distress, and orthopnea. Acute pulmonary edema ensues when the heart can no longer compensate and overcome the increased systemic vascular resistance. Our patient in case 2 ("just a prescription refill") might have reported a history of "only mild heart failure," but given her increased shortness of breath, she will likely need much more than a refill of her Lasix. Often this agenda—to get a prescription and continue on one's merry way—might work if it was only 1 day of excess fluid. However, after 4 days of being overloaded with fluid, her heart is now being excessively strained and her pulmonary edema is manifested in her shortened sentences. Additional history further revealed that she had to rest twice, approximately every 30 to 40 feet, on her way in from the parking lot. She also reported using an extra two pillows to help her sleep the previous night.

Hypertensive Kidney Damage

The kidneys become stressed when overpressurized fluid is bursting through the glomeruli. Acute elevations of BP wreak havoc on the glomeruli and nephrons, leading to glomerular ischemia, tubular necrosis, and microscopic hematuria. Activation of the renin-angiotensin pathway only exacerbates the problem. Oftentimes the patient might have recently taken extra diuretics or might have decreased their fluid intake, and this further accelerates the effects of the renin-angiotensin activation, leading to acute kidney injury (AKI). AKI is defined as an increase in serum creatinine by >0.3 mg/dL, or a 50% increase in serum creatinine, or urine output of <0.5mL/kg per hour over 6 hours.[5] The patient often reports a vague, poorly defined illness that is likely caused by the patient's elevated uremia, possible electrolyte disturbances, and hypovolemia. The patient may report decreased fluid intake and may feel thirsty. They may have not properly hydrated in the setting of increased fluid loss from sweating after heavy labor or exercise. These combined effects usually result in decreased urinary output. The patient in case 3 ("worn out") had the majority of the aforementioned symptoms: vague illness, poor fluid intake, increased fluid loss from hard labor on a warm day, and decreased urinary output. He likely has had HTN for years but does not see a physician regularly enough for his condition to be diagnosed. He now unknowingly relies greatly on his urgent care provider to protect his kidneys from further thrashing and complete renal failure.

Work-Up and Treatment

In all patients who present any of the symptoms or red flags, it is important to use a monitor for frequent BP checks. Proper management of hypertensive emergencies relies heavily on accurate BP measurements. This often means that if the BP was initially assessed by an automated machine, then it should be verified manually for accuracy and then rechecked frequently on the monitor. The patient's symptoms can deteriorate if the BP continues to rise. A decrease in BP should also correlate with a decrease in symptoms.

Physical Examination Pearls

An organ-system approach to examination is essential. For example, in patients with symptoms indicating possible brain ischemia, a complete neurologic examination should be performed, including a cranial nerve examination, cerebellar examination, and a funduscopic examination. How often do we actually spend several minutes attempting funduscopy? Well, this is exactly the time to do just that. Papilledema and hypertensive retinopathy are well described in hypertensive states. Although the examination findings are sometimes difficult to fully appreciate without dilation, the clinician may find cotton wool spots and flame hemorrhages in addition to papilledema.

The patient in case 1 ("painkiller for a headache") initially had completely normal findings on neurologic examination. Approximately 30 minutes into the examination, his daughter came out of the examination room and reported that "he is doing it again." The patient had become agitated, his speech was slowed, and his

responses were slow but angry. He had no focal motor deficits. His BP was immediately retaken, and it had spiked to 269/146 mm Hg.

Laboratory Tests and Imaging

In patients with a hypertensive emergency, the following tests and imaging are commonly ordered on the basis of the target organ that is facing damage:

- Electrocardiography, looking for signs of cardiac ischemia
- Chest radiography, focusing on evidence of pulmonary edema and cardiomegaly. Although electrocardiographs (ECGs) and chest radiographs are commonly ordered, both have a very poor sensitivity (failing to identify problems in 75% of patients) for finding left ventricular dysfunction in hypertensive heart disease.[6]
- Complete blood count (CBC), with differential, because occasionally in HTN syndromes, a hemolytic uremic syndrome may develop, and some argue that a blood smear is justified to rule out a microangiopathic hemolytic anemia
- Serum electrolytes, with a focus on blood urea nitrogen (BUN) and creatinine levels, compared with previous findings
- Cardiac enzymes, especially when symptoms may suggest cardiac ischemia
- Pro–brain natriuretic peptide, if patients have a history and/or symptoms of congestive heart failure
- Urinalysis, looking for microscopic hematuria and elevated protein levels
- Urine pregnancy test in females of childbearing age, to exclude preeclampsia
- Head computed tomography (CT), in patients with symptoms of neurologic dysfunction. In the majority of patients with hypertensive encephalopathy and cerebral ischemia, findings on head CT should be normal. Head CT is much better than magnetic resonance imaging at identifying cerebral hemorrhage, head trauma, or other mass effects.
- Chest CT with intravenous (IV) contrast, in search of possible aortic dissection
- Renal ultrasound, usually completed during admission, to rule out renal artery stenosis

Correcting the Hypertension

In most symptomatic HTN syndromes, the goal within the first 1 to 2 hours is to reduce the mean arterial pressure by 20% to 25%. It is important not to overshoot when correcting a patient's BP, because this can exacerbate end-organ damage.[7] Patients with long-standing HTN adapt to their hypertensive state via an autoregulatory process. This process shifts the cerebral blood flow according to the degree of their baseline hypertensive state. This means that if the BP is aggressively overcorrected (i.e., the patient's BP is decreased to within a normal range of <140/90 mm Hg), then the patient's cerebral blood flow can potentially drop off a cliff to a state that is equivalent to a normotensive patient exhibiting hypotension. Emphasis should be placed on reducing the BP to a point at which the patient's symptoms resolve. Depending on the degree of elevation, if a reduction of 20% to 25% does not reverse symptoms of end-organ damage, then a reduction of 30% to 40% may be needed. The patient in case 1 was asymptomatic on initial presentation but became symptomatic when his BP spiked to 269/146 mm Hg (MAP, 187 mm Hg) from 217/103 mm Hg (MAP, 141 mm Hg). The goal in his case should be to return his BP to an asymptomatic level, knowing that at 217/103 mm Hg, the patient has no symptoms.

As a patient's baseline mean arterial BP increases, the baseline pathophysiology can change so that potentially the cerebral blood flow could drastically drop but still be within the normotensive range. This should be kept in mind when correcting a patient's BP, and a gentle correction should be emphasized. Because of the real possibility of ischemia from overshooting while controlling the patient's BP, it is generally recommended that the patient's BP not be decreased by more that 25% to 30% in the first 24 hours of treatment.

Diagnoses That Necessitate Aggressive Blood Pressure Control

There are a *few exceptions* to the general rule to gently decrease the patient's BP. More-aggressive BP management is required for the following:

- **Aortic dissection:** It is recommended to achieve heart rate control first with β-blockers (ideally to a heart rate of <60 bpm). The heart rate is decreased first in order to decrease the shear forces on the aortic wall. The β-blockade is followed by an α-mediated blockade (usually with a calcium-channel blocker) to a goal systolic BP (SBP) of 100 to 120 mm Hg.
- **Intracranial hemorrhage:** SBP goals have recently changed for this condition. The goal used to be a SBP of <180 mm Hg, but more recent evidence suggests a SBP of <140 mm Hg is superior. In reference to intracerebral hemorrhage, the main body of evidence comes from two recent trials, the Intensive

BP Reduction in Acute Cerebral Haemorrhage Trial (INTERACT 1 released in 2010, and INTERACT 2 released in 2015) and the Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATACH) study.[8–10] Essentially the evidence suggests that in intracerebral hemorrhage, the BP should be aggressively managed, being decreased to a SBP of <140 mm Hg within 6 hours, instead of <180 mm Hg under the old guidelines. More aggressive BP management has been shown to be correlated with decreased expansion of intracerebral bleeding and improved 30- and 90-day outcomes.

- **Thrombolysis for brain ischemia:** The goal BP for thrombolysis is <185/110 mm Hg. If the patient does not meet criteria for thrombolysis but cardiac ischemia is ongoing, the goal BP is a systolic of <220 mm Hg and a diastolic of <120 mm Hg.

Medications for Hypertensive Emergencies

No single antihypertensive has been proven to be superior in efficacy or lowest in morbidity and mortality. According to Studying of Treatment of Acute hyperTension (STAT), the most commonly used bolus medication in the ED is labetalol and the most common infusion is nitroglycerin.[11] Both nicardipine and labetalol are generally good choices in the setting of brain end-organ damage. Nitroglycerin is commonly used for acute heart failure because it is very effective in decreasing preload and reducing effects of acute pulmonary edema. Continuous positive airway pressure (CPAP) has also been proven to be effective in treating acute pulmonary edema from congestive heart failure. Fenoldopam is often used when AKI is suspected, given that it improves corticomedullary perfusion of the kidneys and it is not renally cleared.

In cases of eclampsia or preeclampsia, the threshold for treatment is lower (treat when the SBP is >160 mm Hg), given that adverse outcomes occur at relatively lower BPs. Delivery of the infant is the ultimate treatment. Magnesium sulfate is the first-line treatment, and adjunctive treatments includes hydralazine, labetalol, and/or nicardipine. Benzodiazepines are often administered to patients with cocaine or other stimulant-induced HTN.

Disposition and Case Scenario Outcomes

All patients with evidence of end-organ damage should be transferred to an ED for management of their hypertensive crisis. They are often admitted to an intensive care unit (ICU) for frequent monitoring.

The patient in case 1 ("painkiller for a headache") was treated for hypertensive encephalopathy. He met criteria for the diagnosis because his symptoms (altered mental status, disorientation, agitation, slowed speech, headache) occurred and resolved with spikes in BP. He was given a 10-mg IV bolus of labetalol; his symptoms decreased, and his BP dropped from 269/146 to 213/116 mm Hg. His findings on head CT, ECG, chest radiographs, CBC, basic metabolic profile, and urinalysis were all unremarkable. The clinician did find evidence of slight retinal hemorrhages on limited funduscopy. The patient was admitted to an ICU, and his BP was gently brought down over several days. Magnetic resonance images showed evidence of small-vessel ischemia changes but otherwise, the findings were nonacute. His daughter later discovered that he had not been taking his antihypertensives as frequently as he stated.

In case 2 ("just a prescription refill"), the urgent care provider did not succumb to the patient's agenda. Instead, he astutely did a work-up for acute heart failure and pulmonary edema. An IV line was started, but no fluids were given. The patient was given 0.4 mg of sublingual nitroglycerin and oxygen. Her ECG showed no acute findings, but her chest radiographs showed evidence of bilateral increased fluffy interstitial markings, consistent with pulmonary edema. She was transferred to the local ED and admitted to the ICU, where she received CPAP and a nitroglycerin drip. She was given 80 g of Lasix intravenously for diuresis. After 12 L of diuresis over several days, she was eventually able to return home with her prescription for Lasix.

In case 3 ("worn out"), the patient's serum creatinine level was 2.1 mg/dL (0.8 mg/dL at baseline 7 years earlier). His blood urea nitrogen level was also markedly elevated at 45 mg/dL (baseline level, 18 mg/dL). He was unable to provide a urine sample even after he was given 2 L of fluids intravenously. His urinary output was collected via Foley catheter, and it showed 21 to 40 red blood cells/HPF and tubular casts. He was given a small IV dose of nicardipine in the ED and later a dose of fenoldopam. He produced only 0.3 mL/kg per hour of urine in the first 24 hours. His feelings of general malaise decreased with IV hydration and a decrease in BP. His renal ultrasound findings were negative for renal artery stenosis. His creatinine clearance peaked at 3.2 mL/min. He slowly regained kidney function over the next several days and did well. He now sees his primary-care provider frequently.

Conclusion

Managing elevated BP in the ED and urgent care settings is a common occurrence; it is required in 40% of U.S.

patients each year. If the patient has no signs or symptoms of end-organ damage, then is the patient is deemed to have asymptomatic HTN and can be discharged home. The patient should be advised to seek treatment by their primary-care provider and should be given appropriate antihypertensive medication in accordance with JNC 8 standards.

Patients with evidence of end-organ damage require an approach based on the organ system that is affected, most commonly the brain, heart, and kidneys. The urgent care provider must focus on decreasing the patient's BP, recognizing that overshooting can actually lead to decreased organ perfusion because of autoregulatory effects. With some exceptions (aortic dissection, intracranial hemorrhage, plan for thrombolysis in acute stroke), the BP should *not* be aggressively treated. Rather, a gentle decrease of 10% to 20% in the first hour and 25% to 30% in the first 24 hours is an appropriate goal. Nicardipine or labetalol is commonly used for HTN-related brain damage. Nitroglycerin is often given for hypertensive emergencies affecting the heart, including heart failure and pulmonary edema. Fenoldopam is beneficial for hypertensive emergencies involving the kidneys. Most patients with hypertensive emergencies are admitted to an ICU for further treatment. ■

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