A 62-Year-Old Man With Worsening Hypertension Symptoms

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In this column, we will continue discussing the 62-year-old man from last month's column.[1] The patient had been adherent to the prescribed treatment regimen and was doing well following his episode of hypertensive urgency. Thereafter, however, he markedly loosened his connection with his internist and was essentially lost to follow-up. He had resumed his rather cavalier lifestyle, including consuming generous amounts of salt and alcohol.

After 3 months of this lifestyle, he resurfaced, this time at the emergency department (ED). He had been experiencing increasing shortness of breath for about a week, and in the last 24 hours, he had an intermittent nosebleed.

At presentation in the ED, he denied chest pains or neurologic symptoms. A series of blood pressure (BP) readings averaged 225/140 mmHg. His heart rate was 100 bpm and respiratory rate was 18 breaths/min; his breathing was somewhat labored. An examination of his head, eyes, ears, nose, and throat was significant for retinal hemorrhage on fundoscopy and distended neck veins. Chest radiography revealed cardiomegaly and alveolar pulmonary edema, and he had 2+ bilateral ankle and pedal edema.

Baseline blood test results showed a normal complete blood cell count, an elevated troponin level of 0.03 ng/mL, and an elevated creatinine level of 2.5 mg/dL, which were significant changes from his visit 6 months prior.

Which of the following is the correct statement regarding the management of the patient at this time?

A. The protocol of management—setting and specific antihypertensives—he received 6 months ago should be repeated.
B. He should be immediately treated in the ED with intravenous (IV) hydralazine to rapidly lower his BP.
C. He should be treated in an intensive care unit (ICU) setting with IV antihypertensive medications with a goal of BP reduction to less than 120/80 mmHg.
D. He should be admitted to and treated in an ICU setting with IV antihypertensive medications to lower his BP by 25% with use of oral medications after 6 to 12 hours.

Correct Answer: D

He should be admitted to and treated in an ICU setting with IV antihypertensive medications to lower his BP by 25% with use of oral medications after 6 to 12 hours.

Unfortunately, the patient we have been following has "graduated," so to speak, and matriculated into the pinnacle of acute severe hypertension crises: a hypertensive emergency. As previously discussed,[1] this is defined as severe acute hypertension numerically (>180/110-120 mmHg). In addition, there is the presence of acute target organ damage. This can include brain (ie, stroke, intracerebral hemorrhage), retina (ie, hemorrhages, papilledema), or aortic dissection or renal involvement (ie, acute kidney injury).[2] It is somewhat remarkable how this drastic and life-threatening situation can so easily be evaluated—even today with our often so-detailed technical capabilities. Old fashioned symptoms and signs, classical history, and physical examination often are demonstrative and adequate. Has there been a new neurological event? Is there new chest pain or congestive heart failure (CHF)? On physical evaluation, are there focal neurological findings? Does fundoscopy show hemorrhage and/or papilledema? Is there pulmonary edema on auscultation?

Routine intake studies such as chest radiography, troponin level, and metabolic levels will confirm CHF, acute coronary insufficiency, and acute renal injury. Central nervous system (CNS) imaging eventually needs to be conducted, as certain treatment intensity specifics (eg, extent and speed of BP lowering) will depend on the presence of stroke, hemorrhage, or nothing. Regardless of the specifics, any and all of the above buys the patient a ticket to the ICU.

So, unlike "hypertensive urgency" where management usually will not require hospital admission, when clinical criteria defining "hypertensive emergen-

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CITATION:
Rubin RN. A 62-year-old man with worsening hypertension symptoms. *Consultant*. 2021;61(3):e14-e15. doi:10.25270/con.2021.03.00002

DISCLOSURES:
The author reports no relevant financial relationships.

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cy" are present, immediate admission to ICU care is indicated. This is because treatment is needed literally within minutes, IV medications are indicated, and real-time titration of BP to appropriately, yet not excessively, lower the BP to safe levels is needed. These facts immediately make Answer A an incorrect one. The patient's syndrome is not what he manifested 6 months prior, and his indicated management will not be either.

One of the key factors in treatment of hypertensive emergency is determining the patient's target BP level and how quickly we can safely achieve that goal in a given patient. This is related to the concept of autoregulation of blood flow in patients with chronic high-grade hypertension, which refers to their ability to tolerate a higher BP level than individuals with normal BP and, in fact, being at risk for hypotension/ischemia (particularly in the CNS) if BP is lowered too quickly or aggressively.[3]

Current guidelines recommend using IV antihypertensive agents with a goal of 20% to 25% BP reduction in the first hour and then 160/100-110 mmHg for the next 2 to 6 hours.[4] More-aggressive lowering actually increases the risk of death.5 Thus Answer C is far too aggressive—and actually is dangerous—and not correct here.

There exist a host of agents available that can be used with good data supporting their efficacy.

They share the characteristics of IV administration, very rapid onset of action (minutes), and ability to quickly and nimbly titrate dosage as required. Examples include nitroprusside, labetalol, nicardipine, and nitroglycerin. A notable exception is IV hydralazine, which is capricious and unpredictable in effect, with a greater risk for hypertension.[4] This makes Answer B an incorrect choice here. IV hydralazine would not be one of the first choice to treat hypertensive emergency.

There are nuances to therapy specifics that vary a bit between target organs, which are beyond the scope of this article and best left to the ICU. But very generally, they include being a bit more aggressive in cardiac situations, such as decreasing systolic BP to 140 mmHg rather than 160 mmHg in patients with acute heart failure and acute coronary syndromes and decreasing systolic BP even lower in patients with aortic dissection. Conversely, we would be less aggressive with patients with an intracerebral hemorrhage or ischemic stroke to avoid the risk of worsening cerebral hypoperfusion.[2]

After the initial 6 hours, longer-acting agents can replace the rapid agents with a goal of achieving normal BP with 2 or 3 days. This overall strategy is essentially stated verbatim in Answer D, which is the correct answer here.

TAKE-HOME MESSAGE

At the apex of clinical manifestations of acute severe hypertension is hypertensive crisis. This is defined by a BP reading of more than 180/110-120 mmHg accompanied by findings of acute target organ damage, including any one or more of the following: brain (stroke or intracerebral hemorrhage); microvasculature (retinal hemorrhages, microangiopathic hemolytic anemia, thrombocytopenia); aortic dissection; or acute kidney or renal injury. Any such patient requires immediate admission to an ICU setting where a variety of rapid-acting IV medications—such as nitroglycerin, labetalol, furosemide, and nicardipine—can be titrated in real time to lower BP to acceptable "safe" levels within an hour or two. Generally speaking, neurological hypertensive emergency goals are within the 160/100-110 mmHg range at one hour, while the goal during a cardiac emergency is lower than 140/100-110 mmHg at one hour. Care needs to be taken so as to not be overly aggressive with BP lowering, as this can result in tissue ischemia and even increased risk of mortality. Even with the current outstanding array of ambulatory medications that are available, the number of admissions and the in-hospital mortality from hypertensive emergencies remain disturbingly high.

PATIENT FOLLOW-UP

The patient was immediately admitted to the ICU. As there were no neurological symptoms or signs, CNS imaging was deferred. The treatment approach was for a hypertensive emergency involving acute decompensated CHF with the possibility of an acute coronary syndrome as well. The patient was given nitroglycerin and IV furosemide. Within 1 hour, his BP lowered to 140/85 mmHg, and after 6 hours, his BP was 125/75 mmHg.

His shortness of breath markedly improved in concert with the BP lowering. After 8 hours, the patient was gradually and successfully weaned onto an oral regimen of furosemide, an angiotensin-converting-enzyme inhibitor. He remained in the hospital for 6 days, during which clinical findings and BP readings were proven to be stable. Prior to discharge, laboratory studies showed that his creatinine level remained elevated in the 2.5 to 3.0 mg/dL range and his left ventricular ejection fraction, which was normal 6 months ago, had now decreased to 40%. Results of a thallium stress test were negative for coronary artery disease.

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