Hypertensive crisis—pathophysiology, initial evaluation, and management

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BACKGROUND

The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure recently classified hypertension according to the degree of blood pressure elevation. Stage 1 patients have systolic blood pressure (SBP) of 140–159 mmHg or diastolic blood pressure (DBP) of 90–99 mmHg. Stage 2 patients have SBP of 160–179 mmHg or DBP of 100–109 mmHg, whereas stage 3 corresponds to SBP of 180 mmHg or greater or DBP of 110 mmHg or greater. Stage 3 hypertension has also been called severe hypertension or accelerated hypertension.[1] Although chronic hypertension is an established risk factor for cardiovascular, cerebrovascular, and renal disease, acute elevations in BP can result in acute end-organ damage with significant morbidity. Prompt recognition, evaluation, and appropriate treatment of these conditions are crucial to prevent permanent end-organ damage.

Hypertensive crisis refers to a syndrome characterized by severe acute elevations in blood pressure associated with imminent risks to the patient.[2,3] Although not specifically addressed in the JNC-7 report, patients with a SBP >179 mmHg or a DBP >109 mmHg are usually considered to be having a "hypertensive crisis." The absolute level of BP itself is less important, as acute increases in BP of even modest degree can lead to critical target organ damage in previously normotensive patients and those with certain medical conditions like acute myocardial infarction or aortic dissestion.[4]

Prior to the introduction of antihypertensive medications, approximately 7% of hypertensive patients had a hypertensive emergency.[5] Currently, it is estimated that ~1% of patients with hypertension will, at some point, develop a hypertensive crisis.[6,7] It is a common medical problem and accounts for 27.5% of all medical emergencies presenting to the emergency department.[8] The epidemiology of hypertensive crises is similar to that of hypertension (i.e. higher among African-Americans and the elderly); however, men are affected approximately two times more frequently than women.[9–12]

Hypertensive urgencies are hypertensive crises associated with severe elevations in BP without progressive target-organ dysfunction.[11,13–19] The majority of these patients present as noncompliant or inadequately treated hypertensive individuals, often with little or no evidence of target-organ damage. Hypertensive emergencies are hypertension crises characterized by severe elevations in BP >180/120 mmHg complicated by evidence of impending or progressive target-organ dysfunction. They require immediate BP reduction (not necessarily to normal levels) to prevent or limit target-organ damage.[20,21]

PATHOPHYSIOLOGY

Acute severe hypertension can develop *de novo* or can complicate underlying essential or secondary hypertension. The exact mechanism of hypertensive crisis is not known but the rapidity of onset suggests a triggering factor superimposed on pre-existing hypertension.[22] Most accepted hypothesis is shown in Figure 1. Under normal circumstances, the renin-angiotensin-aldosterone system plays a central role in the regulation of normal BP homeostasis.[23] Overproduction of renin by the kidney stimulates the formation of angiotensin II, a potent vasoconstrictor. Consequently, both peripheral vascular resistance and BP increase. Hypertensive crisis is thought to be initiated by an abrupt increase in systemic vascular resistance likely related to humoral vasoconstrictors.[24,25] In the hypertensive crises state, amplification of renin system activity occurs, leading to vascular injury, tissue ischemia, and further overproduction of renin-angiotensin. This vicious cycle contributes to the pathogenesis of hypertensive crises. The pathophysiologic link between the

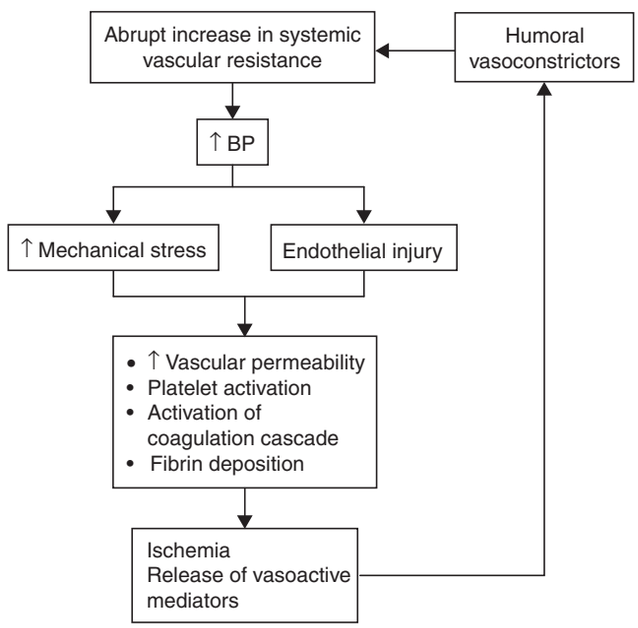

Figure 1 Pathophysiology of hypertensive crisis.

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Table 1 Initial evaluation and management of hypertensive crisis.

A) Hypertensive urgency
- BP usually > 180/110 mmHg
- Presents with headache, dyspnea, edema
- Observe for 4–6 hours, oral agents for BP control and follow-up evaluation within 24 hours

B) Hypertensive emergency
- BP usually > 220/140 mmHg
- Presents with dyspnea, chest pain, dysarthria, focal neurological signs, altered sensorium, pulmonary edema, encephalopathy, stroke, renal failure
- Admit to ICU, investigate for associated condition, and start parenteral antihypertensive therapy

renin system and hypertensive crises has been established by demonstrating that this process can be arrested when the renin-angiotensin system is interrupted either pharmacologically (i.e., angiotensin converting enzyme [ACE] inhibitor, beta-blocker, or type 1 angiotensin receptor antagonist) or by removal of an ischemic kidney.[23,26,27] Other factors induced by excess renin-angiotensin include pro-inflammatory cytokines and vascular cell adhesion molecules, which may contribute to the vascular sequelae and target organ damage.[28–30]

EVALUATION AND INITIAL MANAGEMENT

Patients with hypertensive emergency usually present for evaluation as a result of a new symptom complex related to their elevated BP. Patient triage and physician evaluation should proceed expeditiously to prevent ongoing end organ damage. A focused medical history that includes the use of any prescribed or over-the-counter medications should be obtained. If the patient is known to have hypertension, their hypertensive history, previous control, current antihypertensive medications with dosing, compliance, and time from last dose are important facts to know as subsequent treatment decisions are made. Also use of recreational drugs (amphetamines, cocaine, and phencyclidine) should be considered. Confirmation of the blood pressure should be obtained in both arms by a physician using a BP cuff of appropriate size. Patients with long-standing hypertension may tolerate SBP > 200 mmHg or DBP > 150 mmHg without developing clinical signs or symptoms of end organ damage; however, in postoperative or pregnancy patients a much lower but more rapidly progressive increase in blood pressure may result in end organ damage. The physical examination should attempt to identify evidence of end organ damage by assessing pulses in all extremities, auscultating the lungs for evidence of pulmonary edema, the heart for murmurs or gallops, the renal arteries for bruits, and performing a focused neurologic and fundoscopic examination. Headache and altered levels of consciousness are the usual manifestations of hypertensive encephalopathy.[31,32] Focal neurological findings, especially lateralizing signs, are uncommon in hypertensive encephalopathy, being more suggestive of a cerebrovascular accident. Subarachnoid hemorrhage should be considered in patients with a sudden onset of a severe headache.

Initial objective evaluation should include a metabolic panel to assess electrolytes, creatinine, and blood urea nitrogen; a complete blood count (and smear if microangiopathic hemolytic anemia is suspected); a urinalysis to look for proteinuria or microscopic hematuria; and an electrocardiogram to assess for cardiac ischemia.[33] Supportive radiographic studies such as a chest radiograph in a patient with dyspnea or chest pain or a head computed tomography scan in a patient with neurologic symptoms should be obtained in the appropriate clinical scenario. In patients presenting with pulmonary edema it is important to obtain an echocardiogram to distinguish between diastolic dysfunction, transient systolic dysfunction, or mitral regurgitation.[34]

Hypertensive crises share all the pathophysiologic mechanisms and target organ complications (MI, stroke, renal failure) similar to patients with milder forms of high BP and, thus, together can be viewed as part of the spectrum of hypertensive diseases. BP is reduced, in the hypertensive crises and in the chronic forms of hypertension, by the same drugs that interrupt relevant pathophysiologic mechanisms. An algorithm for initial management of hypertensive crisis is given in Table 1.

The majority of patients in whom severe hypertension (SBP > 160 mmHg, DBP > 100 mmHg) is identified on initial evaluation will not have evidence of end organ damage and thus have hypertensive urgency. As no acute end organ damage is present, these patients may present for evaluation of another complaint, and the elevated blood pressure may represent an acute recognition of chronic hypertension. In these patients, utilizing oral medications to lower the blood pressure gradually over 24–48 hours is the best approach to management.[35–37] In fact, rapid reduction of blood pressure may be associated with significant morbidity in hypertensive urgency because of a rightward shift in the pressure/flow auto-regulatory curve in critical arterial beds (cerebral, coronary, and renal).[38] Rapid correction of severely elevated blood pressure below the autoregulatory range of these vascular beds can result in a marked reduction in perfusion causing ischemia and infarction.[39–42] Therefore, although the blood pressure must be reduced in these patients, it must be lowered in a slow and controlled fashion to prevent this impaired autoregulatory hypoperfusion problem.

Patients with a hypertensive emergency should be managed in an intensive care unit with close monitoring and are best managed with a continuous infusion of a short-acting, titratable antihypertensive agent. Because of unpredictable pharmacodynamics,

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