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Hypertension Management: The Critical Role of Nurses

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ABSTRACT

Hypertensive crises, encompassing hypertensive emergencies and urgencies, are acute elevations in blood pressure that require prompt intervention to prevent target organ damage. Hypertensive emergencies, characterized by a systolic blood pressure exceeding 180 mmHg or a diastolic blood pressure above 120 mmHg with evidence of acute target organ damage, are associated with significant mortality risk if left

untreated. Most patients experiencing hypertensive crises have a history of uncontrolled essential hypertension, with medication nonadherence and sympathomimetic agents being common contributors. Pathophysiological mechanisms involve overactivation of the renin-angiotensin-aldosterone system, pressure-induced natriuresis, and increased systemic vascular resistance. Assessment includes a focused history, physical examination, and diagnostic testing to identify the underlying cause and target organ dysfunction. Treatment goals prioritize identifying the etiology, mitigating organ damage through controlled blood pressure reduction, and monitoring for adverse effects. First-line pharmacologic therapy involves intravenous medications tailored to the specific target organ damage and patient comorbidities. Nurses play a critical role in monitoring, ensuring patient comfort, and providing comprehensive discharge education. Discharge planning should address social determinants of health, medication adherence strategies, and the importance of lifelong hypertension management through nonpharmacologic and pharmacologic interventions.

Keywords: Hypertension, Hypertensive crisis, Nursing, Hypertensive emergencies, Hypertensive urgency

Introduction

Approximately half of all adults in the United States diagnosed with hypertension (HTN) fail to achieve their blood pressure (BP) treatment goals. One potential complication of poorly controlled HTN is an acute and significant elevation in BP, a relatively common condition among adults presenting to the emergency department or hospitalized. The term "hypertensive crisis" encompasses the spectrum of acute, severe HTN conditions, characterized by a systolic BP exceeding 180 mm Hg or a diastolic BP exceeding 120 mm Hg (Mathews et al., 2021). Hypertensive crises are further classified into two categories: hypertensive emergency and hypertensive urgency. A hypertensive emergency is defined by a sudden increase in BP above 180/120 mm Hg accompanied by new or worsening target organ damage, such as neurologic, cardiovascular, or renal complications (Whelton et al., 2018). Conversely, hypertensive urgency involves the same BP thresholds but without evidence of acute target organ damage. The prevalence of suspected hypertensive emergencies among emergency department presentations is approximately 0.5%, or 1 in 200 individuals, a statistic that remains consistent globally (van den Born et al., 2019). Within this broader category, hypertensive urgencies are more prevalent than hypertensive emergencies, occurring in approximately 1 in 100 adults compared to 1 in 300 adults, respectively (Astarita et al., 2020).

Hypertensive emergencies are associated with significant mortality risk if left untreated. For instance, patients experiencing a hypertensive emergency have a median survival of only 10.4 months without treatment. The urgency of treatment is dictated more by the rapid escalation in BP than by the absolute BP value, as patients with chronic, uncontrolled HTN often tolerate higher BP levels compared to normotensive individuals. In contrast, hypertensive urgency does not necessitate immediate BP reduction, emergency department referral, or hospitalization. These cases are generally managed in an outpatient setting, where treatment typically

involves resuming previously discontinued BP medications or intensifying the regimen with regular follow-up care (Whelton et al., 2018).

This paper aims to address the diagnostic approach for patients suspected of having a hypertensive emergency, including initial evaluation through history, physical examination, and diagnostic testing to identify the underlying cause of the acute BP elevation. It also outlines first-line treatments based on the suspected etiology, priorities for nursing care in acute settings, and considerations for patient and family education.

Pathophysiology

Hypertensive crises, encompassing both hypertensive urgency and emergency, represent a diverse group of conditions with variable causes and clinical presentations. Most patients experiencing hypertensive crises have a history of uncontrolled essential (primary) HTN. Studies indicate that 82.5% of patients with hypertensive emergencies and 78% of those with hypertensive urgencies have underlying HTN (Astarita et al., 2020). The primary contributors to these crises are often nonadherence to prescribed treatment regimens or the use of sympathomimetic agents. Notably, some individuals with preexisting HTN remain unaware of their condition due to the asymptomatic nature of elevated BP. A smaller proportion of hypertensive crises, approximately 20%–40%, are attributed to secondary causes of HTN, most commonly glomerulonephritis or renal artery stenosis. The following paragraph highlights potential causes of hypertensive crises.

Potential Causes of a Hypertensive Crisis

Hypertensive crises can arise due to a variety of acute factors that result in a sudden and significant elevation in blood pressure. Among the most common causes are nonadherence to prescribed antihypertensive medications, particularly if there is an abrupt withdrawal of agents such as clonidine or beta-blockers. Additionally, dietary indiscretions, including excessive sodium intake, can exacerbate the condition. Certain medications, such as corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), cyclosporine, sympathomimetics, and drugs used in antiangiogenic therapy, are also known contributors (Williams et al., 2018).

Drug-drug interactions represent another potential cause. For instance, combining monoamine oxidase inhibitors with tricyclic antidepressants, antihistamines, or tyramine-containing foods can provoke hypertensive episodes. Similarly, the use of interfering substances like cocaine, amphetamines, phencyclidine (PCP), or stimulant diet supplements is associated with acute blood pressure surges.

Secondary causes of uncontrolled blood pressure can stem from underlying medical conditions affecting various systems. Cardiovascular anomalies, such as coarctation of the aorta, and renal conditions, including chronic kidney disease and renal artery stenosis, are notable contributors. Endocrine disorders, such as pheochromocytoma, Cushing syndrome, hyperthyroidism, and primary hyperaldosteronism, can also drive hypertensive crises.

Pregnancy-related complications, including preeclampsia or eclampsia, are significant causes in pregnant individuals. Neurologic conditions, such as head or

spinal cord injuries, ischemic or hemorrhagic stroke, and brain tumors, are additional factors that can precipitate a crisis. Finally, autoimmune conditions like systemic lupus erythematosus and other collagen vascular disorders may also play a role in triggering a hypertensive emergency.

This comprehensive understanding of potential causes highlights the multifactorial nature of hypertensive crises and underscores the importance of identifying and addressing the underlying etiology for effective management.

Although the precise pathophysiological mechanisms underlying hypertensive crises are not fully understood, several interrelated processes are believed to contribute. These include overactivation of the renin-angiotensin-aldosterone system, pressure-induced natriuresis (a maladaptive diuretic response resulting in increased sodium excretion by the kidneys), and a sudden rise in systemic vascular resistance (van den Born et al., 2019). The resulting microvascular damage perpetuates a cycle that disrupts normal autoregulatory functions, leading to target organ damage from the abrupt BP elevation. As noted, the presence of target organ damage is the distinguishing feature between a hypertensive emergency and hypertensive urgency, with the latter showing no evidence of such damage (van den Born et al., 2019).

Assessment

History Taking

History taking for individuals presenting with a hypertensive crisis should prioritize identifying symptoms and potential causes of the acute BP elevation. Symptom presentation varies widely, with some patients remaining asymptomatic and others exhibiting life-threatening manifestations associated with target organ damage. According to a 2020 meta-analysis encompassing eight studies with a combined sample of 1,970 hypertensive emergencies and 4,983 hypertensive urgencies, the most frequent symptoms in hypertensive emergencies were neurological symptoms (35%) and dyspnea (31%), followed by nonspecific symptoms (24%) (5). In contrast, hypertensive urgency cases predominantly presented with nonspecific symptoms (48%) and headache (22%) (5). Additional symptoms of a hypertensive crisis may include chest pain, palpitations, visual disturbances, dizziness, gastrointestinal complaints (e.g., abdominal pain, nausea, vomiting, anorexia), and epistaxis (Saladini et al., 2020).

Beyond symptom assessment, the patient's history should include whether they have a diagnosis of HTN, their treatment regimen, and their adherence to prescribed therapy. Clinicians should also inquire about potential factors contributing to the acute BP increase, particularly in those with preexisting uncontrolled HTN.

Physical Examination

The physical examination begins with obtaining baseline vital signs, including temperature, respiratory rate, heart rate, and BP. Blood pressure measurements should be conducted using an appropriately sized automated BP cuff to

avoid erroneous readings. Undersized cuffs may yield falsely elevated readings, while oversized cuffs may underestimate BP. Initial BP assessments should include readings from both arms. Typically, inter-arm BP differences are less than 10%, and more substantial discrepancies may indicate conditions such as aortic dissection, which is associated with hypertensive emergencies. Additionally, as patient tolerance permits, BP should be measured in various positions (supine, sitting, and standing) to assess for volume depletion. Subsequent BP and heart rate measurements are necessary for monitoring trends over time.

After vital signs are obtained, a focused physical examination should evaluate cardiovascular, pulmonary, and neurological functions. Starting with the head and neck, jugular vein distention should be inspected, as its presence may indicate acute heart failure, a potential complication of hypertensive emergencies. Heart and lung auscultation should follow, with optimal heart sound assessments performed at the aortic (second intercostal space, right of the sternum), pulmonic (second intercostal space, left of the sternum), tricuspid (fourth intercostal space, left of the sternum), mitral (fifth intercostal space, midclavicular line), and Erb's point (third intercostal space, left of the sternum). Heart auscultation should assess the S1 sound (synchronous with the carotid pulse) and the S2 sound, focusing on rate, rhythm, and the presence of abnormal sounds such as gallops, murmurs, or rubs. For instance, an S3 sound heard best in the mitral area may signify pulmonary edema, while a new diastolic murmur detected at Erb's point could suggest aortic regurgitation related to acute aortic dissection (Muiesan et al., 2015).

Lung auscultation should cover all fields and include full inspiration and expiration cycles to detect bibasilar crackles (rales), indicative of acute pulmonary edema. Extremity assessments should include peripheral pulses for absence or delay (potentially suggesting aortic dissection), the presence of peripheral edema (often linked to heart failure), and evaluations of bilateral movement, sensation, and muscle strength. More detailed neurological, abdominal, and fundoscopic examinations, including cranial nerve evaluation, cerebellar testing, palpation for abdominal masses or bruits, and dilated eye exams for soft exudates, hemorrhages, or papilledema, are typically performed by the primary provider, Clinical Presentation, Diagnosis, and Prevalence of Target Organ Dysfunction:

Neurologic Target Organ Dysfunction

Neurologic target organ damage is prevalent in approximately 45% of cases and includes conditions such as cerebral infarction, hemorrhagic stroke, and hypertensive encephalopathy. Clinically, patients may present with symptoms such as headaches, visual disturbances, and nausea. Observable signs may include altered mental status, focal neurologic deficits, unsteady gait, seizures, cortical blindness, and visual field deficits. To confirm a diagnosis, imaging techniques like computed tomography (CT) or magnetic resonance imaging (MRI) of the brain are utilized.

Cardiovascular Target Organ Dysfunction

Cardiovascular target organ damage is observed in nearly 49% of cases and may manifest as acute coronary syndrome (ACS) or acute heart failure, often accompanied by pulmonary edema. Symptoms can include chest pain or pressure

radiating to the jaw, epigastrium, shoulders, or arms, along with dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cough, fatigue, and palpitations. Physical examination findings may reveal jugular vein distension, edema, basilar lung crackles, the presence of a third heart sound, or a new-onset murmur. Diagnostic evaluation typically involves a 12-lead electrocardiogram (ECG) and chest radiography. Cardiac biomarkers such as troponin are measured if ACS is suspected, while B-type natriuretic peptide (BNP) levels are evaluated if acute heart failure is a concern. Additional assessments may include echocardiography for structural or functional insights.

Renal Target Organ Dysfunction

Renal involvement, with a prevalence of approximately 10%, may present as acute kidney injury or failure. Symptoms such as hematuria or oliguria are commonly reported. Laboratory evaluations include urinalysis, serum creatinine, sodium, potassium, and blood urea nitrogen (BUN) levels. Imaging studies, such as renal ultrasonography, can provide further diagnostic clarity.

Vascular Target Organ Dysfunction

Vascular complications occur in around 6.5% of cases and include eclampsia and acute aortic dissection (type A or B). Eclampsia typically occurs in pregnant individuals beyond 20 weeks of gestation and is associated with dyspnea, visual disturbances, headaches, and seizures. Diagnostic testing for eclampsia involves evaluating electrolytes, serum creatinine, urinalysis, and liver function tests. Acute aortic dissection, characterized by syncope and severe chest pain with or without radiation to the back, can present with neurologic deficits, limb ischemia, new-onset murmurs, and asymmetry in pulse and blood pressure between arms. Diagnostic measures include chest radiography, CT angiography of the chest and abdomen, and transesophageal echocardiography for precise imaging.

This structured approach ensures a comprehensive understanding and diagnosis of target organ dysfunctions across neurologic, cardiovascular, renal, and vascular systems.

Treatment

Goals of Care

When managing hypertensive emergencies, the primary treatment goals are to identify the underlying cause of the acute BP elevation, (2) mitigate target organ damage through controlled BP reduction, and (3) closely monitor the patient for adverse medication effects and signs of deteriorating vital organ perfusion. Patients diagnosed with a hypertensive emergency require intensive care unit admission for rapid intravenous BP-lowering therapy and continuous hemodynamic monitoring (Weder, 2011).

BP reduction targets depend on the type of target organ damage and the required speed of BP reduction. Although no randomized controlled trial data exist to determine the ideal BP reduction rate or extent for hypertensive emergencies, specific situations necessitate urgent intervention. These include aortic dissection, severe preeclampsia or eclampsia, and pheochromocytoma-related hypertensive crises. For such cases, systolic BP should be reduced within the first hour to less than 140 mm Hg for preeclampsia, eclampsia, and pheochromocytoma, and to less than 120 mm Hg for aortic dissection.

In most other cases, systolic BP should initially be reduced by no more than 25% within the first hour to prevent ischemic complications in the brain, coronary arteries, or kidneys. Subsequent goals include reducing systolic BP to 160 mm Hg and/or diastolic BP to 100 mm Hg within 2 to 6 hours, with outpatient treatment goals achievable within 24 to 48 hours. Exceptions include ischemic and hemorrhagic strokes, which require more gradual BP reductions. While treatment timelines and goals vary internationally, these recommendations provide a general framework. Specific BP goals and timelines for each patient are determined by the medical team. The following paragraph provides BP targets based on clinical presentation, timeline, and commonly used medications for different target organ damage scenarios.

In patients experiencing hypertensive emergencies, blood pressure (BP) management strategies are tailored to the specific target organ damage involved. Each condition requires precise BP reduction goals within an appropriate timeline to prevent further complications.

For hypertensive encephalopathy, immediate intervention is required to reduce the mean arterial pressure (MAP) by 20%–25%. Commonly used agents include labetalol, nicardipine, and nitroprusside. In cases of acute aortic dissection, the target is to achieve a systolic blood pressure (SBP) below 120 mm Hg and a heart rate below 60 beats per minute. Medications such as esmolol, labetalol, nitroglycerin, nicardipine, and metoprolol is effective in this scenario.

Patients with acute coronary syndrome should have their SBP reduced to below 140 mm Hg immediately. Esmolol, labetalol, nicardipine, and nitroglycerin are among the recommended treatments. Similarly, in acute pulmonary edema, the SBP should be brought under 140 mm Hg promptly, with therapeutic options including clevidipine, nitroglycerin, and nitroprusside, often in conjunction with a loop diuretic.

In the context of eclampsia or severe preeclampsia, immediate BP reduction to an SBP below 140 mm Hg and a diastolic BP (DBP) below 105 mm Hg is essential. Hydralazine, labetalol, and nicardipine are commonly employed for this purpose. For acute hemorrhagic stroke, a gradual reduction of SBP to below 180 mm Hg is recommended while avoiding sudden BP drops. Medications such as labetalol, nicardipine, and urapidil are used to achieve this goal.

Management of acute ischemic stroke varies based on treatment plans. If thrombolysis or thrombectomy is anticipated, the BP should be reduced to below 185/110 mm Hg within an hour. Post-thrombolysis, the target BP for the next 24 hours should be less than 180/105 mm Hg. In cases where thrombolysis is not indicated, the initial BP can be allowed to remain below 220/120 mm Hg with a gradual reduction

of MAP by 15% over 24–48 hours unless other comorbidities necessitate stricter control. Medications such as labetalol, nicardipine, and nitroprusside are typically used, often in combination with thrombolytic therapy when appropriate.

This detailed approach emphasizes the need for immediate and targeted interventions to achieve optimal outcomes in hypertensive emergencies.

First-Line Pharmacologic Therapy

The selection of first-line pharmacologic agents for BP reduction in hypertensive emergencies depends on the specific target organ damage and the patient's comorbid conditions, which may contraindicate certain medications. Intravenous administration is the preferred route due to its rapid onset, short half-life, ease of dose titration, and quick resolution of effects in case of adverse reactions. Next section outlines intravenous medications commonly utilized in managing hypertensive emergencies (Bowry et al., 2014).

Medications Used in the Treatment of Hypertensive Emergencies

The treatment of hypertensive emergencies involves various medications tailored to the patient's condition, with careful consideration of the onset and duration of action, dosage, and potential contraindications.

Adrenergic blockers are frequently utilized in this context, including esmolol (a beta-1 blocker), labetalol (a blocker of alpha-1, beta-1, and beta-2 receptors), metoprolol (a beta-1 blocker), phentolamine (a nonselective alpha-blocker), and urapidil (an alpha-1 blocker with serotonin agonist properties). Esmolol has an onset of action of 1–2 minutes and a duration of 10–30 minutes, with a usual dose of 0.5–1 mg/kg as an intravenous (IV) bolus, followed by an infusion of 5–200 mg/kg per minute. Labetalol has a slightly slower onset of 5–10 minutes and lasts for 3–6 hours; it is administered as a slow IV injection of 0.3–1.0 mg/kg every 10 minutes or as an infusion up to 3 mg/kg per hour, not exceeding a cumulative dose of 300 mg. Metoprolol is given as a 2.5–5 mg IV bolus over 2 minutes, with repeated doses every 5 minutes up to a maximum of 15 mg. Phentolamine and urapidil also have rapid onsets of 1–2 and 3–5 minutes, respectively, with phentolamine dosed at 0.5–1 mg/kg as a bolus or 50–300 mg/kg per minute as an infusion, and urapidil administered as a 12.5–25 mg IV bolus with an infusion rate of 5–40 mg/hour. These agents require monitoring for side effects such as bradycardia, heart failure, bronchospasm (with labetalol), or flushing and tachyarrhythmias (with phentolamine).

Enalaprilat, an angiotensin-converting enzyme inhibitor, has an onset of action of 5–15 minutes and a duration of 4–6 hours. It is dosed at 0.625–1.25 mg IV over 5 minutes, with subsequent increases up to 5 mg every 6 hours as needed. However, it is contraindicated in patients with a history of angioedema, pregnancy, bilateral renal artery stenosis, or acute myocardial infarction. Enalaprilat's relatively slow onset requires careful monitoring for adverse effects like hyperkalemia, worsening renal function, headache, and nausea.

Calcium channel blockers, particularly clevidipine and nicardipine, are effective in managing hypertensive emergencies. Clevidipine has a rapid onset of 2–3 minutes and a duration of 5–15 minutes. It is initiated at 2 mg/hour IV, titrated every 2 minutes to a maximum of 32 mg/hour, with a maximum duration of 72 hours. Nicardipine, with a slightly longer onset of 5–15 minutes and a duration of 30–40 minutes, is dosed at 5 mg/hour IV, increased by 2.5 mg/hour up to 15 mg/hour. Both agents require monitoring for reflex tachycardia and headache, with nicardipine being contraindicated in liver failure.

Fenoldopam, a dopamine-1 receptor agonist, has an onset of 5–15 minutes and lasts for 30–60 minutes. It is administered at 0.1–0.3 mg/kg/min IV, titrated every 15 minutes up to a maximum infusion rate of 1.6 mg/kg/min. Contraindications include increased intraocular or intracranial pressure and sulfite allergy. Patients must be monitored for dizziness, hypokalemia, reflex tachycardia, or bradycardia.

Vasodilators are another cornerstone of hypertensive emergency treatment. Hydralazine, a direct vasodilator, has an onset of 10–30 minutes and a prolonged duration of 2–4 hours. It is given as a slow IV infusion of 10–20 mg, repeated every 4–6 hours. Nitroglycerin, a nitric oxide-dependent vasodilator, has a faster onset of 1–5 minutes and lasts 3–5 minutes. It is initiated at 5 mg/min IV, titrated to a maximum of 20 mg/min. Sodium nitroprusside, also a nitric oxide-dependent vasodilator, acts immediately and is dosed at 0.3–0.5 mg/kg/min, increasing by 0.5 mg/kg/min to a maximum of 10 mg/kg/min. Hydralazine's unpredictable BP response necessitates close monitoring, while nitroglycerin is reserved for acute coronary syndrome or pulmonary edema and is contraindicated in volume depletion or cerebral hemorrhage. Prolonged use of nitroprusside requires caution due to potential cyanide toxicity, mitigated by administering thiosulfate if infusion rates exceed 4–10 mg/kg/min.

This spectrum of therapeutic options underscores the importance of individualized management in hypertensive emergencies to balance efficacy and safety effectively.

Implications for Nursing Care

All patients experiencing a hypertensive emergency should be admitted to the intensive care unit (ICU), where they can receive comprehensive monitoring, including intraarterial BP monitoring and continuous cardiac telemetry for dysrhythmias. Compared to non-invasive BP measurements (obtained via automated BP cuffs), intraarterial BP monitoring provides greater accuracy and real-time BP readings, especially crucial for patients receiving intravenous antihypertensive therapy. Nevertheless, the initiation of treatment should not be delayed while awaiting the placement of an intraarterial line.

Nurses play a critical role in anticipating BP fluctuations in response to treatment and must closely monitor BP, heart rate, and rhythm, remaining vigilant for abrupt changes that could result in ischemia of the brain, heart, or kidneys. For pregnant patients, fetal monitoring will likely be part of the care plan as ordered by the medical team. Other monitoring priorities include changes in mental orientation, mood, consciousness, vision, nausea, vomiting, and intake/output patterns, as these

may indicate altered perfusion to vital organs. Ensuring patient comfort is essential, as pain and anxiety can exacerbate BP elevations and should be promptly addressed.

Discharge Planning

Discharge planning should commence early, well before the patient's actual discharge. Nurses should evaluate social determinants of health that might hinder adherence to the treatment regimen post-hospitalization. This assessment includes determining whether the patient has stable housing, access to heart-healthy foods and clean water, physical and mental health care, social support, and affordable, reliable transportation (Tiase et al., 2022). To promote adherence, nurses can advocate for simplified medication regimens, such as once-daily dosing and the use of generic drugs when appropriate.

Discharge education should cover the timing and provider of the first follow-up appointment, as well as guidance on whom to contact for medication-related side effects. Patients who have suffered significant target organ damage, such as stroke, myocardial infarction, or heart failure, should be referred for specialized programs like cardiac rehabilitation. Priorities for educating patients and their families are detailed below.

- Treatment of HTN is lifelong.
- Elevated BP is not typically associated with symptoms, thus ongoing monitoring of BP is
- essential.
- Importance of adherence to nonpharmacologic and pharmacologic therapy to control BP.
- Lifestyle modifications to obtain/maintain BP control (including DASH eating plan, reduction in dietary sodium, daily physical activity, moderation of alcohol, weight reduction in those with overweight or obese, adequate sleep, and tobacco cessation).
- Benefits of home BP monitoring and how to implement home BP monitoring once
- discharged from the hospital.
- Importance of follow-up care with primary care provider and specialty provider.
- How to access a pharmacy assistance program or social worker if financial challenges or other social determinants of health are present, which influences the patient’s ability to follow the treatment regimen.

Conclusion

Nurses are integral to the effective management of hypertensive crises, ensuring timely diagnosis, intervention, and follow-up care. Their expertise in monitoring patient status, administering medications, and educating patients and families is vital in reducing the risks associated with acute blood pressure elevations. Tailored interventions, guided by evidence-based practices, enable nurses to address

the multifaceted challenges of hypertensive emergencies, safeguard organ function, and improve patient outcomes. Furthermore, nurses play a key role in post-discharge planning, emphasizing adherence to treatment regimens and lifestyle modifications to prevent recurrence.

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