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Review Article

Clinical Diagnosis and Treatment of Hypertensive Emergencies

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Abstract

Hypertensive emergency is a rise in blood pressure (BP) >180/120 mmHg with an associated with end-organ injury, and symptoms and signs are usually dramatic. Complete medical history and quick clinical examination key to treatment. Intravenous antihypertensive medications chosen based on comorbid conditions, type and extent of end-organ injury, pharmacodynamics, and pharmacokinetic property of drugs. Target BP reduction is to reduce 25% of mean arterial pressure in 1st h and further reduce systolic blood pressure to < 160 and diastolic blood pressure < 100 in next 2–6 h, and maintain the same BP for next 24 h, exceptions are aortic dissection, Eclampsia/Preeclampsia, acute stroke, and pheochromocytoma crisis. Labetalol, nitroglycerine, nicardipine, and esmolol cover most of the spectrum of hypertensive emergencies. Over enthusiastic blood pressure correction to be avoided to prevent ischemic effects may arise from rapid reduction in blood pressure.

Key words: Hypertensive emergency, End-organ damage, Compelling conditions, Clevidipine, Nicardipine, Nitroglycerine, Esmolol, Labetalol

Introduction

"Hypertensive Crisis" is an abrupt increase in systolic blood pressure (SBP) >180 mmHg and/or elevation diastolic blood pressure (DBP) >120 mmHg. Based on the presence of acute end-organ damage, a hypertensive crisis can be defined as Hypertensive Emergency or Urgency. Hypertensive urgency defined as "severe blood pressure (BP) elevation in chronic hypertensive patients with no acute target organ injury or dysfunction." Do not require hospital admission, and reduction in blood pressure (BP) can be achieved with oral medication in the emergency room with subsequent outpatient follow-up.[1,2]

"Hypertensive emergency is a critical rise in BP >180/120 mmHg with associated newly developed, progressive, or deteriorating target organ injury."[1,3] In 1928, the term "malignant hypertension" was coined to denote hypertensive emergency, as the patient's outcome was as same as the patient diagnosed with cancer. Because of rapid advancement in the medical field and availability of excellent medications, in-hospital mortality reduced drastically to 2.5%.[4]

Clinical Diagnosis

Symptoms and signs are usually dramatic, and when a patient presents to the emergency room with increased BP of >180/120 mmHg with symptoms and signs suggestive of end-organ damage, hypertensive emergency to be considered.

Patient presenting to the emergency room with a new onset of symptoms along with severely elevated BP, a complete medical history along with a quick physical examination to identify end-organ damage plays an important role. In history, the key points are the duration of hypertension, history of compliance to drugs, list of antihypertensive medications in use, and time of last dose administration.[3] Physical examinations include assessment of peripheral pulses, measurement of BP in both upper limbs; other specific clinical features are summarized Table 1.[5]

Therapeutic Approach

Oral antihypertensive medications are to be discouraged in the management of hypertensive emergencies. Once a patient

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Table 1: Features of end-organ damage and prevalence

Clinical condition	Prevalence	Features of end-organ damage
LVF	22.5%	Dyspnea, palpitations, S3 gallop, rales, b lines in chest X-ray
ACS	12%	Acute chest pain, dyspnea, ST-T changes in ECG, positive cardiac biomarkers, LV dysfunction in echocardiogram
AORTIC DISSECTION	2%	Severe acute tearing retrosternal chest pain radiating to back in the hypertensive patient, unequal pulses. Unequal blood pressures. CT imaging suggestive of dissection
Hypertensive encephalopathy- PRES (posterior reversible encephalopathy syndrome)	16.5%	Occipital headache, visual changes altered sensorium, seizures, papilledema, retinal exudates, No focal deficits
CVA- ICH/ Ischemic strike	4.5/24.5%	Headache, focal neurological deficits. Imaging suggestive of infarct or bleed
Eclampsia/Preeclampsia	4.5%	Pregnant or recently postpartum status hyperreflexia, proteinuria peripheral edema, hemolysis, elevated AST, ALT, decreased platelet counts.
Catecholamine excess	-	Clinical diagnosis in the scenario of sympathomimetic drug use (i.e., cocaine or amphetamines) or pheochromocytoma

ICP: Intracranial pressure, LVF: Left ventricular failure, ACS: Acute coronary syndrome, CVA: Company voluntary arrangements

diagnosed to have hypertensive emergencies, the patient needs to be hospitalized and requires intensive care unit care to treat and assess end-organ damage, and to administer intravenous antihypertensive medications as well as to monitor hemodynamic parameters including intra-arterial pressures.[1,2]

The drugs preferred for the management of hypertensive emergencies are continuous intravenously administered short-acting agents, although no evidence is available which class of drugs gives more benefits.[6] Antihypertensive medications are chosen based on comorbid conditions, extent of end-organ damage, pharmacodynamic, and pharmacokinetic properties of drugs.[7] Excessive and overenthusiastic BP reduction may precipitate ischemia in cerebral, myocardial, and renal tissues.[8]

In the treatment of hypertensive emergencies, few sets of patients require different BP goals, also labeled as compelling conditions due to their unique hemodynamic, for rest of the patients BP goals, are as shown in Table 2.

Table 2: BP treatment goals for hypertensive emergency

Goal time	BP target
1st h	Reduce MAP by 25%
2–6 h	SBP 160 mm Hg and/or DBP 100–110 mmHg
6–24 h	Maintain goal for hours 2–6 during first 24 h
24–48 h	Outpatient BP goals according current guidelines

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, MAP: Mean arterial pressure, BP: Blood pressure

Compelling Conditions

These conditions due to their unique hemodynamic, requires a different set of BP targets in treating a hypertensive emergency.

1. Acute stroke
2. Aortic dissection
3. Severe Preeclampsia/Eclampsia
4. Pheochromocytoma crisis.

Pharmacotherapy

The drugs used in the management of hypertensive emergencies are described in detail as class of drugs and each drug is summarised with respect to, common indications and special consideration, and dose in [Tables 3 and 4].

Calcium channel blockers – dihydropyridines

Nicardipine is short-acting, parenteral, dihydropyridine calcium channel blocker. It is a selective coronary and cerebral vasodilator.[9] One of the special characteristic features is it crosses the blood brain barrier and accumulates in ischemic tissues and causes localized vasodilatation without increasing the intracranial pressure (ICP). The onset of action is 5–15 min and acts for 4–6 h. The initial dose is 5 mg/h infusion, and the dose can be escalated every 5 min with an increment of 2.5 mg/h, and the maximum dose is 15 mg/h. The drug is safe in elders and no need of dose reduction. Nicardipine to be avoided in severe aortic stenosis.[1,2]

Clevidipine is an ultra-short acting calcium channel blocker that belongs to the class of dihydropyridine. It causes vasodilatation by blocking L type calcium channel and consequentially reduces peripheral vascular resistance.[10-12] Clevidipine reduces myocardial infarct size, maintains coronary endothelial function, and protects renal function. Elderly patients require dose adjustments. Clevidipine is contraindicated in patients who have abnormal lipid metabolism, and allergic to egg or soya related products.[13,14]

Nitric Oxide-derived Vasodilators

Nitroglycerine is a venodilator, the mechanism by which reduces BP is by reducing both preload and cardiac output.[8] The initial dose is 5 mcg/min, can be increased in every 3–5 min to a maximum of 20 mcg/min with increments of 5 mcg/min. The commonly encountered adverse effect is a headache, which usually subsides once the dose is reduced. In volume-depleted patients, nitroglycerine known to cause hypotension with to reflect tachycardia.[15] It is an ideal drug while treating

Table 3: Indications and Special consideration[1-3,16]

Drugs	Common indications	Special consideration
Nitroglycerine	LVF, acute coronary syndrome	Headache, Tachyphylaxis
Nitroprusside	LVF-pulmonary edema, aortic dissection	Cyanide accumulation, Increases ICP, coronary steal
Nicardipine	Acute CVA, hypertensive encephalopathy	Avoid in patients allergic to egg/soya
Clevidipine	Acute CVA, hypertensive encephalopathy	Reflex tachycardia, avoid in severe aortic stenosis
Esmolol	Aortic dissection, ACS, perioperative hypertension	Avoid in ADHF, ii and iii degree AV Block, bradycardia
Labetalol	Pregnancy, Acute aortic dissection, ACS, CVA	Avoid in ADHF, ii and iii degrees AV Block, bradycardia
Enalprilat	Acute left ventricular failure	Contraindication-pregnancy
Phentolamine	Catecholamine excess state-pheochromocytoma, cocaine toxicity	Use beta-blockers for rate control after adequate alpha blockade if necessary
Fenoldopam	Most of the conditions	Increases ICP, intraocular pressure

ICP: Intracranial pressure, LVF: Left ventricular failure, ACS: Acute coronary syndrome, CVA: Company voluntary arrangements

Table 4: Drug Dosages

Drugs	Dose	Onset of action
Nitroglycerine	5–100 mcg/min, Titrate by 5–25 mcg/min q5–10 min	2–5 min
Nitroprusside	0.25–10 mcg/kg/min, Titrate by 0.1–0.2 mcg/kg/min q5 min	Seconds
Nicardipine	5–15 mg/h, Titrate by 2.5 mg/h q5–10 min	5–10 min
Clevidipine	IV 1–6 mg/h Titrate by 1–2 mg/h	1-4 min
Esmolol	IV 25–300 mcg/kg/min, Titrate by 25 mcg/kg/min q3–5min	1–2 min
Labetalol	20–80 mg iv bolus every 10 min IV 0.5–10 mg/min, Titrate by 1–2 mg/min q2 h	2–5 min, peak 5–15 min
Enalaprilat	1.25–5 MG 1Vq 6 h, max dose: 5 mg q6h	15–30 min
Hydralazine	IV bolus: 10–20 mg IM: 10–40 mg q30 min	IV: 10 min IM: 20 min
Phentolamine	IV bolus: 1–5 mg, max 15 mg	Seconds
Fenoldopam	0.1–0.3 mcg/kg/min	10–15 min

acute pulmonary edema because of its favorable effects on hemodynamics.

Nitroprusside is a nitric oxide donor. It is a vasodilator known to decrease both preload and after load. The action of drug starts within seconds. Unfavorable pharmacodynamic effects are coronary steal phenomenon, and it known to increase ICP. Patients are prone to cyanide toxicity and thiocyanate toxicity in the presence of hepatic dysfunction and renal dysfunction, respectively.[16]

Direct Vasodilators

Hydralazine is peripheral vasodilator acts by arteriolar smooth muscle relaxation, due to its unpredictable BP response and prolonged action, it is no more the first line of the drug in treating a hypertensive emergency.[17]

Adrenergic Blockers

Esmolol is a short-acting selective beta-blocker and metabolism is independent of the liver and kidney function making it as an ideal drug in critically ill patients. While using esmolol heart rate to be monitored continuously. Esmolol is contraindicated in patients with bradycardia, acute decompensated heart failure, and with concomitant beta-blocker use.[8,16]

Labetalol is a non-selective beta-blocker; it can be used both as intermittent bolus doses or continuous intravenous infusion. The initial dose is 0.3–1.0 mg/kg, slow intravenous injection every 10 min (maximum 20 mg). For intravenous infusion starting dose is 0.4–1.0 mg/kg/h up to 3 mg/kg/h. Contraindications are bradycardia, second degree atrioventricular (AV) blocks, acute decompensated heart failure, chronic obstructive pulmonary disease (COPD), and bronchial asthma.[1,16]

Phentolamine is competitive peripheral alpha-blocker with more affinity for alpha1 receptors causes' direct vasodilation. It reduces BP by reducing systemic vascular resistance with a compensatory increase in heart rate and cardiac output. It is used in the state of catecholamine excess states such as pheochromocytoma, cocaine toxicity, and clonidine withdrawal.[1,16] Phentolamine used as a bolus dose of 5 mg intravenously; the dose can be repeated every 10 min until the target BP achieved.[1,3] Common adverse effects are flushing and headache and rebound tachycardia, this tachycardia can worsen an oxygen supply-and-demand mismatch leading to angina or myocardial infarction in patients with coronary artery disease.[18]

Dopamine 1-Receptor Selective Agonist

Fenoldopam is rapid acting D1 receptor agonist. Fenoldopam reduces BP due to its peripheral vasodilator properties and also increases renal blood flow with diuretic effects. Fenoldopam can cause anaphylactic reactions in patients who are allergic to sulfa drugs as it contains sodium metabisulfite. Other adverse effects are headache, nausea, vomiting, and flushing, and hypokalemia.[19]

Angiotensin Enzyme Converting (ACE) Inhibitors

Enalaprilat is an ACE inhibitor, reduces BP due to its vasodilatory property. The usual dose is 1.25 mg intravenous administration over 5 min. The action starts after 15–30 min, and single bolus dose may last up to 24 h. Unpredictable BP responses making dose adjustment difficult. Enalaprilat contraindicated in bilateral renal artery stenosis, pregnancy, and acute myocardial infarction.[2,20]

Furosemide a loop diuretic, is not recommended as first-line therapy in the management of hypertensive emergencies, though furosemide can be used in conditions such as renal parenchymal disease with fluid overload status.[20,21]

Cardiovascular Emergencies

Acute aortic dissection

The progression of aortic dissection depends on shear stress; therefore, in the management of aortic dissection goal is to reduce both heart rate and BP.[22,23] Within minutes starting treatment, heart rate should be brought down to <60/min. Short-acting beta-blockers esmolol or labetalol are the ideal drugs in aortic dissection.[1] BP to be reduced to below 120 mmHg within the first 20 min. With the adequate dose of beta-blocker use, still BP is elevated nitroglycerine or nitroprusside can be initiated. To avoid reflex tachycardia produced by vasodilators, which further worsens the dissection, Beta-blocker to be used before vasodilator administration.[24]

Acute pulmonary edema

Ideal drugs are nitroglycerin, nitroprusside or clevidipine.[1] BP should be reduced by 25% in first 1 h, followed by reduce to at least 160/100 mmHg in the next 6 h with the target of reaching normal BP values in next 48 h. Beta-blockers are contraindicated in the management of acute pulmonary edema as they are known to cause bronco constriction and impair respiratory function.[25]

Acute coronary syndromes (ACS)

According to the American College of Cardiology/American Heart Association guidelines, ACS with a hypertensive emergency the recommended drugs are nitroglycerin, esmolol, labetalol, and nicardipine.[1] The target BP is <140/90 mmHg in stable patients. Diastolic pressure should not be allowed to drop below 60 mmHg as it reduces coronary blood flow with subsequent worsening of ischemia.[26] Beta-blockers are contraindicated in the presence of left ventricular failure (LVF), hypotension, second- or third-degree AV block, COPD, and bronchial asthma.[20]

Hypertensive Emergencies in Pregnancy

The 2018 European Society of Cardiology Task Force on Cardiovascular Diseases during pregnancy considers an SBP of at least 170 mmHg or DBP of at least 110 mmHg an emergency in a pregnant woman.[27]

While treating pregnant patients sudden or abrupt decrease in BP may lead to the harmful fetal outcome, to avoid these complications, reduce mean arterial pressure (MAP) by 20–25% over first few minutes to hours and further reduce BP to the target of 160/110 mmHg or less over subsequent hours.[28,29] The ideal drugs are labetalol, and nicardipine, which have shown to be safe and effective. Fetal heart rate monitoring is necessary while using labetalol, and cumulative dose should not exceed 800 mg/24 h. Intravenous Urapidil can also be used. When a patient presents with pulmonary edema, intravenous nitroglycerine is the drug of choice. Hydralazine is associated with more perinatal adverse effects compared to other drugs.[30] Angiotensin-converting enzyme inhibitors and nitroprusside are contraindicated in Eclampsia/Pre-eclampsia.[31]

Neurological Emergencies

Acute intracerebral hemorrhage

Ideal drugs for acute intracranial hemorrhage are nicardipine, esmolol, and labetalol. Drugs which increase ICP or reduce cerebral perfusion are contraindicated, for example, nitroprusside, and hydralazine. BP reduction is indicated only when SBP is >220 mmHg. Studies have shown routine immediate BP reduction below 140 mmHg can be harmful.[32,33]

Acute ischemic stroke

Elevated BP seen in ischemic stroke is a physiological response to maintain adequate perfusion pressure, governed by the equation, CPP=MAP-ICP (CPP: Cerebral perfusion pressure, MAP: Mean arterial pressure, ICP: Intracranial pressure). ICP is elevated in ischemic stroke, reducing the MAP may decrease the tissue perfusion with worsening of ischemia. However, the indications to reduce the BP in ischemic stroke are

1. Planning thrombolysis or endovascular management for stroke – Before starting thrombolysis reduce BP to <180/105 mmHg and maintain same BP up to 24 h.
2. Presence of other end organ damage – LVF, ACS, and acute aortic dissection
3. BP >220/120 mmHg.

Severe elevation of BP >220/120 mmHg and presence of other end-organ damage, the goal is to reduce 15% MAP over a period of 24 h.[16] If BP is <220/120 mmHg on presentation, routine BP reduction in first 48–72 h may not be beneficial in these set of patients.[34,35] Drug of choice in ischemic stroke are nicardipine and labetalol.

Hypertensive encephalopathy including press

Once company voluntary arrangements is ruled out, reduce MAP by 25% in 1st h and further reduce the BP to 160/100 mmHg over 2–6 h. Recommended agents include nicardipine, labetalol, and clevidipine.

Perioperative Hypertension

It is defined as an elevation of BP of 160/90 mmHg or higher or an SBP elevation of at least 20% of the pre-operative value, that lasts for more than 15 min. Post-operative hypertension may have significant adverse outcomes in both cardiac and non-cardiac patients. Due to increased sympathetic tone and vascular resistance, hypertensive crises, and hypertension, are very common in the early post-operative period. Post-operative hypertension commonly begins ~10–20 min after surgery and may last up to 4 h, commonly related to increased vascular resistance and sympathetic tone. The best approach to treatment is prevention. Often these complications develop due to stopping or withdrawal of antihypertensive medications in pre-operative period. Administering antihypertensive medicines on the morning of the day of surgery effectively prevents post-operative hypertension. Esmolol, nitroglycerine, clevidipine, or nicardipine are ideal drugs manage perioperative hypertension.[1,36]

States with Excessive Catecholamine Discharge

Pheochromocytoma is a hyperadrenergic status. This condition treated by intravenous infusion of phentolamine, nicardipine, or clevidipine.[1] SBP reduced to 140 mmHg in 1st h using above-mentioned drugs. Alpha-adrenergic blockade is important in controlling BP. For significant tachycardia after alpha-blockade, beta-blockers can be used to control heart rate only after adequate alpha-blockade.[37]

Conclusion

Early diagnosis and quick assessment of end-organ damage in emergency room play a crucial role in the early management of a hypertensive emergency, which avoids further end-organ damage. Drug regimen to be tailored according to patient comorbid condition, extent and type of end-organ damage and should be aware of compelling conditions. Labetalol, nitroglycerine, esmolol, and nicardipine effectively covers most of the spectrum of hypertensive emergencies. Overenthusiastic BP correction to be avoided to prevent cerebral and myocardial hypoperfusion. Treat the patient, not the numbers.

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