Hypertensive Crisis in ICU

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• Hypertension is one of the most common chronic medical condition.

• Although there are improvements in the diagnosis and treatment of hypertension but the control rate remains low.

• Uncontrolled hypertension can progress to hypertensive crisis.

• The prevalence of hypertensive crisis in hypertension population is approximately 1% in the United States.

• Hypertensive crisis accounts for more than 25% of all medical visits to ER.
Definition

- **Hypertensive crisis** is defined as a severe elevation in blood pressure (SBP > 180 mmHg or DBP > 120 mmHg).

- **Hypertensive urgency:**
  - Severe elevation in BP without progressive target organ dysfunction.

- **Hypertensive emergency:**
  - Severe elevation in BP complicated by evidence of impending or progressive target organ dysfunction.

- The severity of hypertensive crises is determined by the presence of target organ damage rather than the level of blood pressure.
Causes of Hypertensive crisis

• The most common cause of hypertensive crisis is an abrupt increase in BP in patient with chronic hypertension.

❖ Triggering factors:

➢ Medication noncompliance.

➢ Drug: Abrupt withdrawal of antihypertensives, illicit drug use, interaction with monoamine oxidase inhibitors.
Causes of Hypertensive crisis

Second common causes:

1. Renal disease: Parenchymal disease and Renovascular disease.

2. Endocrine disease: Cushing’s syndrome, Pheochromocytoma, etc.


4. Postoperative state.

5. CVA: infarction/hemorrhage, head injury.
Pathophysiology

- Pathophysiology of hypertensive crisis is not well known and may be due to multifactorial.

1. Elevation in BP and increased systemic vascular resistance:
   - Increased endothelial permeability, cell proliferation, activation of coagulation cascade.
   - Endothelial damage: release of vasoconstrictors.

2. RAAS:
   - Stretch of the vessel wall, spontaneous natriuresis, intravascular volume depletion, activation of RAAS.
Clinical manifestations and assessment

• Blood pressure must be measured correctly.

• Adequate assessment is very important to determine whether pt has hypertensive urgency or emergency.

• Perform complete evaluation in patients with a hypertensive crisis to effectively reverse, intervene and correct the underlying trigger, as well as improved long term outcomes after the episode.

• A detailed history, physical examination, relevant lab tests, EKG, echo and radiographs.
Hypertensive Urgency:

- Pt with inadequately controlled HTN or noncompliance.
- Severe headache, epistaxis, chest pain, severe anxiety, shortness of breath.
Hypertensive Emergency:

- Acute coronary syndrome: unstable angina and myocardial infarction.
- Acute pulmonary edema.
- Acute aortic dissection.
- Pre-eclampsia and eclampsia.
- Acute renal failure.
- Catecholamine excess.
- Hypertensive encephalopathy and Stroke.
- Postoperative hypertensive crisis.
Management

Hypertensive Urgency:

- Pt can be managed in an outpatient setting or inpatient observation (Diabetes, Hx of stroke or CAD or medication non-compliance).

- Treated with oral antihypertensive agents. Start with very low dose of oral agents using incremental doses as needed.

- Gradual and controlled reduction of BP, especially in pts with highest risk for hypotensive complications: the elderly, severe PVD, severe CAD and intracranial disease.

- Goal: reduce BP to 160/110 mm Hg over several hours to days. Outpatient follow up within 24-48 hrs after discharge.
Management

Hypertensive emergency:

• Require admission to an ICU.

• Treatment must be individualized, based on the extent of end-organ damage as well as other comorbid conditions.

• Control BP with a parenteral, titratable antihypertensive agents to avoid further end organ damage.

• Continuously monitoring BP.
Management

• Goal: mean arterial pressure should not be lowered more than 20% over a period of several minutes or hours.

• Aortic dissection: BP must be achieved within 10 minutes (SBP <120 and MAP <80).

• Volume expansion with IV saline solution in the presence of concomitant hypovolemia.

• Caution when treating a hypertensive crisis in pts with stroke.
Parenteral agents for hypertensive emergency

1 Nicardipine:
- Dihydropyridine calcium channel blocker. Arterial vasodilator
- Rapid onset of action (1-5 minutes), easy to use.
- Limitation: long half-life.
- Indication: all hypertensive emergencies, perioperative HTN and controlled hypotension during anesthesia.

2 Clevidipine:
- Short acting dihydropyridine CCBs.
- Reduce BP without affecting cardiac filling pressure or causing tachycardia.
- Indication: intraoperative and critical care setting.
Parenteral agents for hypertensive emergency

3 Labetalol:
- Alpha and beta blocker.
- Maintain cardiac output and reduce total peripheral resistance.
- Rapid onset (less than 5 minutes).
- Indication: all hypertensive emergency except acute heart failure.

4 Esmolol:
- Very short acting beta blocker, short half life (9 minutes), duration (30 minutes).
- Cardioselective beta blocker.
- Independent of liver and kidney function.
- Useful for perioperative hypertensive crisis. Not recommended for pt with catecholamine excess.
**Parenteral agents for hypertensive emergency**

4 **Fenoldopam:**
- Peripheral dopamine-1 receptor agonist.
- Cause vasodilatation and sodium excretion without alpha 1 or beta 1 activation.
- Maintain or increase renal perfusion.
- Does not rebound hypertension when discontinued.
- Safely used in all hypertensive emergency, particularly in pts with renal insufficiency.

5 **Enalaprilat:**
- Parenteral ACEI. Slow onset: 1hr, duration: 6hrs.
- Response: variable and unpredictable. Used in pt with heart failure, contraindication in pregnancy and bilateral renal artery stenosis.
Parenteral agents for hypertensive emergency

6 Nitroglycerin:
• Venodilator.
• Reduce preload and cardiac output.
• Used with other meds in pt with pulmonary edema and acute MI.

7 Nitroprusside:
• Dilates both arterioles and veins. Reduce preload and afterload.
• Rapid onset and short half life.
• Side effects: increase intracranial pressure, induce a coronary steal phenomenon. Cyanide toxicity.
• Used in pt with acute pulmonary edema, severe left ventricular dysfunction and aortic dissection.
8 Phentolamine:
- Peripheral alpha 1 and 2 receptors antagonist.
- Used for pt with catecholamine excess, interactions between monoamine oxidase inhibitors and other drugs or food, cocaine toxicity, amphetamine overdose, or clonidine withdrawal.
- Used cautiously in patients with CAD, as it can induce angina or MI.

9 Hydralazine:
- A peripheral vasodilator.
- The unpredictability of response and prolonged duration of action do not make hydralazine a desirable first-line agent in pts with hypertensive emergencies.
Specific indications

1 Hypertensive encephalopathy:

- Cerebral hyperperfusion causing cerebral edema (auto-regulatory mechanism fails).
- Severe HA, nausea/vomiting, visual disorders, altered mental status and/or seizure. Symptoms appear progressively over 24-48hrs.
- It may occur with or without retinopathy and proteinuria.
- CT scan of head to rule out intracranial hemorrhage.
- Gradual lowering of the blood pressure frequently leads to rapid improvement of neurologic symptoms.
- Labetalol, nicardipine, fenoldopam, clevidipine.
Specific indications

2 Aortic dissection:

- Chest pain, back pain or abdominal pain with hypertension.
- Asymmetric pulses or blood pressure, a vascular murmur, an aortic incompetence murmur, or signs of cerebral or limb ischaemia.
- CXR: widening of mediastinum.
- CT angiogram of chest or transesophageal echocardiogram, MRI of chest or aortography.
- Achieve an SBP < 120 mmHg within 10 minutes.
- Labetalol alone or a combination of Beta Blocker with a vasodilator (increased shear stress in the vessel wall).
Specific indications

3 Acute coronary syndrome:

- Unstable angina and myocardial infarction.
- HTN causes increased myocardial stress and oxygen consumption.
- Chest pain, changes in EKG and elevated cardiac enzymes.
- Nitroglycerin with esmolol, fenoldopam, labetalol.
Specific indications

4 Acute pulmonary edema:

- HTN crisis: causative or aggravating factors.
- Shortness of breath, chest pain.
- CXR: vascular congestion, cardiomegaly.
- Loop diuretic, Enalaprilat, nitroglycerin, nitroprusside.
Specific indications

5 Pre-eclampsia and eclampsia:

• Hypertension may be present before pregnancy or after the twentieth week of pregnancy.
  ❖ Pre-eclampsia: HTN, proteinuria; HELLP
     predisposing factors: DM, twin pregnancy, molar pregnancy etc….
  ❖ Eclampsia: severe pre-eclampsia with seizure.
• Close monitoring.
• Indication for induction of labor.
• Labetalol, hydralazine, nicardipine (caution with magnesium sulfate).
Specific indications

6 Acute renal failure:

- May be the cause or a consequence of HTN emergency.
- Worsen preexisting renal failure. Increase in extracellular volume and vasoconstriction due to RAAS activation.
- Nicardipine, fenoldopam.

7 Postoperative hypertensive emergency:

- Occurs in early postoperative period.
- Due to adrenergic mechanism.
- Nicardipine, nitroprusside, esmolol and labetalol.
Specific indications

8 Pheochromocytoma and catecholamine excess:

- Pheochromocytoma can cause paroxysmal HTN: pulsatile HA, sweating and palpitation. Highly suggestive of the disease if pt has orthostatic hypotension.

- Catecholamine excess: ingestion of tyramine containing foods with taking monoamine oxidase inhibitors, withdrawal of central acting antihypertensives, illicit drug use.

- Risk of sudden death from arrhythmia and cardiogenic shock.

- Nicardipine, labetalol, nitroprusside. Pure beta blocker is contraindicated. Benzodiazepine is adjuvant therapy in these cases.
Specific indications

9 Stroke:

- HTN during acute stroke could be a physiological response to maintain adequate cerebral perfusion.

- In hemorrhagic stroke: disruption of autoregulatory mechanism of bled area, blood flow and oxygen delivery depending on systemic perfusion pressure.

- Cerebral perfusion pressure = MAP – ICP.

- In ischemic stroke: perfusion pressure distal to obstructed vessel is low and a mechanism of compensatory vasodilatation of these vessels to maintain perfusion.
• Subarachnoid hemorrhage increases risks of intracerebral hemorrhage and hydrocephalus.

• MAP should not be lowered than more than 20-25% of the previous level.

• BP is carefully controlled.

• Nicardipine, fenoldopam, labetalol and clevidipine.
Take home messages

• Distinguishing between hypertensive urgency and emergency is very important for appropriate treatment.
• A complete evaluation of pt with hypertensive crisis is to detect and reverse the crisis and avoid further target organ damage.
• Hypertensive emergency needs to be admitted to ICU for close monitoring of blood pressure and using parenteral antihypertensive drugs.
• Hypertensive urgency can be managed at outpatient setting and oral antihypertensive can be used.
• MAP is not lowered than 20% of initial level in few hours in hypertensive emergency.
• Looking for and correcting triggering factors to improve outcomes.
References


