HTN EMERGENCIES

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References:

- UPTODATE
- ROSEN's Emergency Medicine 10th edition
- TINTINALLI's Emergency medicine 9th edition

• Hypertensive crisis

- An acute elevation of blood pressure : the systolic blood pressure is >180 mm Hg and/or the diastolic blood pressure is >120 mm Hg
- There are two forms of hypertensive crisis.
- Hypertensive emergency is a hypertensive crisis (systolic blood pressure >180 mm Hg and/or diastolic blood pressure >120 mm Hg) with concomitant end-organ damage; the targeted end organs include the brain, heart, aorta, kidneys, or eyes
- Hypertensive urgency is a controversial term , a marked and acutely elevated blood pressure without acute or worsening target organ dysfunction.

- Most hypertensive emergencies occur in patients with chronic HTN , the most important exception is in pregnancy
- Organ system involvement is dominated by vascular injury leading to impaired function of the heart, brain, or kidneys
- Frequently accompanied by an elevated BP, symptoms such as headache, epistaxis, and dizziness are not, evidence of acute TOD
- Current recommendations for patients with hypertensive urgency are : oral antihypertensive therapy and prompt outpatient follow-up
- Gradual blood pressure reduction should occur over days to weeks. There is no clinical benefit of such treatment (parental therapy) and precipitous drops in blood pressure can be harmful.

Blood Pressure Goals

- Use parenteral antihypertensive agents to reduce systolic blood pressure no more than 25% in the first hour; if stable, then reduce to 160/100 mm Hg over the next 2 to 6 hours and then to normal over the following 24 to 48 hours

- For most hypertensive emergencies, mean arterial pressure should be reduced gradually by approximately 10% to 20% in the first hour and by a further 5 to 15 percent over the next 23 hours . This often results in a target blood pressure of <180/120 mmHg for the first hour and <160/110 mmHg for the next 23 hours

- The major exceptions to gradual blood pressure lowering over the first day are:

•Acute phase of an ischemic stroke
•Acute aortic dissection
•Intracerebral hemorrhage

CHEST PAIN AND SEVERE HYPERTENSION

- Acute Aortic Dissection
- Pulmunary Edema
- Acute myocardial infarction

Acute aortic dissection

- Presents with abrupt, sudden onset of pain, usually in the chest, often described as tearing or ripping, and radiating to the interscapular region , Chest pain, back pain
- Unequal blood pressures (>20 mm Hg difference) in upper extremities
- Goal is to reduce shearing force by lowering BP & PR
- Systolic blood pressure between 100- 120 mm Hg and a heart rate ≤60 beats/min, ideally within the first hour of presentation
- Pain control with opioids helps decrease sympathetic tone.

Treatment:

- **Labetalol** : 10-20 mg iv over 2 min (may administer 40-80 mg at 10min interval up to 300 mg total dose) then continius 2mg/min , titrate to response up to 300 mg total dose
- **Esmolol** ; 250-300 mg/kg over 1-3 min followed by 50 microgram/kh/mon over 4 min
  - This regimen can be repeated for 4 bolus doses and to an infusion rate of 300 microgram/kg/min
- **Nicardipin** iv continious infusion (after betablocker) start at rate 5mg/h if target bp not achieved in 5-15min increase by 2.5 mg/h every 5-15min until target bp or maximum dose of 15mg/h is reached
- **Nitroprusside** continiuos infusion (after beta blocker)

- Attention : beta blocker in asthma , COPD is harmfull , use diltiazem instead
- Measure BP in both arms and treat higher BP

Pulmunary Edema

- Shortness of breath
- Most patients have existing poorly controlled hypertension with cardiac remodeling and left ventricular hypertrophy, stiffness, and diastolic dysfunction. With an acute rise in blood pressure there is an increase in afterload and a decrease in venous capacitance
- Interventions that improve forward flow, via afterload reduction, tend to work better than diuresis
- Goal : reduce BP 20-30%

Treatment:

- **Nitroglycerin** :Continiuos infusion :starts 5 microgram/min increase by 5 micrograms/min every 3-5 min to 20microgram/min ,if no response increase 10microgram/min every 3-5 min up to 200 microgram/min
- Attention to avoid in concurrent use of sildenafil,tadalafil(phosphodiestrase –5 inhibitor

- **Clevedipine** infusion tare 1-2 mg/h , maximum dose 32microgram/h , maximum duration 72h
- Attention ; may cause hypotention and reflex tachycardia
- Contra indicated in severe aortic stenosis , egg soy hypersensitivity
- **Nitroprusside** IV continius infusion , 0.3-0.5 microgram/kg/min
- Rate more than 2microgram/kh/min may lead cyanide toxicity
- Attention : is recommended when other agents fail
- Avoid in kidney/ hepatic failure.elevated ICP,AV shunt,
- **Enalaprilate** IV
- **Nicardipine**
- **Nesiritide** IV

Acute myocardial infarction / Acute coronary syndrome

- Chest pain, nausea, vomiting, diaphoresis
- Changes on ECG or elevated levels of cardiac biomarkers
- Avoid ≤25% reduction of MAP

Treatment:

- **Nitroglycerin** :Continiuos infusion :starts 5 microgram/min increase by 5 micrograms/min every 3-5 min to 20microgram/min ,if no response increase 10microgram/min every 3-5 min up to 200 microgram/min

- **Esmolol** ; 250-300 mg/kg over 1-3 min followed by 50 microgram/kh/mon over 4 min

- **Labetalol** : 10-20 mg iv over 2 min (may administer 40-80 mg at 10min interval up to 300 mg total dose) then continius 2mg/min , titrate to response up to 300 mg total dose

Do not give nitrates in patients who have taken phosphodiesterase inhibitors for erectile dysfunction ≤24 h for sildenafil and 48 h for tadalafil

- Do not give β-blockers in CHF, low-output states, or other contraindications to β-blockers

NEUROLOGIC EMERGENCIES

- Hypertensive encephalopathy
- Subarachnoid hemorrhage
- Intracranial hemorrhage
- Acute ischemic stroke

Hypertensive Encephalopathy

- Altered mental status, nausea, vomiting, headache
- May see papilledema or arteriolar hemorrhage or exudates on funduscopic examination, may note cerebral edema with a predilection for the posterior white matter of the brain on MRI
- Decrease MAP 20%–25% in the first hour of presentation; more aggressive lowering may lead to ischemic infarction
- Autoregulation of cerebral perfusion may be significantly impaired, so avoid rapid BP lowering to prevent cerebral hypoperfusion
- Do not give nitroglycerin as it may worsen cerebral autoregulation

Treatment:

- **Labetalol** 10-20 mg iv over 2 min (may administer 40-80 mg at 10min interval up to 300 mg total dose) then continius 2mg/min , titrate to response up to 300 mg total dose

- **Nicardipine** IV continuous infusion : start at rate of 5 milligrams/h. If target BP not achieved in 5–15 min, increase dose by 2.5 milligrams/h every 5–15 min until target pressure or the maximum dose of 15 milligrams/h is reached.

- **Clevedipine** infusion tare 1-2 mg/h , maximum dose 32microgram/h , maximum duration 72h

Intracranial hemorrhage

- Headache, new neurologic deficits
- Abnormal CT of the brain
- If SBP >220 mm Hg, consider aggressive management with IV infusion
- If SBP 150–220 mm Hg, IV boluses of antihypertensive medications should be used to acutely lower SBP to 140 mm Hg
- Drops in SBP <150 mm Hg are not associated with increased morbidity

- Early hemorrhage growth often occurs in first 6 h. Recent data suggest that at this time, aggressive BP control (SBP 130–139 mm Hg) diminishes hematoma growth, morbidity, and mortality

Treatment:

- **Labetalol** IV bolus or continuous infusion
- **Nicardipine** IV continuous infusion
- **Esmolol** IV bolus, then continuous infusio

Subarachnoid hemorrhage

- Headache, focal neurologic deficits
- Abnormal CT of the brain; red blood cells on lumbar puncture
- SBP <160 mm Hg to prevent rebleeding BP parameters have not yet been defined
- Avoid hypotension to preserve cerebral perfusion
- Nimodipine is used to decrease mortality.

Treatment:

- **Nicardipine** IV continuous infusion
- **Labetalol** IV bolus, 10–20 milligrams IV, or continuous infusion
- **Esmolol** IV bolus, then continuous infusion
- **Clevidipine** IV continuous infusion

Acute ischemic stroke

- New neurologic deficits
- Abnormal MRI or CT of the brain

- If fibrinolytic therapy planned, treat if BP remains >185/110 mm Hg after 3 measurements

- Excess BP lowering may worsen ischemia

Treatment:

The following antihypertensive recommendations are for immediate BP control prior to reperfusion

- **Labetalol** : 10–20 milligrams IV over 1–2 min; may repeat once
- **Nicardipine** : 5 milligrams/h IV infusion, titrate up by 2.5 milligrams/h every 5–15 min until desired BP is reached; maximum 15 milligrams/h
- **Clevidipine** : 1–2 milligrams/h IV infusion, double the dose every 2–5 min until desired BP is reached; maximum 21 milligrams/h Nitroprusside may be used if BP is not controlled with above agents or DBP >140 mm Hg

Management of BP during and after reperfusion therapy

- If SBP >180–230 mm Hg or DBP >105–120 mm Hg, then consider
- **Labetalol** 10 milligrams IV bolus followed by continuous IV infusion 2–8 milligrams/min
- **Nicardipine** 5 milligrams/h IV infusion, titrate up by 2.5 milligrams/h every 5–15 min to desired effect; maximum 15 milligrams/h
- **Clevidipine** 1–2 milligrams/h IV infusion; titrate up by doubling the dose every 2–5 min to desired effect; maximum 21 milligrams/h

Acute ischemic stroke, hypertension excludes reperfusion therapy

- Treat if ≥220/120 mm Hg on third of 3 measurements, spaced 15 min apart; BP should be reduced by ~15% in the first 24 h
- Early treatment of hypertension is indicated if required by other comorbid conditions (i.e., acute coronary syndrome, aortic dissection, preeclampsia/eclampsia). Lowering by 15% acutely is probably safe
- Do not lower SBP by >15% in first 24 h
- BP that is lower during the acute ischemic stroke than the premorbid pressure could be considered hypotension

ACUTE RENAL FAILURE, PERIPHERAL EDEMA

- Patients with new-onset renal failure may have peripheral edema, oliguria, loss of appetite, nausea and vomiting, orthostatic changes, or confusion
- Elevated serum creatinine confirms the diagnosis, and urinary sediment is also abnormal
- Reduce BP by no more than 20% acutely
- Fenoldopam, nicardipine, and clevidipine are all suitable for acute hypertension-induced isolated renal failure, because they reduce systemic vascular resistance while preserving renal blood flow.
- Fenoldopam improves natriuresis and creatinine clearance in patients with elevated blood pressure and impaired renal function.

Treatment:

- **Fenoldopam:** IV Continuous infusion: start 0.1–0.3 microgram/kg/min, titrate by 0.05–0.1 microgram/kg/min every 15 min to desired BP.

  Maximum infusion rate: 1.6 microgram/kg/min.

- **Nicardipine ;** IV continuous infusion : start at rate of 5 milligrams/h. If target BP not achieved in 5–15 min, increase dose by 2.5 milligrams/h every 5–15 min until target pressure or the maximum dose of 15 milligrams/h is reached.

- **Clevidipine** IV Continuous infusion: initiate infusion at 1–2 milligrams/h.

  Maximum dose 32 micrograms/h; maximum duration is 72 h.

- Avoid nitroprusside, as it results in cyanide and thiocyanate toxicity

SYMPATHETIC CRISIS

- Anxiety, palpitations, tachycardia, diaphoresis

- Clinical diagnosis in the setting of sympathomimetic drug use (i.e., cocaine or amphetamines) or pheochromocytoma (24-h urine assay for catecholamines and metanephrine or plasma fractionated metanephrines)

- There are four settings in which an excess of catecholamines can result in a hypertensive emergency:

1)Sympathomimetic drugs
2)Pheochromocytoma
3)Autonomic dysfunction
4) An acute catecholaminergic syndrome

- Reduce excessive sympathetic drive and symptomatic relief Aim for SBP <140 mm Hg in the first hour

Treatment:

- **Benzodiazepines** are first-line agents for cocaine-induced hypertension
- **Phentolamine** is first-line therapy for pheochromocytoma
- **Phentolamine** IV or IM , Bolus load: 1–5 milligrams IV; may repeat every 10 min. Continuous infusion: 0.2–0.5 milligram/min.

  *effective for pheochromocytoma and hypercatecholaminergic-induced hypertension.

- Calcium channel blockers in cocaineinduced hypertension are considered third-line agents, after benzodiazepines and nitroglycerin

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In chronic HTN without TOD

1) Acute BP reduction in the ED provides absolutely no benefit

2) Symptoms such as headache or chest pain, and no suspicion of acute TOD, treatment should be directed toward the symptoms, not the BP.

3) Anxiolytic or analgesic medication administration accompanied by resumption of chronic oral antihypertensive therapy is more rational and beneficial than the initiation of short-acting antihypertensive therapy.

CONCLUSION

- Individuals without evidence of acute TOD can be discharged home
- Hypertensive emergency patients warrant admission
- Acute management – Optimal therapy, including the choice of agent and the blood pressure goal, varies according to the specific hypertensive emergency.
- It is generally unwise to lower the blood pressure too quickly or too much, as ischemic damage can occur in vascular beds that have reset their autoregulatory threshold to the higher level of blood pressure.
- For most hypertensive emergencies, mean arterial pressure should be reduced by approximately 10 to 20 percent in the first hour and then gradually during the next 23 hours so that the final pressure is reduced by approximately 25 percent compared with baseline.

THANKS FOR YOUR ATTENTION