HYPERTENSIVE EMERGENCIES

DEFINITIONS

- Systolic blood pressure >180 and diastolic >120mmHg.
- -**HYPERTENSIVE EMERGENCY**: also called hypertensive crisis, is severe hypertension with acute impairment of an organ

-**HYPERTENSIVE URGENCY**: the BP is a potential risk but has not yet caused acute end-organ damage.

ETIOLOGY

- **Essential hypertension** :

Inadequate blood pressure control and noncompliance are common precipitants

- **Secondary hypertension**

Renovascular

Acute glomerulonephritis

Pheochromocytoma

Hyperaldosteronism

Drug-induced hypertension

Coarctation of aorta

Head injuries and CNS trauma

Eclampsia

PATHOPHYSIOLOGY

<u>NORMAL AUTOREGULATION</u>

RISE IN BP

↓

ARTERIAL AND ARTERIOLAR CONSTRICTION

↓

Normal flow.

<u>AUTOREGULATION FAILURE</u>

RISE IN BP

↓

FAILURE OF VASO CONSTRICTION

↓

ENDOTHELIAL DAMAGE

(due to shear stress on the wall)

PATHOPHYSIOLOGY

- BP=PVR*CO(SV*HR)
- Rate at which MAP rises more important than absolute rise.

Acute rise in BP → Failure of vasoconstriction by autoregulation → Endothelial damage

FIBRINOID NECROSIS → Activates coagn and inflammation → Depsn. of proteins/ fibrinogen in vessel wall

- RAAS plays an important role in initiating and perpetuating BP rise by causing vasoconstriction and fluid retention.

- Ischemic stroke
- Hemorrhagic stroke
- Hypertensive encephalopathy

CENTRAL NERVOUS SYSTEM

CARDIOVASCULAR SYSTEM

- Heart failure
- Myocardial infarction
- Aortic dissection

RENAL SYSTEM

- Hematuria
- (RBC) cast formation, and/or proteinuria
- Acute renal failure

EPIDEMIOLOGY

- **In the US:** More than 60 million Americans, about 25-30% of the population, have hypertension. Of these individuals, 70% have mild disease, 20% moderate, and 10% severe hypertension (diastolic BP [DBP] >110 mm Hg). Approximately 1-2% develop a hypertensive emergency with end-organ damage.
- **Mortality/Morbidity:** Morbidity and mortality depend on the extent of end-organ damage on presentation and the degree to which BP is controlled subsequently. BP control may prevent progression to end-organ impairment. I yr mortality in untreated pts. >90%.

EPIDEMIOLOGY

- **Race:** African Americans have a higher incidence of hypertensive emergencies than Caucasians.
- **Sex:** Males are at greater risk of hypertensive emergencies than females.
- **Age:**Most commonly in middle-aged people.Peak age:40-50yrs.

HISTORY

- **Focus on complications :**

  -CNS:headaches,blurred vision,wt. loss,nausea,vomiting,weakness,fatigue,

  confusion and mental status changes.

  -CVS:symptoms of CHF,angina,dissection,SOB

  -Renal:hematuria,oliguria.

PHYSICAL

- Use an approach based on organ systems to identify signs of end-organ damage

  -CNS: focal neuro deficits,seizures,stupor,coma, papilledema, hemorrhages, exudates

CVS:JVD,lung auscultaion for crackles,peripheral edema,extra heart sounds, equal and symmetric BP and pulses bilaterally.

WORK-UP

- CBC,Chemestry
- Urinanalysis:hematuria,proteinuria,RBCs,RBC casts.
- Toxicology,pregnancy,endocrine causes.
- Imaging:Chest X-ray,Head CT,Chest CT
- ECG,cardiac enzymes

TREATMENT

- Weight risks of decreasing end-organ perfusion v.s benefits. Important steps include:

  -Appropriately evaluating patients with an elevated BP

  -Correctly classifying the hypertension

  -Determining aggressiveness of therapy

TREATMENT

- Initial considerations: Place patient who is not in distress in a quiet room and reevaluate after an initial interview.
- Screen for end-organ damage- Patients with end-organ damage usually require admission and rapid lowering of BP using iv meds. Suggested meds depend on the end-organ system damaged.

TREATMENT

- Patients without evidence of end-organ effects may be discharged with follow–up. It is a misconception that a patient should not be discharged from the ER with elevated BP. Giving oral meds such as nifedipine to rapidly lower BP may be dangerous as the BP may have been elevated for sometime and there may be organ hypoperfusion. Acute control has not improved long term mortality and morbidity rates.

CV RAPID BP REDUCTION

- Acute myocardial ischemia:
  IV NTG,b-blockers,ACE inhibitors

- CHF with pulmonary edema:
  IV NTG,furosemide,morphine

- Acute aortic dissection:
  IV nitroprusside and b-blockers
- Ischemic stroke with thrombolytic therapy
- Eclampsia

DRUG	DOSAGE	ONSET/DUR	ADV.EFFE
Nitroprusside	0.25-10mcg/kg/min	Instant/1-2min.	Thiocyanate,cyanide poisoning
Nitroglycerine	5-100mcg/min	1-5min/3-5min	Flushing,headache,methemoglobin
Nicardipine	5-15mg/hr	5-10min/1-4hr	Tachycardia,flushing.avoid-heart failure
Hydralazine	10-20mg	5-15min/3-8hr	Flushing,tachy,avoid-A.diss,MI
Enalapril	10-40mg IM,1.25-5MG1Vq6hr	20-30min/6hr	Hypotension,renal failure,hyperkalemia
Fenoldopam	0.1-0.3mcg/kg/min	5min/10-15min	Flushing,headache,tachy

VASODILATORS

ADRENERGIC INHIBITORS

DRUG	DOSAGE	ONSET/DUR	ADV.EFF
Labetalol (a+b blocker)	20-80mgiv bolus every 10 min,2mg.min iv infusion	5-10min/3-6hrs	Heart block,ortho hypotension.avoid-heart failure,asthma
Esmolol (b-1 selective blocker)	200-500 mcg/kg/min for 4min,then 150-300mcg/kg/min	1-2min/10-20min	Hypotension,avoid-heart failure,asthma
Phentolamine (a1 blocker)	5-15mg iv	1-2min/3-10min	Tachycardia,flushing ,headache

ORAL DRUGS

DRUG	DOSAGE	ONSET/DURATION	ADV. EFF.
CAPTOPRIL (ACE inhibitor)	6.25-25MG q 6hrs.	15-30min/6 hrs.	Hypotension in high renin states
CLONIDINE (a2 agonist-centrally acting)	0.1-0.2 mg hrly, Upto max 0.8mg in 24hrs.	30-60min/6-12hrs.	Sedation,bradycardia,dry mouth

SPECIFIC TREATMENT

- **Hypertensive Encephalopathy**: Goal is to reduce MAP by no more than 25% over 8 hours **Labetalol,fenoldopam** .

- **Intracerebral Hemorrhage**: CPP=MAP-ICP.As ICP rises,MAP must rise for perfusion but this raises risk of bleeding from small arteries and arterioles. **Labetalol,esmolol** agents of choice.

SPECIFIC TREATMENT

- **SAH: Nimodipine** decreases vasospasm that occurs due to chemical irritation of arteries by blood.Labetalol,esmolol agents of choice.Maintain SBP < 160mmHg

- **Acute Ischemic Stroke:** High BP can cause hemorrhagic transformation ,cerebral edema..AHA guidelines:BP be reduced only if SBP>220 .**Labetalol,nicardipine**-agents of choice.For thrombolysis,BP<185/110.

SPECIFIC TREATMENT

- Aortic dissection: Immediate redn. In BP and mainly,shear stress is essential to limit the extension of damage as surgery is being considered.**Labetalol or nitroprusside+b-blocker like propranolol** agents of choice.

- **MI: NTG,b-blockers,ACE inhibitors.**

SPECIFIC TREATMENT

- Acute LVF: usually associated with pulm edema and diastolic/systolic dysfx.

  IV nitroprusside,NTG agents of choice.Titrate until BP controlled and signs of heart failure alleviated.

- Renal insufficiency: is a cause and effect of high BP.Goal is to prevent further renal damage by maintaining adequate blood flow.**Nitroprusside** effective.

- Hypertensive encephalopathy ,ischemic or hemorarhgic stroke sub-arachnoid hemorrhage:

  IV labetalol, nicardipine

- Cocaine, Pheochromocytoma MAO-tyramine interactions with acute hypertension: phentolamine and benzodiazepine.

SUMMARY

- *Accurate BP measurement to improve diagnosis and management of hypertension*
- *More intensive BP control than previously recommended to reduce risk of CVD and mortality*
- *Employ strategies known to improve BP contol*

THANK YOU