Hypertensive Crisis

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Hypertension

- Whether end organ symptoms or damage is present or not - is more important than the absolute degree of BP elevation

- The renin angiotensin system plays an important role in the genesis of hypertension crisis

- Important to assess whether the HT is chronic, acute, or acute on chronic
Definitions

- **SEVERE HYPERTENSION**: defined as 20 mm hg above the 95th centile

- **HYPERTENSIVE CRISIS**: defined as a sudden and abrupt elevation in blood pressure from baseline—generally 50% above normal— or >180/120

- **HYPERTENSIVE EMERGENCY**—a severe symptomatic elevation in BP with evidence of potentially life threatening symptoms or acute target organ damage

- **HYPERTENSIVE URGENCY** is severe hypertension with no target organ damage
Complications of Hypertension: Target-Organ Damage

- TIA, stroke
- Retinopathy
- Peripheral vascular disease
- LVH, CHD, HF
- Renal failure

TIA, transient ischemic attack; LVH, left ventricular hypertrophy; CHD, coronary heart disease; HF, heart failure
Clinical evidence of End organ damage

- **CNS**: altered mental status - lethargy, coma, confusion, seizures, irritability, Facial N palsy, hemiplegia
  
  (Exclude Head trauma or mass lesion)

- **Eye**: Papilloedema, retinal hemorrhages

- **Heart**: LVF, Pulmonary edema, S 3 gallop, new heart murmurs, LV hypertrophy

- **Renal**: Hematuria, proteinuria (Acute GN)
Hypertensive Encephalopathy

Most Emergencies manifest as ENCEPHALOPATHY which is defined by

- Severe BP elevation with cerebral edema
- Neurological symptoms of lethargy, coma and seizures

Caused by cerebro vascular endothelial break down secondary to failure of cerebral autoregulation
Presentation to review

- Etiology
- Evaluation
- Treatment options
Renovascular disease
- UA cath and TE
- Congenital renal anomalies
- Coarctation of Aorta
- Broncho pulmonary dysplasia
- CAH
- Renal vein thrombosis
- Renal parenchymal disease
- Iatrogenic Fluid overload
- ARPKD
- Tumours
- Hypercalemia

Most often secondary to an underlying disease
- Renal parenchymal disease: 60-70%
  - Ac GN, Reflux N
- Renovascular disease: 5-25%
  - Renal A stenosis
- Coarctation of aorta - important cause in infancy
- Essential hypertension - emerging health concern in adolescents - but rarely causes HT crisis
ECF expansion
- Salt intake
  - Increased cardiac output and peripheral resistance
  - High AT-II levels
  - Sympathetic over activity
  - Hyper PTH
  - Uremic toxins
  - Erythropoietin
  - Non-compliance with antihypertensives
  - Endothelial dependent Vd

Acute CNI toxicity
- TMA
- Effect of sirolimus
- High dose steroids
- Fluid overload
- Severe rejection and MA

Renovascular hypertension
**RENAL ARTERY STENOSIS**
- FMD, NF 1, TS
- Takayasu’s arteritis
- Middle aorta syndrome
- William syndrome

HTN post transplant pts

HT in CKD patients and Chronic Dialysis
Examination

- **Anthropometry**
  - weight
  - height
  - BMI
- All peripheral pulses to be palpated
- Four limb BP
- Good general and systemic examination
- Neurocutaneous markers
- Peripheral edema

- **Waist/Hip Ratio**
  - Waist circumference - Abdominal obesity

- **Birth weight**
- **Post natal growth pattern**
- **High salt intake**
**EVALUATION**

**Phase 1**
- CBC
- Urinalysis
- Urine Culture
- Na/K/creat/BUN/
- Ca/Uric acid
- Lipid profile
- Chest Xray
- Renal USG,
- Doppler
- ECHO,ECG

**Phase 2**
- Renal scan with ACE inhibitor
- Renin profile
- MCU, DMSA
- Urine catecholamines
- Plasma and urine steroids
Phase 3
Renal artery imaging
Renal vein renin sampling

CO MORBIDITY
Fasting lipid profile
Fasting Glucose
Drug screen
Polysomnography
Specific Gravity
Proteinuria
  - Prot/Cr
  - UPEP
+ Hematuria + RBC
+ Hematuria − RBC
  - CPK
  - LDH
If

- Hypochloremic Metabolic Alkalosis
  - Adrenal induced Htn
  - RAS
- Renal insufficiency, low plasma proteins/albumin due to renal loss
  - bx
- Low complement
  - PIGN, SLE, MPGN may need to be bx
Renal imaging

- If there is a discrepancy in renal size
  - Angiogram with renal vein renins
    - Avoid use of diuretics, ACE, ARB or Beta blockers 1-2 weeks prior to this setting
  - DMSA scan for scar
  - Thin cut CT
# Renal Vein Renins

<table>
<thead>
<tr>
<th>IVC Level</th>
<th>Right Renal Vein</th>
<th>Left Renal Vein</th>
</tr>
</thead>
<tbody>
<tr>
<td>High IVC</td>
<td>12 ng/ml/hr</td>
<td></td>
</tr>
<tr>
<td>Rt Renal Vein</td>
<td>20 ng/ml/hr</td>
<td>Left Renal Vein</td>
</tr>
<tr>
<td>Low IVC</td>
<td></td>
<td>3 ng/ml/hr</td>
</tr>
</tbody>
</table>
Why not draw?

- **Renins**
  - Random renins have not been found to be diagnostic...
    - effected by volume status, ACE inhibitors, ARBs, Beta blockers

- **Aldosterone**
  - May be helpful if evidence of a hypochloremic metabolic alkalosis
Case 1

- 6 years old male child with SRNS was diagnosed to have hypertension outside, started on Enalapril
  - Had one episode of change in is mental status
  - Intubated due to worsening of sensorium
  - He developed irrelevant speech and unable to bear weight after extubation
  - Referred here for further management.
- In casualty he had one episode of seizure for which he was started on antiepileptic
- EEG done was suggestive of bilateral epileptiform discharges
- MRI Brain with MRA was done
Bilateral asymmetric gyral swelling and hyperintensity and symmetric posterior thalamic hyperintensities
After a month - resolution of findings
DIAGNOSIS ???

PRES

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME
Hypertensive encephalopathy occurs due to:
- Cerebral hyper perfusion
- Endothelial dysfunction
- Microvascular injury
- Cerebral edema

These manifests as PRES in imaging studies.
**PRES**

- Occurs due to vasogenic edema, onset of hypertension is acute

- Often a trigger in other conditions like
  - Treatment with immunosuppressive drugs
  - Vasculitis
  - Renal insufficiency

- Endothelial dysfunction and NO depletion are proposed mechanisms

- Symptoms
  - Altered mental status, Headache, Seizures
Treatment Options for acute HTN

- Angiotensin Receptor Blockade (ARB) - avoid
- Central Action
- Diuretics - avoid
- Vasodilators/alpha blockers
- Beta Blockers - avoid if hyperkalemia
- Calcium Channel blockers
- ACE inhibitors - avoid
Angiotensin Receptor Blockade (ARB)

- Newer generation of class of drugs
- Not a role in the Rx of malignant Hypertension
- Used best in patients with CKD (chronic kidney disease)
- Avoid if CKD/AKI/hyperkalemia
Central Action

- Not a role as a primary agent
- PO/Transdermal Clonidine good option for sedation withdrawal hypertension
- Problem with patch is at the time of hypotension a patch can be forgotten
- If on po Clonidine (data on patch less clear) and drug abruptly stopped, one is at risk for rebound significant hypertension
Diuretics

- Use as an adjunct to other agents only
- Not a role as a primary antihypertensive
- Side effects of electrolyte disturbances, elevated uric acid, lipids
Beta Blockers

- Pure Beta blockers have limited role in hypertensive crisis
- Avoid if hyperkalemia, bradycardia
Beta Blockers

- **Esmolol**-good for tachycardia
  - Ld 100-500 mic/kg then 50-500 mic/kg/min
    - Wiest et al, J Thor Cardiov Sg 1998 4:890

- **Labetalol (Alpha/Beta)**
  - Ld 0.2-1 mg/kg then 0.5 – 2 mg/kg/hr
    - J Peds 1992 120:140

- **Metoprolol infusions**
  - 1-5 mics/kg/min to titrate to effect
    - Peds Nephrol 2017 32:2107-2113
ACE inhibitors

- Should be limited as a primary agent due to the risk of
  - Influencing renin data if measured
  - Risk of AKI, hyperkalemia
  - Avoid in severe coarctation of the aorta or in bilateral renal artery stenosis due to risk of AKI
ACE inhibitors

- The only IV option is enalaprilat
  - 5-10 mic/kg/dose q 4-8 hrs
    - Wells et al, Pediatr 1988 113:403
- Preferred drug in micro embolic states with associated Htn
  - UAC induced
  - Renal vein thrombosis
Vasodilators/alpha blockers

- Nitroprusside
  - Direct arterial vasodilator
  - Use with caution in AKI
    - Cyanide toxicity
  - Replaced by newer generation

- Nitroglycerin
  - Arterial and veno dilator
  - Intravenous or Transdermal (0.5-2 inches)
Vasodilators/alpha blockers

- Fenoldopam
  - Dilator with improvement in renal blood flow
  - 0.1 mic/kg/min to max 5 mic/kg/min
    - Stauser et al, AM J Ther 1999, 6:283
- Use in patients **without** Htn to enhance Renal Blood Flow and UO has not been well studied but may decrease BP to much to offset benefit of RBF preference
Vasodilators/alpha blockers

- Phentolamine
  - Used exclusively in Rx of Pheochromocytoma
  - 0.1-0.2 mg/kg bolus and q 2-4 hrs
  - Use preop to effect systemic symptoms of flushing, significant BP swings

- Bholah R et al, Review of Pediatric Pheochromocytoma and Paraganglioma.
Calcium Channel Blockers

- Primary drug used for Malignant Htn
- Safe as an intravenous agent
- Class of drug of choice in patients on calcinurin inhibitors (tacro, csa, rapamycin)
- Nifedipine SL
- Nicardipine IV
SL Nifedipine

- Immediately effective but watch for rapid and worsening rebound
- No longer used in adult due to sudden death
- Leonard et al Ped Emg Med 2001 17:435
  - 2 children given SL due to Malignant Htn in the ED..both improved but within 30 mins both rebound and had strokes!
Nicardipine

- Immediate on set of action
- Easy to titrate at bedside
  - 0.5-5 mic/kg/min as a continuous gtt
  - J Pediatr 2001 139:38
- Perhaps a role as an intermittent bolus
  - 10-20 mic/kg/dose IV/5 mins every 1-3 hrs
Algorithm for Treatment of Hypertensive Crisis

**Intravenous Drugs**

- **Nicardipine**
- **Labetalol**
  - Avoid in hyperkalemia and acute heart failure
- **Sodium Nitroprusside**
  - Monitor thiocyanate toxicity
- **Fenoldopam**
- **Hydralazine**
- **Enalaprilat**
  - Avoid in neonates and renal failure
- **Clevidipine**

**Hypertensive crisis**

- **Hypertensive Emergency**
  - **Nicardipine**
  - **Labetalol**
  - **Sodium Nitroprusside**
    - Monitor thiocyanate toxicity
  - **Fenoldopam**
  - **Hydralazine**
  - **Enalaprilat**
    - Avoid in neonates and renal failure
  - **Clevidipine**

- **Hypertensive Urgency**
  - **Isradipine / Nifedipine**
    - **Clonidine**
    - **Minoxidil**
    - **Furosemide**

**Oral Drugs**

- **Isradipine / Nifedipine**
  - **Clonidine**
  - **Minoxidil**
  - **Furosemide**

**Hypertensive Crisis in children**

Ped Nephrology

Jayanthi Chander 2012
Treatment Strategy

- Like antibiotics decision making learn one from each class and stay with it
- If the goal is to get the patient improved but to get them to the ward
  - Begin oral, Transdermal therapy soon after beginning IV therapy if the patient can tolerate it
Drugs that are easy to use

- Consider CCB or vasodilators as the first line drugs for Htn for these have low risk profiles, less influence on interpretation of other data
Thank you

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  - Contains all talks given at the PCRRRT meetings from 2000 to the most recent in 2017 in Orlando
Signs and symptoms of hypertensive crisis includes

- seizures
- Heart failure
- Pulmonary edema
- All of the above
With a child has a bilateral renal artery stenosis with renal insufficiency then the intravenous drug to avoid is

1. IV Nicardipine
2. IV enalaprilat
3. IV atentolol
4. IV nitroglycerine
If a child has a seizure from a hypertensive crisis then the priority (of the first thing to do) is to

- Check a head CT scan
- Treat with seizure medications
- Bring the blood pressure down
- All of the above