HYPERTENSION IN PAEDIATRICS

Priya Gajjar

Paediatric Nephrology

RED CROSS CHILDREN’S HOSPITAL
Case Study

1 yr 10 mo girl

- Presented to local Level 2 Hospital
  - 1st episode generalized tonic-clonic seizures
  - Concerns about her vision
Clinical Findings

- Depressed level of consciousness
  - No focal signs
  - No meningism

- Pale

- Normal glucose

- Tachycardiac
  - All pulses palpable

- Congestive cardiac failure.
  - displaced apex
  - enlarged liver
  - gallop

- Anthropometry:
  - Wt = 10.7 kg (25th centile)
  - Ht = 85 cm (50th centile)
  - COH = 47 cm (25th centile)
Perinatal / Birth history:
- HIV + mother (not on HAART - no perinatal program)
- NVD at term, BW > 3 kg, good Apgars
- Well neonate, no interventions
- HIV PCR negative at 3 months of age

Immunizations:
- UTD
- BCG at birth

Growth
- Growing along the 50th centile and fell to the 25th centile two months ago
No history of toxin ingestion

Significant Previous Medical history
- TB treatment at local clinic 2 months prior
  - Coughing > 1 month
  - LOW (wt ↓50th to 10th centile)
  - Household TB contact
  - Reactive Tine
Investigations

- Normal electrolytes
  - Glucose
  - Calcium
  - Urea and Creatinine
- Normal CSF
- FBC:
  - WCC 10.6  Hb 8.6  MCV 64.8
  - Plt 807
- CRP : 12.3
- ESR : 35

- CT brain
  - Cortical Atrophy
And **finally** a **BP** was done….she was found to be **HYPERTENSIVE IN ALL 4 LIMBS**

- Initial values of 190-200/130-140
Working diagnosis …

- HYPERTENSIVE ENCEPHALOPATHY with seizures and depressed level of consciousness

- CONGESTIVE CARDIAC FAILURE secondary to LONGSTANDING HYPERTENSION

- Possible RENO-VASCULAR cause for her hypertension
Further investigations prior to referral

- **Ultrasound Abdomen**:  
  - Rt kidney 6.5 cm and Lt kidney 8.5 cm.  
  - Ureters and bladder normal.  
  - Nil other intra–abdominal abnormalities found

- **CT Abdomen**:  
  - Rt kidney smaller than Lt  
  - Rt renal artery diameter (3.4 mm) is smaller than Lt (3.8 mm).  
  - No aneurysms  
  - Aorta normal in size.
- Stabilised
- Antihypertensives commenced
- Transferred to RXH
  - Clinically significantly improved
Investigations at RXH

- Doppler Ultrasound KUB
  - Rt kidney 60mm, Lt kidney 82mm
  - Rt renal artery stenosis
    - Tardus parvus doppler waveform with decreased peak systolic velocity
Mag 3
Essentially a solitary kidney
Rt kidney functioning at 10%
Lt kidney 90%
Echocardiogram

- Dilated cardiomyopathy
- Ejection Fraction 26%
- ▲ Rt Renal Artery Stenosis
  - ▲ ▲ ? Takayasu’s Arteritis
    - Very young presentation
    - ? In the setting of possible active TB
    - Very focal disease
  - ▲ ▲ Congenital
    - Fibromuscular dysplasia
Treatment

- To treat with anti-inflammatories
  - ESR ranged from 19 to 46
  - Already on TB therapy
    - Needed an angiogram; concerns re acutely inflamed vessels
      - Pulsed with Medrol
      - One dose IVI Cyclophosphamide
      - Methotrexate and Prednisone

- Antihypertensives
  - Amlodipine
  - Lasix
  - Spironolactone
  - Atenolol
Angiogram

- Severe Rt renal artery stenosis, tapering to 1mm
  - Normal Aortic arch and head and neck vessels
  - Normal Abdominal Aorta, SMA and iliacs
Mx
- Underwent a laparoscopic Rt Nephrectomy
  - Good post-op recovery

Progress
- Weaned off all her antihypertensives
- Kept on small dose Enalapril for the cardiomyopathy
Paediatric Hypertension
Considereable advances have been made in the detection, evaluation and management of hypertension, as well as in obesity-related hypertension in children and adolescents.

- Publication of national norms
- BP measurement in the routine paediatric examination
  - Identify and treat secondary forms of hypertension
  - Detection of mild elevations in BP representing early onset of essential hypertension
Rising prevalence of Hypertension in the paediatric population is related to the epidemic of childhood obesity
- 25% of children in US are overweight
- 11% are obese

Childhood obesity contributes to the development of adult obesity, with its subsequent cardiovascular events
- 70% of obese adolescents become obese adults
  - Pediatrics, 2004;114;555-576
Normograms

- The revised BP tables include the 50th, 90th, 95th and 99th percentiles for gender, age and height
DEFINITIONS

- Adults: BP > 140/90 → **120/80mmHg**

- **Systolic Blood Pressure and/or a diastolic BP that is on repeated measurements >/=95th%**

- **Prehypertension** replaces *High normal BP*
  - Syst or diast BP between 90th - 95th centiles
    - Indication for lifestyle modification and continued monitoring
    - Adults and adolescents with BP > 120/80 should be considered prehypertensive
      - Affects clinical management with additional resources required at all levels of health care
- Systolic BP > 100 + (3 x pat’s age in yrs)
- Diastolic BP > 70 + (1.5 x pat’s age in yrs)
Staging of Blood Pressure

- Management plan for evaluation and treatment
- Difference between 95\(^{th}\) and 99\(^{th}\) percentile BP is 7 to 10mmHg.
  - **Stage 1 Hypertension**
    - Syst or diast BP between 95\(^{th}\) – to 5mmHg above the 99\(^{th}\) centile
    - Evaluate before initiating treatment, unless symptomatic
  
  - **Stage 2 Hypertension**
    - Syst or diast BP >5mmHg above the 99\(^{th}\) centile
    - Prompt evaluation and treatment
    - Symptomatic require immediate consultation with experts
Measurement of Blood Pressure

- **Appropriate cuff**
  - Bladder width at least 40% of the arm circumference at a point midway between the olecranon and the acromion
  - Bladder length should cover 80 to 100% of the circumference of the arm

- **Manometers**
  - BP tables are based on auscultatory measurements
  - Aneroid

- **Oscillometric devices**
  - Measures mean BP, then calculates the syst and diast BP
24-hour Ambulatory BP

- **BP Load**
  - Percentage of measurements above the 95th percentile
    - Over 50%, closely related to end organ damage
- **Physiological fall in nocturnal BP and rise with wakening**
  - Non-dipping status and nocturnal hypertension is described in children with secondary hypertension as opposed to patients with essential hypertension
- **White-coat hypertension:**
  - BP>95th %, when measured in clinic, whereas the patient’s average BP is <90th % outside of a clinical setting.
Essential Hypertension

- Usually mild or stage 1 hypertension

- Positive family history of hypertension or of cardiovascular disease.
  - Familial component with genetic factors contributing 40-50%
  - Likely to be related to multiple genes

- Frequently overweight
  - Prevalence of hypertension increases progressively with increase in BMI
  - Hypertension is detectable in 30% of overweight children

- Clustering of other cardiovascular risk factors
  - Hyperlipidaemia, truncal obesity, insulin resistance
Children with Essential Hypertension had lower birth weights

- BW <2.5kg, 38% were hypertensive
  - Elevated Uric acid levels
    - Inhibit proliferation of glomerular endothelial cells
    - Rx Allopurinol?
    - Predictor of cardiovascular risks in adults
# Causes of Secondary Hypertension

<table>
<thead>
<tr>
<th>Renal Parenchymal</th>
<th>Dysplasia, chronic GN, Reflux Nephropathy, Wilm’s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reno-vascular</td>
<td>Takayasu’s Arteritis</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Co-arctation of Aorta</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Phaeochromocytoma, Cushings, Conn’s</td>
</tr>
<tr>
<td>Central</td>
<td>Tumours, Benign raised intracranial hypertension</td>
</tr>
</tbody>
</table>
Renovascular Hypertension

- Potentially amenable to curative treatment
- Accounts for 5-10% of all childhood hypertension.
- Angiography still the gold standard imaging modality
- In general ACEI are contraindicated in patients suspected of having renovascular hypertension
Causes of Renovascular Hypertension

- Fibromuscular Dysplasia
- Syndromic
  - Neurofibromatosis type 1
  - Tuberous Sclerosis
  - William’s Syndrome
- Vasculitic
  - Takayasu’s
  - Polyarteritis Nodosa
- Extrinsic Compression
  - Neuroblastoma
  - Wilm’s
- Others
  - Umbilical vein catheterisation
Causes of Sustained Hypertension *(PD Thomson)*

<table>
<thead>
<tr>
<th>Cause</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic Glomerulonephritis</td>
<td>30%</td>
</tr>
<tr>
<td>Chronic Pyelonephritis</td>
<td>20%</td>
</tr>
<tr>
<td>Coarctation of Aorta</td>
<td>10%</td>
</tr>
<tr>
<td>Cystic Disease of Kidney</td>
<td>10%</td>
</tr>
<tr>
<td>Haemolytic Uraemic Syndrome (HUS)</td>
<td>8%</td>
</tr>
<tr>
<td>Renovascular</td>
<td>8%</td>
</tr>
<tr>
<td>Hypoplastic/Dysplastic Kidneys</td>
<td>4%</td>
</tr>
<tr>
<td>Essential</td>
<td>4%</td>
</tr>
<tr>
<td>Others – phaeo, pap nec, Wilms</td>
<td>6%</td>
</tr>
</tbody>
</table>
## History in paediatric hypertension - 1

<table>
<thead>
<tr>
<th>Family history hpt, pre-eclampsia, renal dx, tumours</th>
<th>Essential hpt, inherited dx, familial phaeochromocytoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonatal hx</td>
<td>Birth weight, Use of umbilical catheters</td>
</tr>
<tr>
<td>Dietary hx</td>
<td>Sodium &amp; calorie intake</td>
</tr>
<tr>
<td>Headaches, dizziness, epistaxis</td>
<td>Non-specific symptoms</td>
</tr>
<tr>
<td>Abdominal pain, dysuria, frequency, nocturia, enuresis</td>
<td>Underlying renal disease</td>
</tr>
<tr>
<td>Joint pains/swelling, rash facial/peripheral oedema,</td>
<td>Collagen vascular disease or other nephritis</td>
</tr>
<tr>
<td>Wt loss, FTT, excessive sweating, flushing, pallor, fever</td>
<td>Phaeochromocytoma</td>
</tr>
<tr>
<td>Muscle cramps, weakness, constipation</td>
<td>Hyperaldosteronism with hypokalaemia</td>
</tr>
<tr>
<td>Drug History</td>
<td>Steroids, NSAIDs, Ritalin</td>
</tr>
</tbody>
</table>
DRUGS CAUSING HYPERTENSION

- Adrenaline
- Amphetamines
- Caffeine
- Carbamazepine
- Cocaine
- Cyclosporin
- Erythropoietin
- Ketoconazole
- Methylphenidate (Ritalin)
- Metoclopramide
- NSAID’s
- OCP’s
- Promethazine
- Steroids
- Terbutaline
Clinical Assessment

- General
  - Dysmorphism, vitals, wt & ht, BMI
  - Skin, joints
- Renal
- Endocrine
- Cardiac
- Central
Diagnostic - Step 1

- **Renal**
  - Urine dipstix, MC&S, Protein: creatinine ratio
  - U&E, Creatinine, Ca, and Po4
  - Ultrasound of kidneys
    - Sizes
    - Dopplers of renal vessels

- **Cardiac**
  - CXR, ECG,
  - Echo, to exclude Coarctation

- **Inflammation**
  - Mantoux
  - FBC & ESR
Diagnostic – step 2
(depending on findings in step 1)

- RENAL:
  - Glomerulonephritis
    - ASOT, Anti-DNAse B,
    - C3/C4
    - ANF, ANCA’s (if nephritis/vasculitis suspected)
  - Reflux Nephropathy
    - DMSA scan
    - MCUG (if unexplained renal scarring) Plasma Renin
  - Renovascular Disease
    - Plasma Aldosterone and renin
    - Mag 3 / Captopril renogram
    - CT scan / MRA
    - Angiography with renal vein renin sampling
Diagnostic – step 2
(depending on findings in step 1)

- **ENDOCRINE:**
  - Phaeochromocytoma
    - Urinary Catecholamine metabolites (VMA/NMA – either as spot or 24hr collections)
    - Adrenal/abdominal U/S
    - MIBG scan
    - CT/MRI
  - Others
    - Cortisol (early morning)
    - 24hr Urine Steroid profile
    - Renin
    - U/S / CT of adrenals
Diagnostic – step 2
(depending on findings in step 1)

- CENTRAL:
  - CT Scan head
# End Organ Damage

<table>
<thead>
<tr>
<th>Cardiac</th>
<th>CXR</th>
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<tbody>
<tr>
<td></td>
<td>ECG</td>
</tr>
<tr>
<td></td>
<td>Echo</td>
</tr>
<tr>
<td>Eyes</td>
<td>Ophthalmology review</td>
</tr>
<tr>
<td>Renal</td>
<td>Urine dipstix</td>
</tr>
<tr>
<td></td>
<td>Urea &amp; Creatinine</td>
</tr>
</tbody>
</table>

- Echocardiography is recommended as a primary tool for evaluating patients for target-organ abnormalities by assessing the presence or absence of LVH.
- If end-organ damage is discovered, hypertension should be aggressively managed.
Evaluation of Co-morbidities

- Fasting lipid panel
- Fasting glucose; HbA1C
Newer Imaging Techniques

- **CT Angiogram**
  - Disease of aorta

- **MRI/Angiogram**
  - Aorta and main renal arteries

- **Conventional Angiogram**
  - Most accurate
  - General anaesthetic
Mx of Acute Hypertension

- **Hypertensive Crisis**
  - Medical emergency with end organ decompensation
    - ICU or High Care Setting
    - Frequent BP monitoring and Neuro observations
    - Reduce BP slowly
      - 1/3 of total desired reduction in 1st 12 hrs
      - Next 1/3 over 12-36hrs
      - Last 1/3 over 36-72hrs

- **Rx**
  - IVI Sodium Nitroprusside 0.5 – 8ug/kg/min
  - IVI Labetalol 0.25 – 4mg/kg/hour
    - Caution in heart failure and Asthma
  - IVI Frusemide 1-5mg/kg/dose slowly
    - If fluid overloaded
Mx of Acute Hypertension

- **Severe Hypertension**
  - ~ 20mmHg above the 95th percentile
  - Rx oral agents
    - Amlodipine newer agent
      - 0.2mg/kg/dose po every 6-12hours
    - Nifedipine 0.15-0.3mg/kg/dose 4-6hrly *Nourse SAJCH 2007*
    - Oral Hydralazine 0.4-1mg/kg/dose(or ivi)
    - Lasix 1-4mg/kg/day (if fluid overloaded)

- *The shorter the duration of being hypertensive, the quicker the BP can be brought down*
Chronic Hypertension

- **A – ACEI**
  - Captopril
  - Enalapril/Ramipril/Perindopril

- **B – B Blocker**
  - Propanolol
  - Atenolol/Metoprolol
  - Labetalol – Combination Alpha and B Blocker

- **C – Calcium Channel Blocker**
  - Nifedipine
  - Amlodipine
Chronic Hypertension

- **D – Diuretic**
  - Lasix
  - Spironolactone

- **E – Everything else**
  - Alpha blocker
    - Prazosin/Doxazosin
  - Direct arteriolar dilatation
    - Hydralazine, Minoxidil
  - Central acting agents
    - Clonidine, Methyldopa
Therapeutic Lifestyle Changes

- Weight reduction
- Regular physical activity and restriction of sedentary activity
- Family-based intervention improves success
Angioplasty

- Balloon dilatation
- Stents
# Percutaneous Transluminal Angioplasty (PTA)

<table>
<thead>
<tr>
<th>Description</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTA for control of Hypertension (12 patients)</td>
<td>14 cases</td>
</tr>
<tr>
<td>Aortic dilatations</td>
<td>4/4 successful</td>
</tr>
<tr>
<td>Renal Vessel dilatation</td>
<td>6/10 successful</td>
</tr>
</tbody>
</table>
Surgical

- Nephrectomy
- Auto-transplant
- Re-vascularisation
- Graft bypass
Take Home Message

- Hypertension is increasing in prevalence in children, likely related to the epidemic of childhood obesity.

- Proper BP measurement is critical in establishing the diagnosis of hypertension.

- Secondary causes of hypertension are more common in children than in adults and require more extensive work-up.
Overweight children are more likely to have essential hypertension so evaluation can be limited in these patients.

Childhood hypertension tracks into adulthood. The impact of hypertension on the risk of cardiovascular disease has been well established, thus the general paediatrician must be comfortable at evaluating and treating elevated blood pressures.

Co-morbidities, if present should be promptly treated.

If end-organ damage is discovered, hypertension should be aggressively managed.