HTN – Case studies

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Case #1

- 45 yo male presents for evaluation of HTN. BP screening last week 175/95. Denies any Sx but has inactive lifestyle.
- PMHx – peptic ulcer, s/p choly
- FHx – HTN

Case #1

- PE – BP R 180/97, L 185/90
- No carotid bruits
- Optic fundi benign
- CV – Increased S2, no murmur, PMI normal location but sustained, S4 gallop
- Abd – no bruits
- Ext – nl

Case #1

- Lab:
  - EKG – NSR with LVH by voltage
  - CXR – normal
  - CBC, Chem panel, UA all normal
- Any more tests before starting treatment?

Case #1

- What is significance of loud S2, sustained PMI, S4 gallop?
- Loud S2 @ 2nd RUSB is from systemic or pulmonary HTN
- Sustained PMI suggest hypertrophy from chronic increased pressure load (HTN, aortic stenosis, hypertrophic cardiomyopathy)
- S4 gallop indicates diastolic dysfunction.

Point of maximal impulse (PMI)

- The heart can either dilate or hypertrophy in response to stress.
- Pressure stress causes hypertrophy
  - HTN, Ao Stenosis
  - PMI will be stronger and more sustained but in the normal location
- Volume stress causes dilation
  - CAD, dilated cardiomyopathy, MR, AoR
  - PMI will be laterally displaced

Heart Sounds

- Diaphragm for high frequency, Bell (applied gently) for low frequency.
- S1 – mitral/tricuspid closure, best heard @ LLSB
  - Soft S1 – systolic dysfunction, MR, TR
- S2 – Ao/Pulmonic closure, best heard @ RUSB and LUSB
  - Loud S2 either systemic or pulmonary HTN

Gallops

- S3 and S4 best (and usually only) heard at the PMI.
- Low frequency, apply bell gently @ PMI
- S3 is systolic dysfunction. Can distinguish an S3 from a split S2 (i.e. LBBB) by putting firm pressure on the bell, thus turning it into a diaphragm and the S3 will disappear.
- S4 is from diastolic dysfunction (LVH, AoS)

Murmurs

- Harsh – High pressure to high pressure
  - Ao Stenosis, Pulm Stenosis
- Blowing – High pressure to low pressure
  - MR, TR, AoR, VSD
- Rumbling – low frequency, low pressure to low pressure
  - Mitral Stenosis
- Where is it best heard?
- Systolic vs. diastolic
- By quality, location and timing can figure out source of most murmurs

Case #1

- What about EKG?
- LVH on EKG confirms the clinical impression that pt already has end-organ damage. Sequence of EKG changes in HTN is:
  - LVH by voltage
  - LAA
  - LAD
  - LV "strain" pattern, (lateral ST-T changes)
- Echo would quantify better but probably won't effect how to treat pt.

Case #1

- This pt has stage 2 HTN so:
- Treat with 2 drugs. In absence of other compelling indications, HCTZ and ACEI would be my choice, though in a young pt with LVH a B-blocker would be good too. ARB and CCB would be ok 2nd drugs too.
- Don't forget to stratify for CV risk (check lipids, FHx CV events, smoking) and address those too
- Don't forget lifestyle modification: he is a self admitted couch potato.

Case #2

- 75 yo pt. with Hx prior CVA. Now has arthritis and mild orthostatic dizziness.
- PMHx – old MI, BPH (prostatism)
- PE: BP both arms 210/80
- Carotids – brisk, R systolic bruit
- Heart – nl S1, S2, grade 2/6 early peaking harsh SEM @ 2nd RICS
- Pulses – decreased w/o bruits

Case #2

Lab

- CXR – mild cardiomegaly, no CHF
- EKG – NSR w/ NSST-T changes
- Creatinine 2.2
- K+ 4.2
- Hgb 11

Case #2

- Should we treat the BP?
- This elderly pt has isolated systolic HTN (ISH) and requires lifestyle and drug therapy.

Case #2

- What is the Rx for carotid bruit?
- Has had prior CVA.
- If the bruit relates to the area of the prior stroke
- and
- there is little residual neurological deficit
- and
- the pt. is a good surgical candidate
- Then get carotid doppler as if there is a significant stenosis then surgery offers benefit.

Case #2

- What is significance of the murmur?
- The heart murmur is aortic stenosis. The length of the murmur relates to the severity. A short murmur is not significant; either mild AS or Ao sclerosis.
- Sx of significant AS include chest pain, SOB, syncope with exertion and sudden death.

Case #2

- Aortic stenosis is the most important murmur not to miss as patient is at high risk of sudden cardiac death if severe.
- Length of murmur is what matters most
  - If only in early systole, then mild
  - If holosystolic, then moderate
  - If S2 is obscured by the murmur, then this is severe and pt. is at risk for SCD.

Case #2

- What drug therapy would you Rx this pt?
- Therapy includes lifestyle modification and drug therapy. Look for compelling indications in starting medical therapy.
- Start low and go slow.
- In ISH thiazides, amlodipine, ACEI, nitrates or B-blocker are helpful.
- The MI and vascular disease favor B-blocker or ACEI.
- An alpha blocker may help BPH but might worsen the orthstatic hypotension.

Case #3

- 59 yo presents with palpitation associated with apprehension. Denies CP, CHF, use of stimulant drugs.
- ROS – intermittent mild abdominal pain, orthostatic dizziness, flushing during micturition.
- PMHx – thyroid nodule, difficulty with anesthesia

Case #3

- BP 135/85
- Carotids normal, no bruits
- Thyroid small non-tender nodule
- Chest clear
- Heart normal S1, S2 w/o murmur
- Abdomen normal
- Extremities normal

Case #3

- EKG normal
- CXR normal
- Chem panel, CBC normal
- What other test would you do?
- Monitor (Holter) NSR with a bouts of sinus tachycardia not associated with activity

Case #3

- What is differential?
- Further evaluation?

Case #3

- Pheochromocytoma
  - 0.3% of HTN in academic centers
  - Tumor secretes usually catecholamine (norepinephrine) but may also excrete somatostatins, ACTH or dopamine
  - Sx vary depending on what is secreted
  - Rule of 10's: 10% bilateral, 10% extra-adrenal, 10% malignant, 10% familial, 10% with normal BP
  - Asso. w/ MEN 2a, MEN 2b, neurofibromatosis, von Hippel-Lindau

Case #3

- Classic triad of HA, sweating, tachycardia
- HTN sustained in only 50%. Paroxysmal in 40%. Normotensive in 10%.
- Hyperglycemia
- Paradoxical response to B-blockers
- Labile BP during anesthesia
- Other Sx: anxiety, CP, abdominal pain, weight loss, weakness, Sx asso. w/ micturition or valsalva.

Case #3

- Diagnosis – 24 hr urine for VMA, metanephrines, catacholamines (need to exclude FP results)
- If available, it is easier to directly measure plasma metanephine, catacholamine
- Provocative testing: Glucagon trial, Clonidine suppression test (usually not needed anymore with MRI or CT imaging

Case #3

- Imaging
  - MRI
  - Abd. CT
  - MIBG scan (lights up chromafin tissue)
  - Octreotide scan

Case #3

- Medical therapy to stabilize if severe HTN
  - Alpha blocker, then B-blocker
  - IV phentolamine + nitroprusside
  - Labetolol is ok (combined alpha/beta blocker)

Surgical – remove tumors with Sx or >5cm if aSx. Should be done at experienced center.

Case #4

- 62 yo with stable angina and paroxysmal tachycardia. Pt. is active and denies CHF.
- PMHx – HTN, depression, CKD

Case #4

- BP 168/95
- Carotids normal
- Chest clear
- Heart regular, nl S1, increased S2, grade 2/6 high frequency holosystolic murmur @ apex to axilla
- Abd normal
- Bilat femoral bruits

Case #4

- EKG – NSR w/ RBBB
- CXR – normal
- Creatinine 2.2, K+ 3.2, Mg++ 1.5

Case #4

- Discuss significance of loud S2 & murmur
- The loud S2 is either from systemic or pulmonary HTN.
- The murmur is mitral regurg. (MR)
- Severity of MR is best detected by echo as physical findings are not very helpful.

Case #4

- Suggest medical therapy for HTN
- Medical therapy could include B-blocker, verapamil or diltiazem. All would cover HTN, angina and SVT. B-blocker has best survival benefit but may worsen depression (especially if fat soluble)
- If SA or AV node disease then a peripheral CCB would be good choice (nifedipine, amlodipine, anything ending in -pine)

Case #4

- How to treat the ankle edema he gets from his CCB?
- Add ACEI. Much more effective than a diuretic for CCB induced edema.

Case #5

- 55 yo pt w/ longstanding HTN. Has been well controlled on atenolol and hydralazine. Recent BPs are much higher. Pt is compliant and no new meds or drug use.
- PMHx – angina, claudication in both thighs at 3 blocks, old CVA w/o residual.

Case #5

- PE – BP 175/100, otherwise PE is unchanged from last year
- EKG – NSR w/ LVH
- CXR – heart enlarged but stable, no HF
- Creatinine now 2.5 (6 months previously it was 1.4), K+ 3.2

Case #5

- What are causes of refractory HTN?
- How to evaluate?
- Rising creatinine with hypokalemia is a red flag for RAS.
- This pt. was not on ACEI but in a pt who is, a rising creatinine is a warning for RAS and ACEI should be stopped until evaluation is completed. Bilateral severe RAS is a contraindication for ACEI

Case #5

- Renal artery stenosis
- 2-4% of HTN
  - If under 30 most likely fibromuscular dysplasia (younger women w/ webs in distal RA), this is 10% of cases
  - 90% of cases in pts over 55 due to atherosclerotic disease in proximal renal artery

Case #5

- Renal artery stenosis (RAS) presents w/:
  - Accelerated HTN
  - Resistant HTN
  - Renal dysfunction 2nd to hyperaldosteronism
  - "Flash" pulmonary edema (this is either acute MI or RAS)
  - Often asymptomatic

Case #5

- Clinical findings:
  - Hypokalemia (especially with increasing creatinine – RAS is high on DDx)
  - PVD
  - Renal insufficiency in 15%
  - Disparate renal size
  - Epigastric bruit 50%

Case #5

- Peripheral renin level
  - Low level in untreated HTN excludes renovascular HTN
  - High level not helpful
- How to prove Dx?

Case #5

- Renal artery duplex US
  - Good for anatomy and flow but fails to visualize in 20% due to obesity
- MRA – good but often not available and $$
- Spiral CT – shows anatomy but not flow
- Angiogram – gold standard but invasive

Case #5

- Treatment:
  - Medical: ACEI, ARB, B-blocker, Clonidine, Aldomet – all will lower renin levels
  - If Bilateral severe RAS then ACEI are contraindicated. If creatinine rises >20% on ACEI then you need to look for RAS.
  - Surgical: if HTN poorly controlled, intolerant of meds, worsening renal function
    - Angioplasty + stent (50% recurrance in 2 years)
    - Open surgery if all else fails

Case #6

- 50 yo presents with severe HA and blurry vision. He is slightly confused, but has no speech or motor defect, CHF or angina.
- PMHx – longstanding HTN

Case #6

- BP 230/130 in both arms
- Pulse 110
- Fundi – bilat blurred optic discs
- Heart S4 noted, 3/6 blowing diastolic murmur at LLSB
- Abdomen – benign
- Neuro – non-focal

Case #6

- EKG – sinus tach
- CXR – normal
- Creatinine 2.0
- K+ 3.8

Case #6

- Explain the physical findings
- The pt has hypertensive encephalopathy. He has severe systolic and diastolic HTN associated with non-focal neurological defects.
- Blurred optic discs should be seen, though sometimes are difficult to visualize.
- S4 gallop shows diastolic dysfunction (HTN, Aortic stenosis, hypertrophic cardiomyopathy.
- The murmur is Ao regurg – related to severe HTN with or without Ao valve disease.

Case #6

- What is differential diagnosis?
- Hypertensive encephalopathy
- Ischemic stroke
- Hemorrhagic stroke
- Aortic dissection
- Withdrawl HTN
- Substance abuse HTN

Case #6

- Evaluation
  - Neuroimaging (CT or MRI)
  - BP all extremities
  - Toxicology screen

Case #6

- What is the initial therapy?
- Acute therapy
  - Lower MAP by 20%. Want to avoid big drop in BP just as for acute CVA.
  - Labetolol drip is good but B-blocker can provoke asthma.
  - Nitroprusside drip is effective but metabolized to cyanide so can't use for long or in renal insufficiency
  - Diltiazem drip is another alternative

Case #7

- 42 yo man presents with severe HTN. He is alert and denies head trauma. He has a Hx of HTN and drinks excessive EtOH.
- Meds
  - atenolol 100mg qd
  - HCTZ 25mg qd
- Last week he stopped his meds suddenly.

Case #7

- BP 210/125 in both arms
- Pulse 130 bpm
- Optic fundi normal
- Carotids hyperdynamic
- Chest bilateral rhonchi
- Heart loud S1, S2, 3/6 diastolic murmur at LSB
- Abd. – distended w/ hepatomegaly
- Extremities normal

Case #7

- EKG sinus tach with LVH
- CXR normal

Case #7

- Discuss physical findings.
- Sinus tach with loud S1, S2 c/w hyperdynamic state.
- The murmur is Aortic insufficiency c/w severe HTN with or w/o Ao valve disease.
- Liver enlargement c/w right heart failure or alcoholic liver disease.

Case #7

- What is DDx?
- Pt has severe HTN with h/o chronic HTN. He is anxious w/o other neuro defects. The could be hypertensive encephalopathy but more likely substance abuse HTN or withdrawl HTN.

Case #7

- Which meds should be avoided in this type of patient?
- Beta blockers and clonidine can cause severe rebound HTN and tachycardia when suddenly stopped. Beta blockers may also cause rebound MI.
- How would you treat this patient?
- Restart beta-blocker and then taper off gradually.

Case #8

- 35 yo with viral congestive cardiomyopathy. SOB w/ minimal exertion, 4 pillow orthopnea and has PND (paroxysmal nocturnal dyspnea)

Case #8

- BP 170/70
- Pulse 110
- Chest – rales 1/3 up the posterior chest
- Heart – S1 soft @LLSB, S2 loud @LUSB, S3 noted, PMI large, displaced to AAL, 3/6 blowing systolic murmur @ apex radiating to axilla
- Abdomen distended
- Bilateral pitting edema of legs to knees

Case #8

- EKG - NSR w/ low voltage
- CXR – cardiomegaly with pulmonary congestion
- Creatinine 1.4
- K+ 4.0

Case #8

- Discussion
- Is this right or left sided heart failure?
- Both, pulmonary congestion (SOB, DOE, rales) from L-sided HF and abdominal distension (ascites) and edema from R-sided HF.
- Soft S1 suggests decreased systolic function

Case #8

- PE –
- PMI is enlarged and displaced showing volume overload and systolic dysfunction.
- Murmur is mitral regurgitation related to papillary muscle dysfunction.

Case #8

- EKG – what causes low voltage?
- Obesity
- Emphysema
- large pericardial effusion
- Hypothyroidism
- Dilated cardiomyopathy

Case #8

- Treatment options
- Diuretics (usually loop when this bad)
- ACEI (or ARB if can't tolerate ACEI)
- Beta blocker (carvediol is best. Start at tiny dose and titrate up every 2 weeks, 3.125mg up to 25mg bid)

Case #8

- Renal disease
- ACEI benefits renal function even with creatinine in the 3-6 range, but the therapeutic window narrows, especially with bilateral renal artery stenosis.
- Start slowly, monitor renal function closely and get renal consult.
- If creatinine rises >30% in 2 months need to stop ACEI or ARB

Case #9

- 75 yo man with chronic HTN, poorly controlled, despited several meds, including HCTZ, metoptolol and ACEI. Only complaint is weakness and malaise.
- Last visit K+ was 2.9 so KCl 20mEq bid was added and HCTZ was stopped.
- On followup 3 weeks later K+ is still only 2.7 and pt insists he is taking it.

Case #9

- PE
- Alert, well developed older man, NAD
- BP 170/100
- Pulse 85 regular
- Lungs clear
- Heart nl S1, S2, w/o MRG (murmurs, rubs or gallops)
- Extrem w/o edema, peripheral pulses nl.

Case #9

- EKG – NSR w/ 1st degree AVB
- CXR – normal
- Creatinine 1.1
- K+ 2.8
- Na+ 148

Case #9

- What is differential?
- The persistent hypokalemia in the face of fairly large doses of potassium repletion shows that patient is wasting K+, and in the absence of another explanation, i.e. diuretic use, hyperaldosteronism is high in the DDx
- There is usually mild hypernatremia and HTN but other symptoms are variable.

Case #9

- Hyperaldosteronism screening tests
  - Serum potassium and bicarbonate levels: Hypokalemia and metabolic alkalosis have low sensitivities and specificities for PH when these levels are tested by themselves. Hypokalemia (potassium level <3.6 mEq/L) has a sensitivity of 75-80% while the patient is on a normal sodium diet. Typically, it is associated with mild metabolic alkalosis (serum bicarbonate level >31 mEq/L) and inappropriate kaliuresis (urinary potassium excretion >30 mmol/d).
  - Random plasma aldosterone/plasma renin activity (PRA) ratio: Because this ratio is fairly constant over many physiologic conditions, it can be used as a screening test. Normal values are less than 270 when aldosterone concentration is expressed in pmol/L or less than 10 when aldosterone concentration is expressed in ng/dL.

Case #9

- Confirmatory testing
- 24-hour urinary aldosterone excretion
- The 24-hour urinary aldosterone (U-Aldo) excretion test is one of the most useful confirmatory diagnostic tools because it is an index for total daily aldosterone secretion (in a fashion similar to the 24-h urinary free cortisol [UFC], which is typically elevated in patients with Cushing syndrome).
- In PH, the 24-hour U-Aldo is greater than 14 mcg/d (after 3 d of salt loading). Only about 7% of patients with PH have values less than 14 mcg/d.

Case #9

- Imaging studies
- Initial radiologic investigation in the workup of PH is high-resolution, thin-slice (2-2.5 mm) adrenal CT scanning with contrast.
  - Because aldosteronomas tend to be small in contrast to cortisol-producing adrenocortical adenomas, only those at least 1.5 cm in diameter can be detected reliably and consistently.
  - The overall sensitivity of the test is greater than 90%, but the picture is further complicated by the many false-positive findings associated with incidentalomas, which are reported in some series to be found in up to 10% of the general population (the prevalence increases with age).
- It is generally accepted that MRI is not superior to contrast-enhanced CT scanning for adrenal visualization. High-resolution CT scans may actually have better adrenal definition.

Case #9

- Many other diagnostic and imaging tests are available but this is in flux and consultation is indicated.
- If primary hyperaldosteronism is confirmed then definitive treatment is surgical removal of the adenoma.

Case #9

- Due to age and other comorbidities, pt is a poor surgical candidate. What would be the best medical therapy?
- Spironolactone is an aldosterone antagonist and is specifically indicated in this situation. Side effects include estrogen like effects, i.e., gynecomastia and ED
- ACEI and ARB are alternatives.