

## The hypertensive man...

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## Malignant HT

**High BP with imminent  
risk to patient's health**

**Grade III or IV  
Retinopathy**

End organ damage to:

- Brain
- Cardiovascular system
- Kidneys

FIBRINOID  
NECROSIS  
OF  
ARTERIOLES

## When is reduction of BP REALLY an emergency?

- Encephalopathy
- Intracranial haemorrhage
- Dissection
- Acute cardiac failure
- Acute renal failure

.....otherwise it is an URGENCY

## Most cases of malignant HT are due to a secondary pathology

- 80% of identified causes are renal:
- Glomerulonephritis
- Tubulo-interstitial nephritis
- Post-renal – e.g. chronic pyelonephritis
- Renovascular disease

## Other causes of malignant HT

- Essential (20-30% in Caucasians)
- Endocrine – Conn's, Cushing's, phaeo
- Drugs – cocaine, MAOIs
- Tumour-related – Renal cell, Wilm's
- Coarctation of aorta
- Pre-eclampsia

## Principles of management of malignant HT

- The absolute BP is much less important than the rate of BP change, in determining symptoms
- Reduce BP slowly if you can get away with it (to avoid cerebral, cardiac renal ischaemia)
- These patients are often dehydrated
- Have low threshold for involving Critical Care

## Conn's syndrome

- Under-diagnosed cause of HT
- Diagnosis is biochemical:
  - Aldosterone:renin ratio high
  - Must stop anti-HT medications beforehand
- If unilateral adenoma > surgery
- If bilateral adrenal hyperplasia > spironolactone

## Low plasma renin + low plasma aldosterone

- Is an APPROPRIATE reaction of Renin-Angiotensin system to HT
- SOMETHING ELSE has caused HT
  - Phaeo, cocaine or amphetamines
  - Cushing's
  - Renal disease e.g. chronic pyelonephitis

# Hypertensive emergencies: therapies to consider

HYPERTENSIVE EMERGENCY	1 <sup>st</sup> line drug	Alternative drug	Drugs to avoid
Pulmonary oedema	GTN, frusemide	Nitroprusside	
Acute coronary syndrome	GTN + atenolol	Nitroprusside	Hydralazine, nifedipine, minoxidil
Adrenergic crisis	Phentolamine, phenoxybenzamine	Nitroprusside	B-blockers
Renal failure	Labetalol	Nitroprusside (reduce dose)	
Dissecting aortic aneurysm	Nitroprusside+b-blocker	labetalol	Hydralazine, nifedipine, minoxidil
Intracranial haemorrhage	Labetalol	Nitroprusside	Hydralazine, nifedipine, minoxidil

Kitiyakara and Guzman. J Am Soc Nephrol 1998

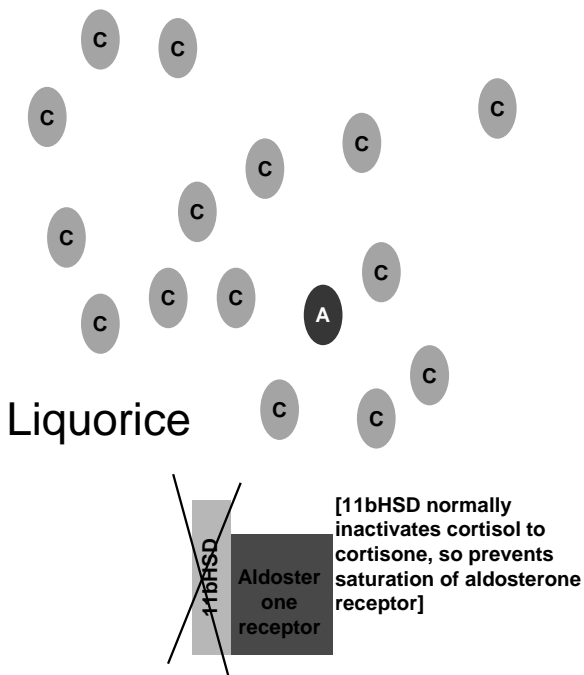
## Secondary hyperaldosteronism

- High plasma renin + high aldosterone
- Most commonly indicates RENAL ARTERY STENOSIS

Cryoactivation occurs if the blood sample gets too cold

- Take blood sample straight to lab
- *Never* put it on ice or refrigerate
- Cooling down will falsely elevate plasma renin measurement

## Apparent mineralocorticoid excess: 11 $\beta$ HSD is inhibited



## Excess catecholamines

- Cocaine
- Amphetamines
- MAOI
- Pheochromocytoma

## Management of suspected phaeo

- THE PATIENT WILL BE DEHYDRATED!
  - Start iv fluids *before*  $\alpha$ -blockade
- $\alpha$ -blockade is ideal therapy
  - iv phentolamine and po phenoxybenzamine
  - Sodium nitroprusside, GTN also work well
- Avoid b-blockade until fully  $\alpha$ -blocked
  - may get paradoxical hypertension

## Investigation of suspected phaeo

- Three 24h urine collections for catecholamines
  - False +ve: TCA and other psychoactive drugs, L-dopa, decongestants
- If raised, further investigations:
  - CT / MRI of abdomen
  - plasma chromogranin A
  - pentolinium / Clonidine suppression test
- NO POINT IN DOING ANY OF THESE IN ACUTE SETTING!

## Risk of cyanide poisoning with nitroprusside

Associated with:

- high dose
  - Infusion rate  $>20\text{mcg/kg/min}$  (recommend 0.5-10)
  - total dose  $>300\text{mg}$
- renal failure

Monitor THIOCYANATE levels

## Take home messages

- HT needs emergency Rx if there is acute end organ damage
- Patients with malignant HT are usually dehydrated
- If you think someone has a phaeo, treat them and investigate later
- Renin / aldosterone samples should be taken straight to the lab, otherwise renin may be falsely raised