Systemic Hypertension•

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Continents •

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2- classification/definition
3- classification/etiology
4-etiology in both categories
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6- clinical finding (symptoms & signs).
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9-Management;
10-**Hypertensive emergencies**
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introduction•

As of 2000, nearly one billion people or ~26% of the adult population of the world had hypertension.

Is a chronic medical condition in which the blood pressure in the arteries is elevated.[1]

sometimes called **arterial hypertension.**

This requires the heart to work harder than normal to circulate blood through the blood vessels.

Normal blood pressure at rest is within the range of 100-140mmHg systolic (top reading) and 60-90mmHg diastolic (bottom reading).

High blood pressure is said to be present if it is persistently at or above 140/90 mmHg.

Classification/Definition•

Classification/Etiology•

1- primary (essential) HTN.

accounting for 90–95%.
no cause can be identified.

2- secondary HTN.

5–10% of cases.
conditions that affect the
kidneys, arteries, heart or endocrine system.

Etiology: type I•

Onset usually : age (25-55yrs)

1- genetic.??
2- environmental.??

3- sympathetic nervous system hyperactivity.

4- renin-angiotensin system.

Only 10% have high levels while 60% N level , 30% low level

5- defect in natriuresis.

Usual response to high BP, Na/ volume load ----increase Na urine excretion

6- intracellular Na, Ca.

? Na-K channel exchange & other Na transport mechanism;

High Na --- high Ca --- high vascular smooth muscle tone ???

7- exacerbating factors:

– obesity – Na intake – alcohol
– smoking – low exercise
– hematological: polycythemia
– drugs: NSAID – low K.

Etiology: type II•

Onset usually : age (<25yrs OR >55yrs)

1- Renal disease.

_Most common cause of 2nd HTN

_May result from: – grumelular disease –tubulointerstitial disease – PCKD .

_mechanism: –fluid over load –rinin-angiotensin-aldosteron activity

_HTN may accelerate progression.

2- Renal Vascular HTN.

A. Renal Artery Stenosis.------ fibromascular hyperplasia.
B. Atherosclerotic Stenosis. ---------- proximal renal artery.
_It can be a single artery stenosis.

Etiology: type II•

3- Primary hyperaldesteronism.
_high aldesteron.
_adrenal disease: adenoma/hyperplasia.

4- Cushing Syndrome.
_excess glucocorticoids.
_mechanism:
–direct effect of mineralocorticoid---salt & water retention
–increase secretions of angiotensinogen.

5- Pheochromocytoma.
_uncommon.
_mechanism: excess catecholamine (–alpha-receptor mediated –beta-receptor mediated)

Etiology: type II•

6- Coarectation of Aorta.
_ uncommon.

7- HTN associated with pregnancy.
_eclampsia/pre-eclampsia

8- Estrogen use.

_OCP

_mechanism: increase rinin-angiotensin activity.

9- Others.

Complications•

_The expected complication are :

–sustained elevated BP with consequent changes in the vasculature & heart

OR

–atherosclerosis accelerated by long standing.

_The mortality & morbidity related to HTN are linked to both systolic and diastolic **BUT** risk is approximately double with diastolic HTN.

<u>Complication of HTN in details in next slides ☺☺</u>

Complications•

1- HYPERTENSIVE CARDIOVASCULAR DISEASES.

_is the major causes of morbidity & mortality in primary HTN.

_**<u>HOW???</u>** LVH →→→ CHF, Ventricular Arrhythmia, MI, … even Sudden Death.

2- HYPERTENSIVE CEREBROVASCULAR DISEASES & DEMENTIA.

_HTN is the major risk factor of stroke /// intracerebral hemorrhage.
_mainly correlate with *systolic HTN*.
_high incidence of Dementia BOTH → (*vascular* & *Alzheimer dementia*).
_effective control modify ***risk*** & ***rate*** of progression.

3- HYPERTENSIVE RENAL DISEASES.
_ Nephrosclerosis.
_HTN can accelerate progression of other renal diseases.

Complications•

4- AORITIC DISSECTION.
_ Is a contributing factor.

5- ATHEROSCLEROSIS COMPLICATION.

Complications•
Clinical Finding;•

_Mainly referable to involvement of the target organs (heart, brain, kidney, eyes, peripheral arteries).

SYMPTOMS:

_In mild/moderate primary (essential) HTN, → usually asymptomatic for many years.

_Most frequent symptoms; HEADACH; is also very non-specific.

_Headache (sub-occipital, early morning); BUT any headache can occur.

_Accelerated HTN associated with Somnolence, confusion, visual disturbance, nausea & vomiting (hypertensive encephalopathy).

Clinical Finding;•

SYMPTOMS:

_Pt's with pheochromocytomas ; may have episodic HTN; attacks of anxiety; palpitation; perfuse respiration; tremors; nausea & vomiting.

_Pt's with primary aldosteronism ;
muscle weakness; polyuria; nocturia; …etc

_Pt's with chronic HTN ; may presented with
cardiac complications ; CHF; CAD/IHD; MI.

_In case of cerebral injuries;
stroke (ischemic or hemorrhagic) ; *hypertensive encephalopathy.*

Clinical Finding;•

SIGNS:

_The main goals on the physical examination are to evaluate for signs of end-organ damage and for evidence of a cause of secondary hypertension.

_ like symptoms; depends on the causes; duration; severity; organ involved.

- BLOOD PRESSURE:

_should be taken in both arms +/- legs. (to exclude coarectation of aorta).

_should be taken in different positions (orthostatic drop in Pheochromocytoma).

_?? Think about pesudohypertension with elderly. (Osler's sign).

- RETINAS:

_ do fundoscopy.

Clinical Finding;•

SIGNS:

- HEART & ARTERIES:

_ Lf ventricular heave →→→ long standing HTN

_CVS exam □□□ signs of valvulars disease.

- PULSES:

_ check timing of upper & lower limbs (to exclude coarectation of aorta).

Investigations•

Lab test:

CBC, U&E, urine chemistry & microscopy
Plasma aldestron , rinin levels
Blood sugar
Lipids profile
Uric acid level
24hrs urine collection for cortisone level

ECG:
_ highly specific but not very sensitive.

Chest X-Ray:
_not necessary .

Investigations•

Echo :
_only if cardiac diseases suspected.

Other Radiological investigations:
_ US

_ CT
_MRI

SINCE MOST HTN CASES ARE PRIMARY (ESSINTIAL) HTN; few investigations are necessary to do *unless therapy is unsuccessful OR there is a suspicion of 2ndry HTN , further investigations are required.*

how to check blood • pressure &measurement strategies

_Three acceptable measurement strategies:

1-Ambulatory blood pressure monitoring (ABPM) 24-48 hrs.

2-Home blood pressure monitoring (one week record)(12-14 times).

3-Office-based blood pressure measurements (at least **three visits**, spaced over a period of one week or more).

_ A patient with elevated office-based BP but normal 24-hour ambulatory BP is considered to have office hypertension or *"white coat" hypertension*.

how to check blood • pressure &measurement strategies

The proper measurement of office-based BP requires attention to all of the following:

_Time of measurement
_Type of measurement device
_Cuff size
_Patient position
_Cuff placement
_Technique of measurement
_Number of measurements

Management;•

1- Life style modification.
_Diet rich in fibers, fruits, low lipids _decrease weight
_decrease alcohol consumptions _ decrease salt intake
_ encourage exercise _smoking cessation.
2- Drug therapy.

_many classes approved
_Diuretics
_Ca channel blockers
_B –blockers
_ACE inhibitors
_ARB
_Others >>>…

3- Treating primary cause if known & possible.

Hypertensive emergencies•

_are acute, life-threatening, and usually associated with marked increases in blood pressure (BP), generally ≥180/120 mmHg ; ***with end organ damage***.

_There are two major clinical syndromes induced :

_with eye involvement :
retinal hemorrhages, exudates, or papilledema.

_with brain involvement :
Hypertensive encephalopathy (signs of cerebral edema)

Hypertensive urgencies•

_are acute, life-threatening, and usually associated with marked increases in blood pressure (BP), generally ≥180/120 mmHg ; ***relatively asymptomatic (****other than perhaps headache****) and have no acute signs of end-organ damage.***

Management:

_ We suggest an initial goal of reducing the blood pressure to ≤160/100 mmHg over several hours to days with conventional oral therapy.

That’s enough

Thanks for attention

Have a nice day