* **Systemic Hypertension**
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* Continents

1- introduction

2- classification/definition

3- classification/etiology

4-etiology in both categories

5- complications

6- clinical finding (symptoms & signs).

7- investigations.

8- how to check blood pressure &measurement strategies

9-Management;

10-**Hypertensive emergencies**

11-**Hypertensive urgencies**

* 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.

**Complication of HTN in details in next slides ☺☺**

* Complications

**1- HYPERTENSIVE CARDIOVASCULAR DISEASES.**

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

\_**HOW???** 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