

## HYPERTENSIVE VASCULAR DISEASE

## Hypertension(HTN)

 Cutoffs in diagnosing hypertension in clinical practice → sustained diastolic pressures >90 mm Hg, and/or sustained systolic pressures >140 mm Hg

#### Malignant hypertension

→ A small percentage of HTN patients (5%) present with <u>a rapidly rising blood pressure</u> that, if untreated, leads to death within 1 to 2 years.

# → systolic pressures > 200 mm Hg or diastolic pressures > 120 mm Hg

→ associated with renal failure and retinal hemorrhages

→ most commonly is superimposed on preexisting benign hypertension

## Hypertension (HTN) has the following complications:

- stroke (CVD)
- multi-infarct dementia
- atherosclerotic coronary heart disease
- cardiac hypertrophy and heart failure (hypertensive heart disease)
- aortic dissection
- renal failure

## **Types of hypertension**

- 1- essential hypertension: most cases (95%) are idiopathic.
- 2- secondary hypertension: Most of the remaining cases (are due to renal disease, or renal artery narrowing ( called renovascular hypertension), and to a lesser degree many other conditions....

Essential HTN	
Accounts for 90% to 95% of all cases	
Secondary HTN	
<u>Renal</u>	
Acute glomerulonephritis	
Chronic renal disease	
Polycystic disease	
Renal artery stenosis	
Renal vasculitis	
Renin-producing tumors	
	Cardiovascular
<u>Endocrine</u>	Coarctation of aorta
Adrenocortical hyperfunction (Cushing syndrome,	Polyarteritis nodosa
primary aldosteronism, CAH	Increased intravascul
licorice ingestion)	Increased cardiac out
Exogenous normones (glucocorticolds, estrogen	Rigidity of the aorta
monoamine oxidase inhibitors)	
Pheochromocytoma	Nourologic
Acromegaly	<u>Neurologic</u> Develogenie
Hypothyroidism (myxedema)	Psychogenic
Hyperthyroidism (thyrotoxicosis)	increased intracrania
Pregnancy-induced (pre-eclampsia)	Sieep apnea
	Acute stress, includin

lar volume tput

I pressure ng surgery

- Pathogenesis of essential HTN
- Genetic factors
- familial clustering of hypertension
- HTN has been linked to specific angiotensinogen polymorphisms and angiotensin II receptor variants; polymorphisms of the renin-angiotensin system.
- Susceptibility genes for essential hypertension are currently **unknown** but probably include those that control renal sodium absorption, etc.

#### Pathogenesis of essential HTN

- Environmental factors
- such as stress, obesity, smoking, physical inactivity, and high levels of salt consumption, modify the impact of genetic determinants.
- Evidence linking dietary sodium intake with the prevalence of hypertension in different population groups is particularly strong.

## Morphology

- HTN is associated with arteriolosclerosis (small arterial disease)
- Two forms of small blood vessel disease are hypertension-related:
- 1- hyaline arteriolosclerosis
- 2- hyperplastic arteriolosclerosis

#### **Hyaline arteriolosclerosis**

- associated with <u>benign</u> hypertension.
- marked by homogeneous, pink hyaline thickening of the arteriolar walls, and luminal narrowing.
- Results from <u>leakage of plasma components</u> <u>across injured endothelial cells</u>, into vessel walls and increased ECM production by smooth muscle cells in response to chronic hemodynamic stress.

- Complications:
- Most significant in the kidneys →
  nephrosclerosis (glomerular scarring).
- Other causes of hyaline arteriolosclerosis (in absence of HTN):
- 1- elderly patients (normo-tensive)
- 2- diabetis mellitus

### Hyperplastic arteriolosclerosis

- is more typical of <u>severe (malignant)</u> hypertension.
- "<u>onionskin</u>," concentric, laminated thickening of arteriolar walls and luminal narrowing.
- The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane.
- In malignant hypertension these changes are accompanied by fibrinoid deposits and vessel wall necrosis (<u>necrotizing arteriolitis</u>), which are particularly prominent in the kidney

A, Hyaline arteriolosclerosis. The arteriolar wall is thickened with the deposition of amorphous proteinaceous material, and the lumen is markedly narrowed.

**B**, Hyperplastic arteriolosclerosis ("onion-skinning") (*arrow*)



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#### DISORDERS OF BLOOD VESSEL HYPERREACTIVITY

- Several disorders are characterized by inappropriate or exaggerated vasoconstriction of blood vessels:
  - 1- <u>Raynaud</u> <u>Phenomenon</u>
  - **2- Myocardial Vessel Vasospasm**

### 1- <u>Raynaud</u> <u>Phenomenon</u>

- results from exaggerated vasoconstriction of arteries and arterioles in the extremities (the fingers and toes, but also sometimes the nose, earlobes, or lips).
  - -restricted blood flow induces paroxysmal pallor or cyanosis
- involved digits characteristically show "red-white-andblue" color changes from most proximal to most distal (reflecting proximal vasodilation, central vasoconstriction, and more distal cyanosis, respectively).

- Raynaud phenomenon can be a primary entity or may be secondary to other disorders

#### **Primary Raynaud phenomenon**

- caused by exaggerated vasomotor responses to cold or emotion (intrinsic hyperreactivity of medial smooth muscle cells)
- affects 3% to 5% of the general population and has a predilection for young women.
- Structural changes in the arterial walls are <u>absent</u> except late in the course, when intimal thickening may appear.
- The course is usually benign
- chronic cases may show atrophy of the skin, subcutaneous tissues, and muscles.
- Ulceration and ischemic gangrene are <u>rare</u>.

#### Secondary Raynaud phenomenon

- refers to vascular insufficiency due to arterial disease caused by other entities
- these include SLE, scleroderma, Buerger disease, or atherosclerosis.
- every patient with Raynaud phenomenon should be evaluated for these secondary causes