Cardiovascular system

Lectures four

Hypertension

Objectives:

At the end of this lecture, the students should be able to:

(1) Know the aetiology
(2) Risk factors
(3) Complications of hypertension
(4) Able to identify patient risk factors amenable to treatment by lifestyle modification
(5) To investigate patients appropriately for causes of secondary hypertension.

Key principles to be discussed:

(1) Raised systemic blood pressure is a major cause of morbidity and mortality.
(2) Hypertension can cause or contribute to: atherosclerosis, left ventricular hypertrophy, chronic renal failure, cerebrovascular disease and retinopathy.
(3) Normal values for blood pressure.
(4) Causes of secondary hypertension.
(5) Genetic and environmental factors contributing to the aetiology of essential hypertension.
(6) Pathology of blood vessels (blood vessels changes) in both primary and secondary hypertension.

Introduction:

- Common problem (25% of population).
- Asymptomatic until later in life.
- Called the Silent Killer – painless –
- Leading risk factor for MI and Stroke.
- Complications alert to diagnosis but late.
- A major cause of morbidity and mortality.

Definition:

- Sustained diastolic pressure more than 90 mm Hg
- Sustained systolic pressure in excess of 140 mm Hg
- (>140/90)

Risk factors for hypertension:

- **Hereditary,** Genetics- family history
- **Race.** African-Americans
- **Gender.** Men & postmenopausal women
- **Age**
- **Obesity**
- **Diet, particularly sodium intake**
- **Lifestyle-stressfu**l
- **Heavy alcohol consumption**
- **Diabetes**
- **Use of oral contraceptives**
- **Sedentary or inactive lifestyle**
- **Smoking**

Classification:

1- Based on etiology (cause):

- **Primary/Essential Hypertension (95%):** It is idiopathic.
- **Secondary Hypertension (5-10%):**
  It can be due to pathology in the renal, endocrine, vascular or neurogenic systems

Causes of Secondary Hypertension

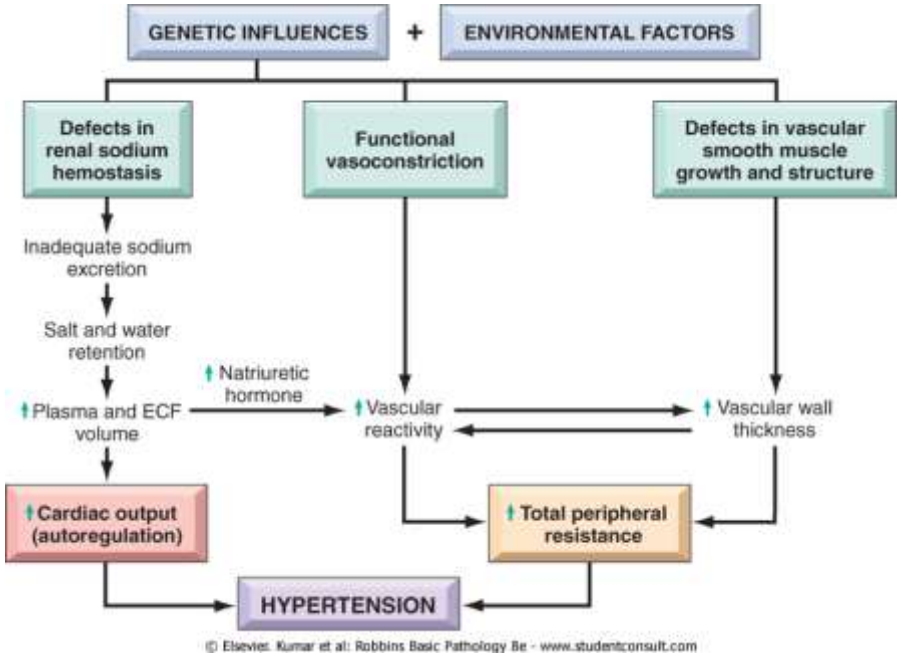
Renal	• **Glomerulonephritis** • **Renal artery stenosis** • **Renal vasculitis** • Adult polycystic disease • Chronic renal disease, • Renin producing tumors • Primary renal disease • Renal artery narrowing (renovascular hypertension) • Adrenal disorders.			
Endocrine	• Adrenocortical hyperfunction (Cushing syndrome, primary aldosteronism, congenital adrenal hyperplasia) • Hyperthyroidism/Thyrotoxicosis • Hypothyroidism/Myxedema. • Pheochromocytoma • Acromegaly • Exogenous hormones (glucocorticoids, estrogen e.g. Oral contraceptives • **Pregnancy-induced**			
Vascular	• Coarctation of aorta • Vasculitis e.g. **Polyarteritis nodosa** • Increased intravascular volume • Increased cardiac output • Rigidity of the aorta			
Neurogenic	• Psychogenic	• Increased intracranial pressure	• Sleep apnea	• Acute stress, including surgery

2- Based on clinical features:

- **Benign:**
  - ✱ Modest level.
  - ✱ Fairly stable over years to decades.
  - ✱ Compatible with long life.
- **Malignant (5%):**
  - ✱ There is rapidly rising BP which often leads to end organ damage (Kidney, Retina, Brain and Heart.)
  - ✱ It can complicate any type of HTN (i.e. essential or secondary)
  - ✱ The diastolic pressure is usually over 120mmHg
  - ✱ It is associated with:
    - Widespread arterial necrosis and thrombosis
    - Rapid development of renal failure
    - Retinal hemorrhage and exudate
    - Hypertensive encephalopathy
    - Left ventricular failure
    - Leads to death in 1 or 2 years if untreated.

Pathogenesis:

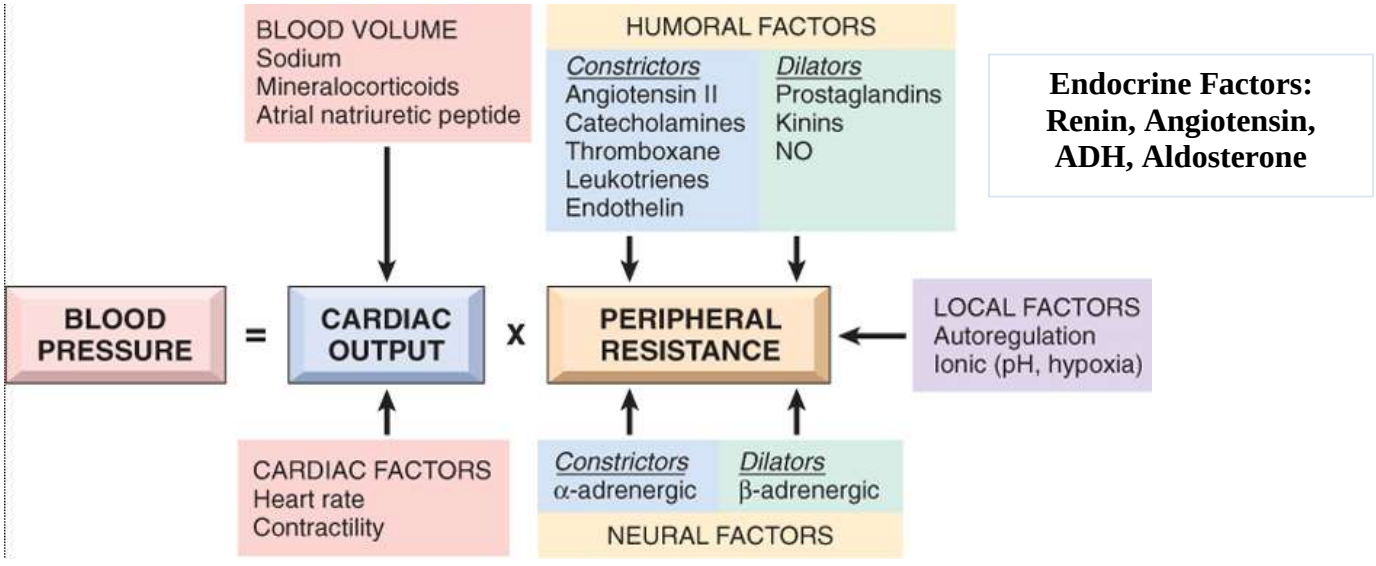
1. Defect in sodium excretion: (common):
   a. Decreased sodium excretion causes an obligatory increase in fluid volume and increased cardiac output, thereby elevating blood pressure.
   b. At the new higher blood pressure, the kidneys excrete additional sodium. Thus, a new steady state of sodium excretion is achieved, but at the expense of an elevated blood pressure.
   c. Defect in cell membrane function: affecting Na/Ca transport
2. Increased sympathetic/ vasoconstrictive response:
   a. Increased vascular resistance may stem from vasoconstriction or structural changes in vessel walls.
3. Rare gene disorders can cause HTN by increasing renal sodium resorption e.g. Liddle syndrome.
   Liddle syndrome is an inherited autosomal dominant type of HTN that begins in childhood. It is caused by mutations of the epithelial sodium channel protein (ENaC), which leads to increased sodium reabsorption in the renal tubules (followed by water), which leads to hypertension. Reabsorption of sodium is also leads with potassium loss (hypokalemia).



Regulation of blood pressure:

Two hemodynamic variables are involved in the regulation of BP: cardiac output and peripheral vascular resistance

- **Cardiac output** is affected by blood volume and is dependent on sodium concentrations.
- **Peripheral resistance** is regulated predominantly at the level of the arterioles (also known as resistance vessels) and is influenced by neural and hormonal inputs.



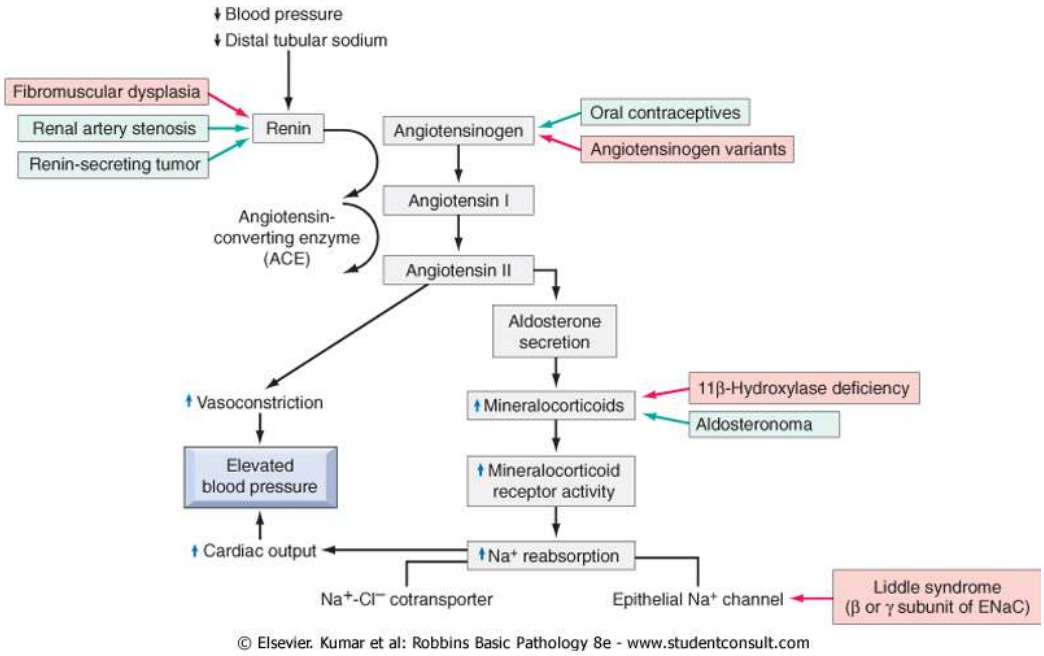
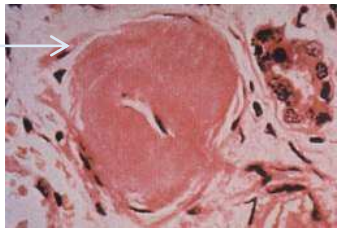
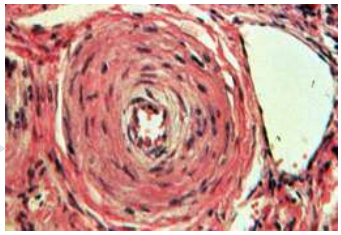


Figure 10-14 Blood pressure variation and the renin-angiotensin system. Components of the systemic renin-angiotensin system are shown in *black.* Some genetic disorders that affect blood pressure by altering activity of this pathway are indicated in *red; arrows* indicate sites in the pathway altered by mutation. Acquired disorders that alter blood pressure through effects on this pathway are indicated in *green.* ENaC, epithelial sodium channel.

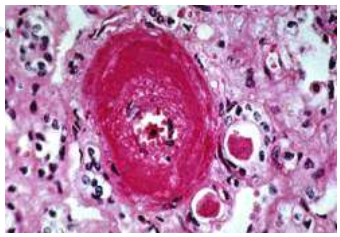
Morphology:

- Large Blood Vessels (Macroangiopathy):
  Atherosclerosis. HT is a major risk factor in AS.
- Small Blood Vessels (Microangiopathy):
  Arteriolosclerosis
  - ✱ Hyaline arteriolosclerosis:
    - Characteristic of **benign hypertension**
    - Can also be seen in elderly without hypertension and in diabetic patients.
    - Leads to benign nephrosclerosis (glomerular scarring). Due to diffuse renal ischemia.
    - Marked by homogeneous, pink hyaline thickening of the arteriolar walls, with loss of underlying structural detail, and luminal narrowing.
    - The lesions stem from leakage of plasma components across injured endothelial cells, into vessel walls and increased ECM production by smooth muscle cells.



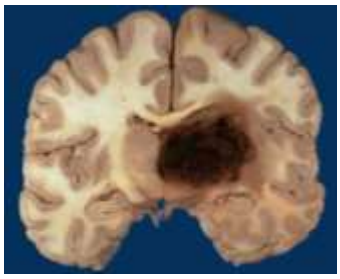


  - ✱ Hyperplastic arteriolosclerosis:
    - Characteristic of malignant hypertension.
    - Onion skin appearance
    - Vessels exhibit "onionskin," concentric, laminated thickening of arteriolar walls and luminal narrowing.
    - May be associated with necrotizing arteriolitis (fibrinoid necrosis), which are particularly prominent in the kidney.
    - The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane.

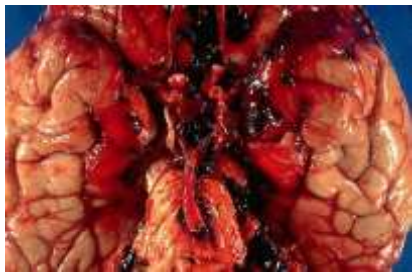


Complications:

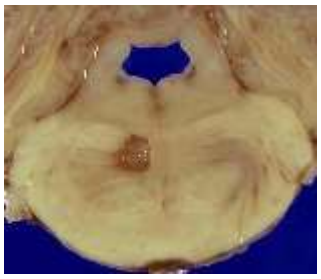
- **Cardiovascular:**
  - Left ventricular hypertrophy
  - Coronary heart disease
  - Aortic dissection
  - Heart failure (*hypertensive heart disease*)
- **Kidney:**
  - Benign nephrosclerosis (photo A).
  - Renal failure in untreated or in malignant hypertension.
- **Eyes:**
  Hypertensive retinopathy (photo B) is especially seen in malignant hypertension.
- **Brain:**
  Hemorrhage, infarction leading to Cerebrovascular accidents. (**Multi-infarct dementia**[1]**)**



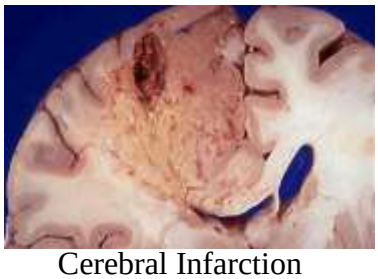
Cerebral Hemorrhage



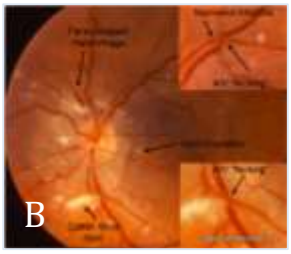
Subarachnoid Hemorrhage



Lacunar Infarct

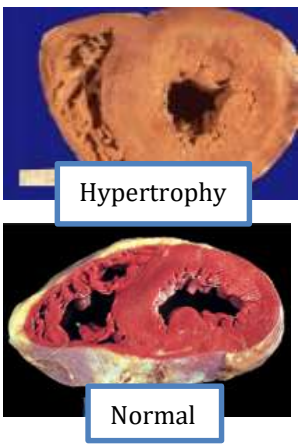


Cerebral Infarction



Left ventricular cardiac hypertrophy (left sided hypertensive cardiomyopathy/ hypertensive heart disease):

- Longstanding poorly treated HTN leads to left sided hypertensive heart disease.
- Hypertrophy of the heart is an adaptive response to pressure overload due to HTN.
- HTN induces left ventricular pressure overload, which leads to hypertrophy of the left ventricle with increase in the weight of the heart.
- The free LV wall is > 2cm and the weight of the heart is > 500 grams



[1] Loss of brain function caused by a series of small strokes. Occurs when the blood flow to any part of the brain is interrupted or blocked.

Please Contact us: Pathology435@gmail.com

Team members:

خالد أبو راس	نوف التويجري
عمار صالح آل منصور	ا لجوهرة المزروع
حمزة عبدالله الفعر	رغد النفيسة
أنس بليغ محمد علي	ريم العقيل
نايف الهادي	سمر العتيبي
ريان منيف	فرح مندوزا
فارس إبراهيم الورهي	منيرة العمري
قصي عبدالباقي عجلان	نوف العبدالكريم
أحمد طه الخياري	
زياد عبدالعزيز السالم	
مشعل الحازمي	

قال صلى الله عليه وسلم: من سلك طريقًا يلتمس فيه علمًا سهّل الله له به طريقًا إلى الجنة

دعواتنَا لكم بالتوفيق.