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جامعة دجلة الاهلية

كلية طب الاسنان

ORAL MEDICINE

LEC. 1 – المحاضرة

المرحلة الرابعة

استاذ المادة

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250

2018-2019

Hypertension

In order to survive and function properly, our tissues and organs need the oxygenated blood that the circulatory system carries throughout the body. When the heart beats, it creates pressure that pushes blood through a network of blood vessels (arteries, capillaries and veins).

This pressure (blood pressure) is the result of two forces, first force (systolic pressure) occurs as blood pumps out of the heart. The second force (diastolic pressure) is created as the heart rests

Hypertension is a condition in which arterial blood (BP) is chronically elevated.

Blood pressure occurs within a continuous range, so cutoff levels are defined according to their effect on patients' risk.

Clinically, hypertension might be defined as that level of BP at which the institution of therapy reduces BP related morbidity

Category	Systolic BP (mmHg)	Diastolic BP (mmHg)
Blood pressure		
Optimal	> 120	> 80
Normal	120 - 129	80 - 84
High normal	130 - 139	85 - 89
Hypertension		
Grade 1	140 - 159	90 - 99
Grade 2	160 - 179	100 - 109
Grade 3	> 180	> 110

Aetiology

In more than 90% of cases, a specific underlying cause of hypertension cannot be found. Such patients are said to have **essential hypertension**.

Hypertension is more common in some ethnic groups, particularly Black Americans and Japanese, and approximately 40–60% is explained by genetic factors. Important environmental factors include a high salt intake, heavy consumption of alcohol, obesity, lack of exercise. There is little evidence that stress causes hypertension

In about 10% of cases, hypertension can be shown to be a consequence of a specific disease or abnormality leading to sodium retention and/or peripheral vasoconstriction (secondary hypertension)

Causes of secondary hypertension

- **Toxemia of Pregnancy (pre-eclampsia)**
- **Renal diseases**
  - Renal vascular disease
  - Glomerulonephritis
  - Polycystic kidney disease
- **Endocrine disease**
  - Phaeochromocytoma
  - Cushing's syndrome Primary
  - hyperaldosteronism (Conn's syndrome)
- **Drugs** oral contraceptive corticosteroid
- **Increased intracranial pressure**
- **Coarctation of aorta**

Mechanisms of Hypertension

Cardiac output and peripheral resistance are the two determinants of arterial BP. Cardiac output is determined by stroke volume and heart rate, stroke volume is related to myocardial contractility and to the size of vascular compartment. Peripheral resistance is determined by functional and anatomic changes in small arteries and arterioles

- **Intravascular volume**

Vascular volume is a primary determinant of arterial pressure over long term. Alterations in total extracellular fluid volume are associated with proportional changes in blood volume. Sodium is a primary determinant of extracellular fluid. When sodium intake exceeds the capacity of the kidney to excrete sodium, vascular volume initially expands and cardiac output increases. As arterial pressure increases in response to high sodium intake, urinary sodium excretion increases and sodium balance is maintained

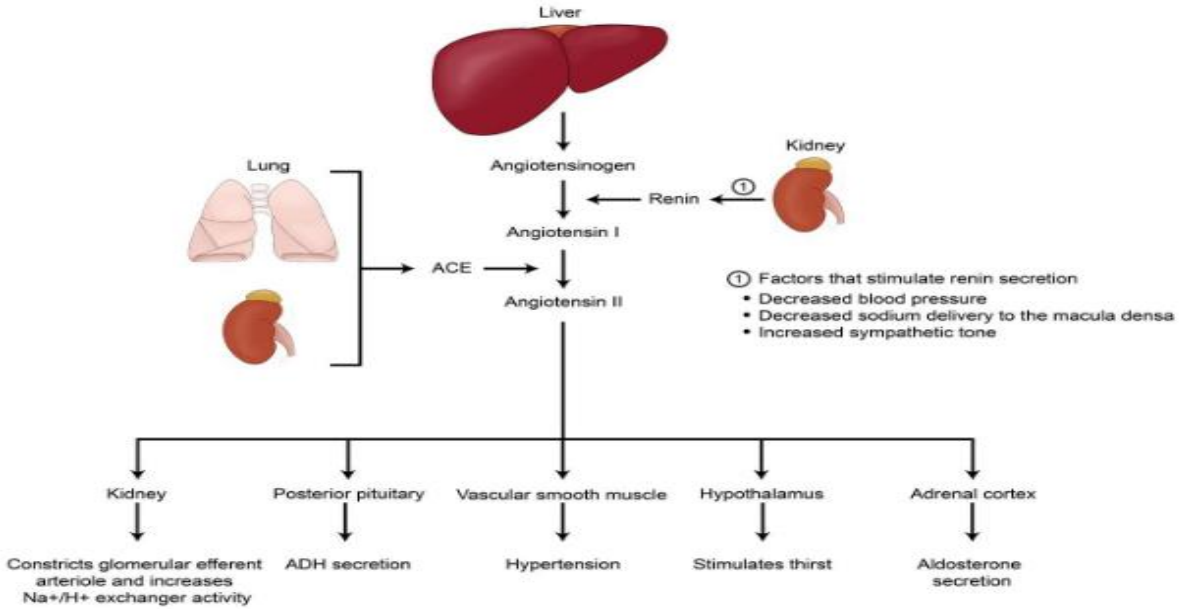
Decrease capacity of the kidneys to excrete sodium (either due to intrinsic renal disease or to increased production of salt retaining hormones) will lead to hypertension

- **Autonomic nervous system**

The autonomic nervous system maintains cardiovascular homeostasis via pressure, volume and chemoreceptor signals.

Adrenergic reflexes modulate BP over short term, and adrenergic function, in concert with hormonal and volume-related factors, contribute to the long term regulation of arterial pressure.

- **Renin-Angiotensin-Aldosterone System**



- **Vascular mechanisms**

Vascular radius and compliance of resistance of arteries are also determinant of arterial blood pressure.

Resistance to flow varies inversely with the fourth power of the radius, and consequently small decrease in lumen size significantly increase resistance.

Lumen diameter is also related to elasticity of the vessel. Vessels with high degree of elasticity can accommodate an increase of volume with relatively little change of pressure

Target organ damage

- **Blood vessels**

The primary way that high BP causes harm is by increasing the workload of the heart and blood vessels - making them work harder and less efficiently.

Over time, the force and friction of high BP damages the delicate tissues inside the arteries. In turn LDL (bad cholesterol) forms plaque along tiny tears in the artery walls, signifying the start of atherosclerosis

The more the plaque and damage increases ,the narrower the inside of the arteries become - raising BP and starting a vicious circle that further harms the arteries, heart and the rest of body organs.

Hypertension is a major risk factor in the pathogenesis of aortic aneurysm and aortic dissection.

- **Heart**

The excess cardiac mortality and morbidity associated with hypertension are largely due to a higher incidence of coronary artery disease. High BP places a pressure load on the heart and may lead to left ventricular hypertrophy.

Atrial fibrillation is common and may be due to Diastolic dysfunction caused by left ventricular hypertrophy or the effects of coronary artery disease.

Severe hypertension can cause left ventricular failure in the absence of coronary artery disease, particularly when renal function, and therefore sodium excretion, is impaired.

- **Central nervous system**

Stroke is a common complication of hypertension and may be due to cerebral haemorrhage or infarction. Carotid atheroma and transient

ischaemic attacks are more common in hypertensive patients. Subarachnoid haemorrhage is also associated with hypertension.

Hypertensive encephalopathy is a rare condition characterised by high BP and neurological symptoms, including transient disturbances of speech or vision, paraesthesiae, disorientation, fits and loss of consciousness.

- **Retina**

The optic fundi reveal a gradation of changes linked to the severity of hypertension; fundoscopy can, therefore, provide an indication of the arteriolar damage occurring elsewhere. Hypertension is also associated with central retinal vein thrombosis

- **Renal**

Long-standing hypertension may cause proteinuria and progressive renal failure by damaging the renal vasculature.

Malignant Hypertension

This rare condition may complicate hypertension of any etiolog and is characterized by accelerated microvascular damage with necrosis in the walls of small arteries and arterioles (‘fibrinoid necrosis’) and by intravascular thrombosis.

The diagnosis is based on evidence of high BP and rapidly progressive end organ damage, such as retinopathy (grade 3 or 4), renal dysfunction (especially proteinuria) and/or hypertensive encephalopathy. Left ventricular failure may occur and, if this is untreated, death occurs within months.

Clinical features

Hypertension is frequently discovered on routine examination of apparently healthy people.

Symptoms are rare, and when present are generally attributed to the complications of hypertension.

Minor symptoms such as headache, dizziness, irritability and fatigue, though wide spread, are inappropriately attributed to hypertension.

Investigations

- Urinalysis for blood, protein and glucose
- Blood urea, electrolytes and creatinine
- Blood glucose
- Lipid profile
- 12-lead ECG (left ventricular hypertrophy, coronary artery disease)

Management

- **Lifestyle modifications**

Appropriate lifestyle measures may obviate the need for drug therapy in patients with borderline hypertension, reduce the dose and/or the number of drugs required in patients with established hypertension, and directly reduce cardiovascular risk.

Correcting obesity, reducing alcohol intake, restricting salt intake, taking regular physical exercise and increasing consumption of fruit and vegetables can all lower BP. Moreover, quitting smoking, eating oily fish

and adopting a diet that is low in saturated fat may produce further reductions in cardiovascular risk.

- **Pharmacological therapy**

There is considerable variation in individual responses to different classes of antihypertensive drugs, and the magnitude of response to any single agent may be limited by activation of counterrgulatory mechanisms that oppose the hypotensive effect of the agent.

Selection of antihypertensive drugs and combination of drugs should be individualized, taking into account age, severity, other cardiovascular disease risk factors, comorbid conditions and practical considerations related to cost, frequency of dosing and side effects.

1. **Diuretics :** hydrochlorthiazide ,chlortalidone
2. **Angiotensin converting enzyme (ACE) inhibitors :** Captopril , enalapril
3. **Angiotensin receptor blockers :** valsartan, candesartan
4. **Aldosterone antagonists :** spironolactone
5. **Calcium channel blockers :** Amlodipine, diltiazem
6. **Beta blockers :** atenolol, metorolol, carvrdilol, labetalol
7. **Direct vasodilator :** Hydralizine, sodium nitrorosside, prazosin