Diseases of the Blood vessels

Normal anatomy

The general architecture and cellular composition are the same throughout the cardiovascular system.

Endothelial Cells (ECs), Smooth Muscle Cells (SMCs) constitute the bulk of vessel wall cellularity, the remainder of the wall composed of Extracellular Matrix (ECM) including elastin & collagen.

Vessel walls are organized in three distinct concentric layers, intima, media and adventitia

The intima consists of endothelial cell monolayer on a basement membrane with minimal underlying ECM, the intima is demarcated from the media by a dense layer of elastic membrane called internal elastic lamina

The media is composed predominantly of smooth muscle cells and ECM and surrounded by loose connective tissue, nerve fibers, and small blood vessels of adventitia. The media is separated from the adventitia by external elastic membrane that is present in some arteries

Based on the size and structural features of arteries, they are classified into 3 basic types:

1– Large or elastic arteries: Including aorta and its large branches, and pulmonary arteries, in these vessels the media consist of elastic fibers alternate with smooth muscle cells.

2– Medium–sized or muscular arteries: Including smaller branches of aorta like coronary arteries and renal arteries, here the media consist mainly of SMCs with limited elastin.

3– Small arteries (2mm) and arterioles: which lie within the interstitial connective tissue of the organs, here the media composed essentially of SMCs.

Atherosclerosis

Is characterized by Intimal lesion called atheroma (atheromatous or atherosclerotic plaques), that protrude into the vascular lumina.

An atheromatous plaque consists of a raised lesion with a soft, yellow core of lipid (mainly cholesterol) covered by a firm white fibrous cap, beside obstructing the blood flow, the atherosclerotic plaque weakens the underlying media.

Constitutional Risk factors:

1- Age: Age is a dominant influence, the incidence of myocardial infarction is increased five folds between ages 40 and 60 years.

2- Gender: Premenopausal women are protected against atherosclerosis in comparisons with age related men, after menopause the risk will be equal.

3- Genetics: Family history is the most important independent risk factor for atherosclerosis.

Major modifiable risk factors:

1- Hyperlipidemia: (hypercholesterolemia): Is a major risk factor of atherosclerosis even in the absence of other risk factors.

The major component of serum cholesterol is LDL (low density lipoprotein). While HDL is protective against atheroma.

2- Hypertension: Both systolic and diastolic levels are important and increase the risk for about 60% in comparisons with normotensive population.

3- Cigarette smoking: Prolonged (years) smoking of one pack per day of cigarette increases the risk for double.

Cessation of smoking reduces the risk.

4- Diabetes mellitus: Induces hypercholesterolemia and increase the risk factor for atherosclerosis.

Pathogenesis The response to injury hypothesis

This hypothesis views the atherosclerosis as chronic inflammatory response of the arterial wall to endothelial injury.

The following events will occur:

1- Chronic endothelial injury with resultant endothelial dysfunction, increased permeability, leukocyte adhesion and thrombosis.

2-Accumulation of lipoprotein mainly LDL in the vessel wall.

3-Monocyte adhesion to the endothelium followed by migration into the intima and transformation into macrophages and foam cells.

4-Platelet adhesion

5-Factors released from activated platelets, macrophages and endothelium induces SMCs recruitment from media.

6- SMCs proliferation and ECM production.

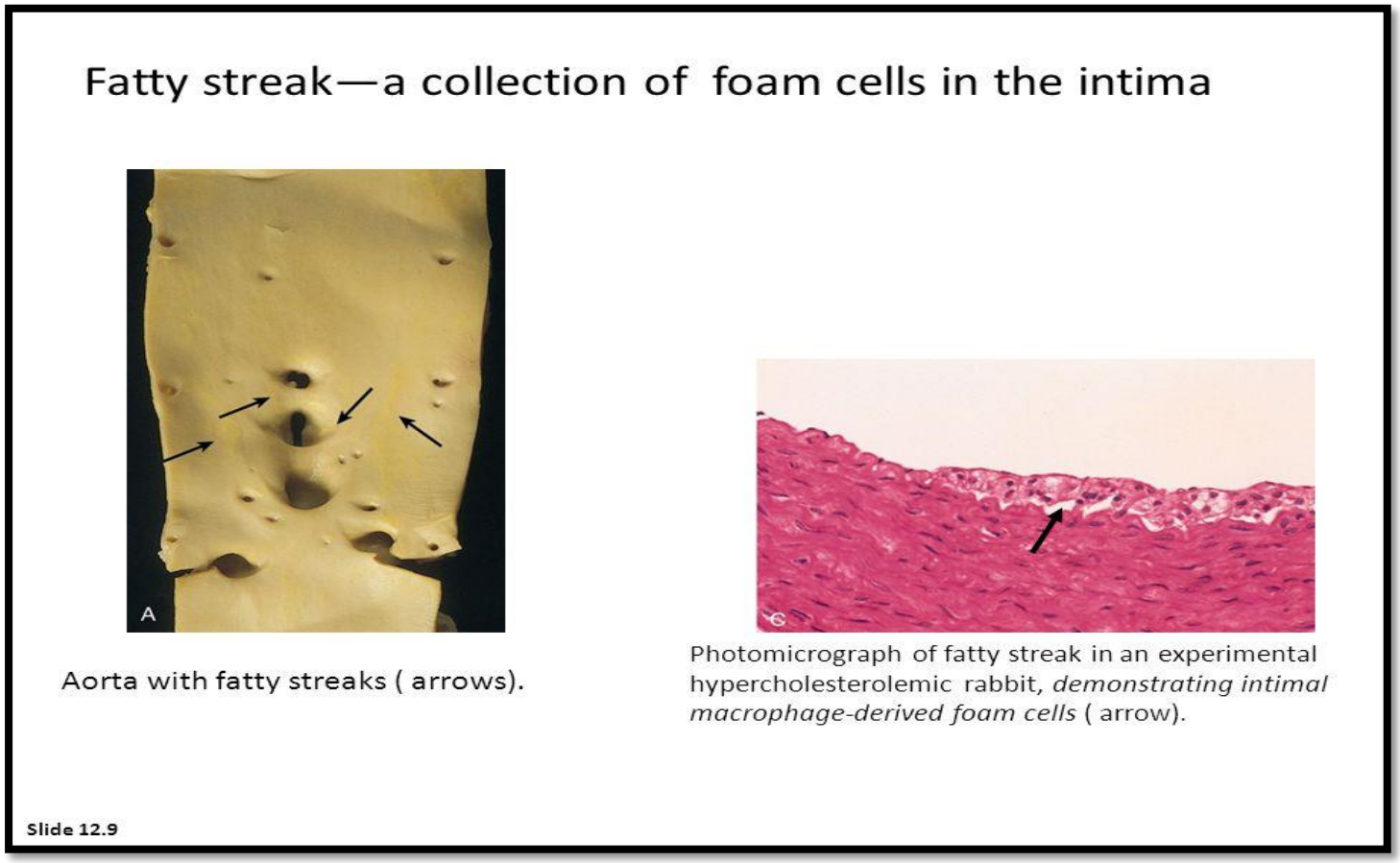
7- Lipid accumulation both extracellularly and within the cells. The accumulation of lipid-containing macrophages in the intima gives rise to fatty streaks, with further evolution fibro-fatty atheroma will developed

Morphology:

1- fatty streak: are composed of lipid-filled foam cells but are not significantly raised and so does not affect the blood flow. They begin as multiple tiny yellow flat spots which then coalesce into elongated streaks 1cm long.

2-Atherosclerotic plaques: are raised masses protrude into the lumen and vary in size from 0.3 to 1.5cm, appear yellow white or red if thrombosis is superimposed.

The most common sites are the lower abdominal aorta, coronary arteries, popliteal arteries, internal carotid arteries and vessels of circle of Willis.



Complications:

1- Rupture, ulceration or erosion of the vessel.

2-Hemorrhage.

3- Atheroembolism.

4- Aneurysmal formation.

Hypertensive Vascular diseases

Sustained diastolic pressure more than 90 mmHg and sustained systolic pressure more than 140 mmHg constitutes hypertension.

Pathogenesis:

90-95% of hypertension is idiopathic (essential hypertension) which is compatible with long life unless myocardial infarction, cerebrovascular accident or other complications supervene.

Most of the remainders of benign hypertension is secondary to renal disease, or narrowing of the renal arteries or less often diseases of adrenal glands like Cushing's syndrome and pheochromocytomas

Accelerated or malignant hypertension

is a clinical syndrome is characterized by severe hypertension diastolic pressure more than 120 mmHg, renal failure and retinal hemorrhage

It may developed in previously normotensive persons or preexisting hypertensive either essential or secondary.

Morphology:

1- Hyaline arteriolosclerosis: Homogenous hyaline thickening of the walls of the arterioles with loss of the underlying structural details and narrowing of the lumen, found in elderly persons even they are normotensive and in diabetes.

2- Hyperplastic arteriolosclerosis: related to severe elevations of blood pressure (diastolic pressure more than 120mmHg). It is characterized by onion-skin concentric laminated thickening of the walls of the arterioles with luminal narrowing, these laminations consist of SMCs with thickened basement membrane.