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ATHEROSCLEROSIS

- Atherosclerosis, also known as Arteriosclerotic Vascular Disease or ASVD.
- It is a disease of the arteries characterized by the deposition of fatty material on their inner walls.
- The condition in which an artery wall thickens as the results of a build-up of material such as cholesterol.
- Affecting arterial blood vessels a chronic inflammatory response in the walls of arteries.
- Due to the accumulation of macrophage white blood cells and promoted by low-density lipoproteins without adequate removal of facts and cholesterol from the macrophages by functional high density lipo-proteins.
- It is commonly ~~~~ referred to as a hardening or furring of the arteries.
- It is caused by the formation of multiple plaques within the arteries.
- It can restrict blood flow, these plaques can also burst causing a blood-clot.
- Although atherosclerosis is often considered a heart Problem, It can affect arteries any where in your body.
- Atherosclerosis is a preventable and treatable condition.

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Terms:

Arteriosclerosis:

It is a general term describing any thickening (and loss of elasticity) of medium or large arteries.

Arteriolosclerosis:

It is any thickening (and loss of elasticity) of arterioles (small arteries)

Atherosclerosis:

It is a thickening of an artery specifically due to an atheromatous plaque.

Atherogenic:

It is used for substances or processes that cause atherosclerosis.

Atherogenesis:

It is the developmental process of atheromatous plaques.

Causes:

⊙ Atherosclerosis starts with damage or injury to the inner layer of an artery.

① The damage may be caused by -

- High blood pressure.
- High cholesterol.
- An irritant, such as nicotine.
- Certain diseases, such as diabetes.

athophysiology:

⊙ Atherosclerosis develops as a chronic inflammatory response of the arterial wall to endothelial injury.

⊙ Lesion progression occurs through interactions of modified lipoproteins, monocyte derived macrophages, T-lympho-cytes and the normal cellular constituent of the arterial wall.

⊙ The contemporary view of atherosclerosis is expressed by the response-to-injury hypothesis.

Response-to-injury-hypothesis:

The following are the steps involved in the hypothesis.

1. chronic endothelial injury.
2. Accumulation of lipoproteins.
3. Monocyte adhesion to the endothelium.
4. SMc (Smooth muscle cell) proliferations and ECM
5. Factor Release.
6. platelet adhesion.

1. Chronic endothelial injury:

⊙ with resultant endothelial dysfunction, causing increased permeability, leukocyte adhesion and throm-bosis.

2. Accumulation of lipoproteins:

⊙ (Mainly LDL and it's oxidized forms) in the vessel wall. Low-density lipoprotein molecules (LDL) becoming

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oxidized (LDL-ox) by free radicals particu-
oxygen free (ROS). When oxidized LDL comes in contact with an artery wall, a series of reactions occur to repair the damage to the artery wall caused by oxidized LDL. cholesterol can move in the blood stream only by being transported by lipoproteins.

3. Monocyte Adhesion to the Endothelium:

Followed by a migration into the intime and transformation into macrophages and foam cells. The body's immue system responds to the damage to the artery local caused by oxidized LDL by sending specialized white blood cells (maenophages and T-lymphocytes) to absorb the oxidized LDL forming specialized foam cells. Unfortunately, these white blood cells are not able to process the oxidized LDL and ultimately grow then rupture depositing a great amount of oxidized cholesterol into the artery wall. This triggers more white blood cells, continuing the cycle.

4. SMC proliferations and ECM production:

Eventually, the artery becomes inflamed. The cholesterol plaque causes the smooth muscle cells to enlarge and form a hard cover the affected area. This hard cover is what causes a narrowing of the artery reduces the blood flow and increases blood pressure.

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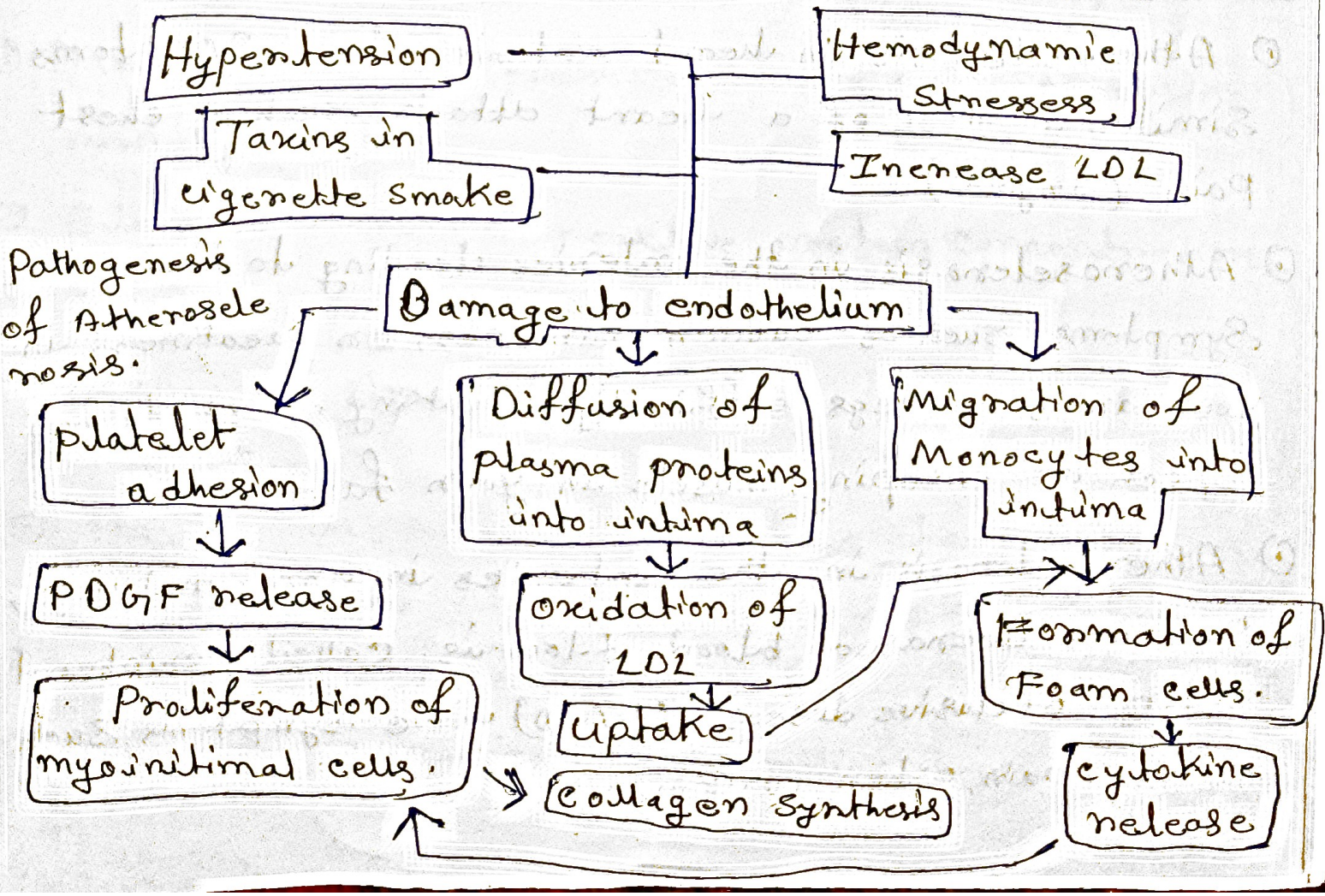
5. Factor release:

From activated platelet macrophages and vascular wall cells, including SMC recruitment, either from the media or from the circulating precursors.

Lipid Accumulation:

Both extracellularly and within cells (macrophages and SMC's), accumulation of lipid-containing macrophages in the intima gives rise to "fatty streaks", with further evalution, fibro fatty-atheroma consisting of proliferated SMC foam cells extracellular lipid and ECM is formed

Pathogenesis of Atherosclerosis:



Sab!

Symptoms:

- Atherosclerosis develops gradually, typically begins in early adolescence, and is usually found in most major arteries.
- No atherosclerosis symptoms until an artery is so narrowed or clogged.
- Sometimes a blood clot completely obstructs blood flow or even breaks apart and cause heart attack or stroke.

Atherosclerosis symptoms depend on which arteries are affected.

For examples:

- Atherosclerosis in heart arteries, have symptoms similar to those of a heart attack such as chest pain (anginal)
- Atherosclerosis in the arteries leading to brain, have symptoms such as sudden numbness or weakness in your arms or legs, difficulty speaking or ~~st~~ slurred speech or drooping muscle in your face.
- Atherosclerosis in the arteries in arms and legs, produces decreased blood flow is called peripheral artery occlusive disease (PAOD) have symptoms such as leg pain, when walking.

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Sometimes atherosclerosis causes erectile dysfunction in men.

- physiological factors that increase risk Various anatomic, physiological and behavioral risk factors for atherosclerosis are known.

These can be divided into various categories - Modifiable and non-modifiable.

1. Modifiable:

- Having diabetes or Impaired glucose tolerance (IGT)
- Dyslipoproteinemia (unhealthy patterns of serum proteins carrying fats and cholesterol)
- Tabacco Smoking.
- Having high blood pressure, on its own increasing risk by 60%.
- Elevated serum C-reactive protein concentrations.

2. Non-modifiable:

- Advanced Age.
- Male Sex.
- Having close relatives who have had some complication of atherosclerosis (Eg -> coronary heart disease or stroke)
- Genetic Abnormalities, Eg - familial hypercholesterolemia.

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3. Lesser or uncertain:

- Being obese (in particular central obesity)
- A sedentary lifestyle.
- post menopausal estrogen deficiency.
- High carbohydrate intake.
- Elevated serum levels of triglycerides.
- Elevated serum lipoprotein concentrations.
- Stress or symptoms of clinical depression.
- Hyperthyroidism.
- Elevated serum insulin levels.
- Short sleep duration.