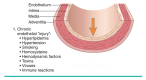
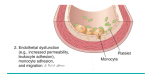
ARTERIOSCLEROSIS: hardening of the arteries

Atherosclerosis

- General info.
  - most frequent and clinically important pattern of arteriosclerosis
  - characterized by ***intimal*** lesions known as atheromas,atheromatous plaque, atherosclerotic plaques
    - raised lesion with a **core of lipid** (cholesterol and cholesterol esters) covered by a firm, white **fibrous cap**
- Pathogenesis (we don't know who is the cause and who is the effect)
  - inflammatory process in endothelial cells of vessel wall
  - retained LDL particles
- Pathogenic events: (with extra notes from the book)
  - 1) Endothelial Cell injury

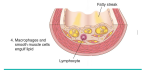


  - 2) Resultant Dysfunctional Endothelium (Intact but dysfunctional)



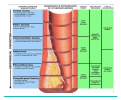
  - 3) Monocytes migrate into the intima and differentiate into macrophages, Smooth muscle recruitment, accumulation of lipoproteins in the vessel wall

  - 4) lipid accumulation within macrophages (foam cells)



  - 5) Smooth muscle proliferation and ECM production

- Progression:



- Vulnerable vs stable plaque
  - Vulnerable Plaque
    - Thick fat core
    - Thin fibrous cap
    - More inflammation
  - Stable Plaque
    - Thin fat core
    - Thick fibrous cap
    - less inflammation
- Risk factors
  - Major RF

    - Non-modifiable (non-controllable/ constitutional)
      - Family history
      - Age
        - Atherosclerosis usually remains clinically silent until lesions reach a critical threshold in middle age or later
          - The incidence of MI increases 5-fold between 40 and 60 years of age.
          - Death rates from IHD rise with each decade
      - Genetics
        - Familial predisposition is multifactorial
          - Well-defined genetic derangements in lipoprotein metabolism (account only small percentage of cases)
            - familial hypercholesterolemia
          - Familial clustering of other risk factors that go hand in hand with atherosclerosis
            - HTN, DM
      - Gender
        - **Premenopausal** women are relatively protected against atherosclerosis (and its consequences) compared with age-matched men.
          - Complications of atherosclerosis like MI are uncommon in premenopausal women in the absence of other predisposing factors such as diabetes, hyperlipidemia, or severe hypertension.
        - **After menopause**, the incidence of atherosclerosis-related disease increases and can even exceed that in men.
    - Potentially modifiable (Controllable)
      - Hyperlipidemia
      - Hypertension
      - Diabetes
      - Cigarette smoking
      - C-reactive protein (inflammation)
  - Additional RF (20% of cardiovascular events occur in the absence of identifiable risk factors)
    - Lesser, Uncertain, or Non-quantitated Risks
      - Lipoprotein (a) levels
      - Hyperhomocystinemia
      - Factors Affecting Hemostasis (Elevated levels of procoagulants...)
      - Metabolic syndrome
      - Obesity
      - High carbohydrate intake
      - Hardened (trans)unsaturated fat intake
      - Postmenopausal estrogen deficiency
      - lack of exercise /Physical inactivity
      - competitive, stressful lifestyle ("type A" personality)
      - Chlamydia pneumoniae infection

Mönckeberg medial calcific sclerosis

- calcific deposits in muscular arteries
- persons > age 50
- radiographically visible
- palpable vessel
- do not encroach on vessel lumen
- usually not clinically significant

Arteriolosclerosis

- affects small arteries and arterioles
- associated with hypertension and/or diabetes mellitus

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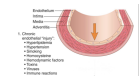
      - 
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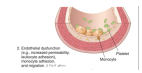
      - 
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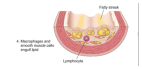
      - 
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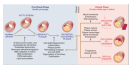
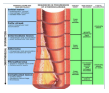
    - 
    - 
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