ARTERIOSCLEROSIS & ATHEROSCLEROSIS
EPIDEMIOLOGY AND RISK FACTORS

LEARNING OBJECTIVES
At the end of the lecture student will able to describe the:
• Etiology/Risk Factors
• Pathogenesis
• Morphology
• Clinical Expression

MORPHOLOGIC CONCEPTS
• Macrophages (really monocytes) infiltrate
• Intimal Thickening
• Lipid Accumulation
• Streak
• Atheroma
• Smooth Muscle Hyperplasia and Migration
• Fibrosis
• Calcification
• Aneurysm
• Thrombosis

Blood vessel wall becomes distended and continues to accumulate cholesterol, some areas become calcified
ADVANCED FEATURES

• RUPTURE
• ULCERATION
• EROSION
• ATHEROEMBOLI
• HEMORRHAGE
• THROMBOSIS
• ANEURYSM

• Related to "development" of nation
• US highest
• AGE
• SEX, M>F until menopause, estrogen “protection”
• GENETICS

MAJOR FACTORS

• Hyperlipidemia
• Hypertension
• Cigarette Smoking
• Diabetes Milletus
HYPERLIPIDEMIA

- Chiefly CHOLESTEROL, LDL>>>HDL
- HDL mobilizes cholesterol FROM atheromas to liver
- LOW CHOLESTEROL diet is GOOD
- UNSATURATED fatty acids GOOD
- Omega-3 fatty acids GOOD
- Exercise GOOD

Foamy" MACROPHAGES
CHOLESTEROL CLEFTS

HYPERTENSION

- HYPERTENSION causes

CIGARETTES

- What more needs to be said?

DIABETES

- If there was one disease which I could challenge you to, as a dare, to PROVE to me that was NOT EXACTLY THE SAME as atherosclerosis, it would be DIABETES! Any takers?
NON MAJOR FACTORS

- Homocysteinuria/homocysteinemia, related to low B6 and folate intake
- Coagulation defects
- Lipoprotein Lp(a), independent of cholesterol. Lp(a) is an altered form of LDL
- Inadequate exercise, Type “A” personality, obesity (independent of diabetes)
- Protective effect of moderate alcohol? Medical LIE, sponsored by the booze industry and alcoholic physicians!

PATHOGENESIS

- “atherosclerosis is a chronic inflammatory response of the arterial wall initiated by injury to the endothelium”

PATHOGENESIS SAGA

- Chronic endothelial injury
- LDL, Cholesterol in arterial WALL
- OXIDATION of lipoproteins
- Monocytes migrate to endothelium
- Platelet adhesion and activation
- Migration of SMOOTH MUSCLE from media to intima to activate macrophages (foam cells)
- Proliferation of SMOOTH MUSCLE and ECM
- Accumulation of lipids in cells and ECM

Main FOUR STARS of PATHOGENESIS SAGA

- 1) Endothelial Injury
• 2) Inflammation
• 3) Lipids
• 4) Smooth Muscle Cells, SMCs

PREVENTION PRINCIPLES

• Know what is preventable
• Know what is MAJOR (vs. minor)
• Know PRIMARY vs. SECONDARY principles
• Understand atherosclerosis begins in CHILDHOOD
• Risk factors in CHILDREN predict the ADULT profile
• Understand SEX, ETHNIC differences

ATHEROMATOUS PLAQUE DEVELOPMENT

Blood vessel wall becomes distended and continues to accumulate cholesterol, some areas become calcified

• Low Density Lipids (LDL) pass through damaged endothelium into blood vessel wall

• LDL’s are oxidized and then induce production of bio active molecules. Platelet Derived Growth Factor, Tumor Necrosis Factor Alpha.
• Monocytes transform to macrophages and take up LDL to form foam cells
• High Density Lipids (HDL) inhibit oxidation of LDL

**ARTERIO-SCLEROSIS**

• GENERIC term for ANYTHING which HARDENS arteries
  – Atherosclerosis (99%)
  – Mönckeberg medial calcific sclerosis (1%)
  – Arteriolosclerosis, involving small arteries and arterioles, generally **regarded** as NOT strictly being part of atherosclerosis, but more related to hypertension and/or diabetes

---------------------THANKS---------------------