Cardiovascular System Disorders 2

Lecture 20
Pathology and Clinical Science 1 (BIOC211)
Department of Bioscience

Text Reference:
Porth’s Pathophysiology: Concepts of Altered Health States
Ninth Edition.
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Session Learning Outcomes

The aim of this session is to:

- Comprehend the pathophysiology of atherosclerosis
- Identify the risk factors and understand how to prevent the atherosclerosis and Coronary Heart Disease (CHD)
- Describe the clinical features, investigations and management of CHDs.
ATHEROSCLEROSIS

Introduction

- May manifest as CAD (angina, MI, sudden death), cerebrovascular disease (stroke and TIA) or peripheral vascular disease (claudication and limb ischemia)
ATHEROSCLEROSIS

- Complex inflammatory process.

- Vascular endothelium is critical for maintaining vascular integrity & homeostasis.

- Atherogenesis thought to be triggered by initial endothelial injury or dysfunction:
  - Mechanical shear stresses
  - Biochemical abnormalities
  - Immunological factors
  - Inflammation
  - Genetic alteration
### RISK FACTORS

#### Traditional Risk Factors

- **Age**
  - Increases with age

- **Gender**
  - $M > F$ (premenopausal)
  - $M = F$ (postmenopausal)

- **Family History**
  - Other risk factors can be familial
  - ? independent

- **Smoking**
  - Proportional to quantity
  - Decrease in risk (almost to normal) after 10yrs abstinence
# RISK FACTORS

- **Diet and Obesity**
  - High fat, low antioxidants

- **Hypertension**
  - Systolic & diastolic

- **Hyperlipidaemia**
  - High LDL
  - Low HDL
  - Decreased cholesterol = reduced progression + regression of coronary artery disease (CAD)
  - Lipid lowering Rx = decreased mortality

- **Diabetes mellitus**
  - Also magnifies the effect of other risk factors
LIPID TRANSPORT

Schematic representation of the exogenous and endogenous pathways for triglyceride and cholesterol transport.

## RISK FACTORS

### Newer risk factors

- **Sedentary life style**
  - Lack of exercise

- **Psychological well-being**
  - Work stress, lack of social support, depression & personality

- **Alcohol**

- **Genetic factors**

- **Lipoprotein (a)**

- **Coagulation factors**
  - Serum fibrinogen, homocysteine

- **C-reactive protein (CRP)**
# RISK FACTOR MANAGEMENT

- **Primary prevention**
  - modification of risk factors that reduce the development of coronary atheroma

- **Secondary prevention**
  - modification of risk factors that slow the progression of established disease

- **Smoking**
  - ↓ amount
  - complete cessation

- **Hypertension**
  - Control HT
RISK FACTOR MANAGEMENT

- **Lipids**
  - any decrease is beneficial
  - Lipid lowering agents (statins)

- **Exercise**
## RISK FACTOR MANAGEMENT

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<table>
<thead>
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<tbody>
<tr>
<td><strong>Weight</strong></td>
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<tr>
<td>• Maintain ideal body weight</td>
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<tr>
<td><strong>Diabetes Mellitus (DM)</strong></td>
<td></td>
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<tr>
<td>• Optimise BGL</td>
<td></td>
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<tr>
<td><strong>Diet</strong></td>
<td></td>
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<tr>
<td>• Reduce saturated fat</td>
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<tr>
<td>• Increase vegetables + grains</td>
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<tr>
<td>• Fish, lean meat</td>
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<tr>
<td>• Limit take-away, snack food, cakes etc</td>
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<tr>
<td>• Avoid high cholesterol foods + offal</td>
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DEVELOPMENT OF ATHEROSCLEROSIS

- Follows endothelial dysfunction
- Accumulation of oxidized lipoproteins
- Lipoproteins are taken up by macrophages
- Macrophages become lipid-laden foam cells
- Fatty streaks appear & progress
- Plaque formation
- Cytokine release further damages endothelium
DEVELOPMENT OF ATHEROSCLEROSIS

Endothelial Cell Injury

Migration of Inflammatory Cells

DEVELOPMENT OF ATHEROSCLEROSIS

Lipid Accumulation & Smooth Muscle Cell Proliferation

Plaque Structure

DEVELOPMENT OF ATHEROSCLEROSIS

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DEVELOPMENT OF ATHEROSCLEROSIS

ATHEROSCLEROSIS

http://www.portalesmedicos.com/imagenes/publicaciones_10/1009_modelo_animal_arteriosclerosis/arteria_coronaria_atherosclerosis.jpg

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DEVELOPMENT OF ATHEROSCLEROSIS

- Collagen produced by smooth muscle cells = advanced/raised fibrolipid plaque

- Advanced plaque can grow slowly (encroaching on lumen) or become unstable (thrombosis with obstruction = complicated plaque)
DEVELOPMENT OF ATHEROSCLEROSIS

- Plaque thrombosis caused by:
  - Superficial endothelial injury exposing connective tissue with platelet adhesion = superficial thrombus
  - Deep endothelial fissuring of advanced plaque allowing blood to enter inside the plaque (highly thrombogenic environment) = thrombus within plaque

- 50% reduction in luminal diameter results in haemodynamically significant stenosis
  - Smaller distal arteries/arterioles become maximally dilated
  - Any increase in myocardial demand = ischaemia
COMPLICATIONS

ATHEROSCLEROSIS

CORONARY HEART DISEASE

CHD is the most common form of heart disease

- Epidemiology
  - Single most important cause of premature death in Europe, South America, Australia and New Zealand
  - In UK 1 in 3 men and 1 in 4 women die from CHD
CORONARY HEART DISEASE

<table>
<thead>
<tr>
<th>CAD results in variety of clinical situations</th>
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<tbody>
<tr>
<td>• Stable angina pectoris (AP)</td>
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<tr>
<td>• Unstable AP (UAP)</td>
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<tr>
<td>• Myocardial Infarction (MI) = advanced plaque with total occlusive thrombus</td>
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<tr>
<td>• Heart failure</td>
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<tr>
<td>• Arrhythmia</td>
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<td>• Sudden death</td>
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STABLE ANGINA

Angina Pectoris
Symptom complex of chest pain, discomfort due to transient myocardial ischaemia

- **Causes**
  - Coronary atheroma is the most common cause
  - Aortic valve disease
  - Hypertrophic cardiomyopathy
STABLE ANGINA

Clinical features

- Central chest pain, discomfort or breathlessness precipitated by exertion or stress
- Promptly relieved by rest

Investigation

- Resting ECG, exercise ECG
- Coronary arteriography
STABLE ANGINA

Management

- Careful assessment
- Risk factors management
- Control symptoms
  - Antiplatelet therapy - Low dose aspirin
  - Antianginal drugs – nitrates, beta-blockers
  - Percutaneous coronary interventions (PCI) - Stents
  - Percutaneous trans-coronary arteriogram (PTCA)
  - Coronary artery bypass graph (CABG)

Prognosis

- Depends on number of diseased vessels, degree of left ventricular dysfunction
- Single vessel and good LV function (5 yr survival > 90%)
Stent insertion

Philadelphia, U.S.A. Walters Kluwer Health - Lippincott, Williams & Wilkins
ANGIOPLASTY

Narrow before angioplasty

Open after angioplasty

http://www.vascular.co.nz/angioplastyrev%20copy.jpg
UNSTABLE ANGINA

Characterised by new onset or rapidly worsening angina, angina on minimal exertion or angina at rest

**Diagnosis**
- Evaluation of ECG
- Biochemical markers for cardiac damage

**Management**
- Admission to hospital due to risk of myocardial infarction (MI)
- Bed rest, antiplatelet therapy
- Same as for stable angina
ANGINA PAIN

MYOCARDIAL INFARCTION

Definition
- Result of sudden decrease or interruption to arterial blood flow to the myocardium

Pathophysiology
- Coronary atheroma with plaque rupture + superimposed thrombus
- Myocardial necrosis = permanent myocardial damage

Causes
- Atheroma (predominantly) as above
# MYOCARDIAL INFARCTION

## Clinical Features

- **Key CF= chest pain**
  - severe, constant pain, unrelieved with rest/nitrates
- Nausea/vomiting/sweating/breathlessness
- Anxiety and fear of impending death
- Collapse and syncope
- Sudden death
MYOCARDIAL INFARCTION

Investigations
- ECG
- Biochemical markers – Creatine Kinase (CK); Troponin
- Chest X ray
- Echocardiography

Treatment
- Immediate access to medical care and defibrillation facility
- Emergency Rx (aspirin, thrombolysis, pain relief (morphine)
- CCU, bed rest
- Identify & Correct Risk Factors
- Subsequent Medical Rx – aspirin, B-blockers, ACEI, statins)
- Ongoing assessment
## MYOCARDIAL INFARCTION

### Complications

- Arrhythmias
- Post infarct ischaemia
- Acute circulatory failure
- Mechanical complications
  - Papillary muscle damage – valve lesions
  - Rupture of interventricular septum – VSD
  - Rupture of ventricle
- Embolism
- Heart failure
- Ventricular aneurysm
MYOCARDIAL INFARCTION

Prognosis
- Mortality 25% without medical care
- 50% of death from MI occur within 24 hrs
- 40% die within 1 month
- Those survive an acute attack – 5 yr survival 75%

Differential Diagnosis
- As by main symptoms
- Cardio/pulmonary (CP) (CVS vs. other)
- MI vs. Upper abdominal pathologies
Readings and Resources

Resources:

- **Set Textbooks:**

- **Additional textbooks:**
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