Title: Pathophysiology of Ischemic Heart Disease (IHD)

1. Introduction

- **Ischemic Heart Disease (IHD)** refers to a condition where the heart muscle (myocardium) receives inadequate oxygen supply, primarily due to reduced coronary blood flow.
- Leading cause of morbidity and mortality worldwide.
- Main manifestations:
    - **Angina Pectoris** (transient, reversible ischemia)
    - **Myocardial Infarction (MI)** (irreversible necrosis)

2. Anatomy of Coronary Circulation

- Heart is supplied by:
    - **Left coronary artery (LCA)**: branches into LAD and circumflex
    - **Right coronary artery (RCA)**
- Coronary perfusion occurs mainly during diastole.
- Obstruction of these arteries impairs oxygen delivery, leading to ischemia or infarction.

3. Atherosclerosis – The Root Cause

A **chronic inflammatory process** affecting the arterial wall, progressing through:

1. **Endothelial injury**
2. **LDL accumulation** → oxidation
3. **Monocyte infiltration** → foam cells
4. **Fatty streak** formation
5. **Fibrous plaque** formation
6. **Plaque rupture or erosion** → thrombus formation

Leads to narrowing or occlusion of coronary arteries.

4. Angina – Reversible Ischemia

Pathophysiology:

- Oxygen demand > supply → ischemia
- Anaerobic metabolism → lactic acid → chest pain
- Pain relieved by rest or nitrates

Types of Angina:

Type	Characteristics
Stable angina	Predictable, triggered by exertion, fixed stenosis
Unstable angina	Unpredictable, at rest, indicates plaque rupture
Variant (Prinzmetal)	Due to coronary vasospasm, not atherosclerosis
Microvascular angina	Small vessel dysfunction, often in women

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5. Myocardial Infarction – Irreversible Ischemia

- **Complete occlusion** of coronary artery → sustained ischemia → necrosis
- Begins 20–40 minutes after obstruction
- Classified as:
    - **STEMI**: full thickness (transmural) infarction
    - **NSTEMI**: partial thickness (subendocardial)

6. Pathological Zones of Infarction

Zone	Status
Infarction	Irreversible necrosis
Injury	Damaged, salvageable if reperfused early
Ischemia	Viable but at risk

7. Cellular & Systemic Effects of Ischemia

Cellular events:

- ↓ Oxygen → ↓ ATP
- Ion pump failure → ↑ intracellular Ca^{2+}
- Membrane rupture → cell death
- Enzyme release: troponins, CK-MB

Systemic responses:

- **Sympathetic activation** → ↑ HR, BP
- Inflammatory cytokine release
- Platelet activation → thrombosis
- Risk of: arrhythmias, shock, hypotension

8. Clinical Features

Condition	Symptoms
Angina	Chest pressure/discomfort, triggered by stress/exertion, relieved by rest
MI	Crushing chest pain (>20 min), nausea, sweating, dyspnea, fatigue; may be atypical in elderly, diabetics, women

9. Diagnosis

Biomarkers:

- **Troponin I/T**: Highly specific and sensitive
- **CK-MB**: Useful in reinfarction
- **Myoglobin**: Early marker, nonspecific

ECG findings:

- **ST depression/T wave inversion** → ischemia (NSTEMI)
- **ST elevation** → infarction (STEMI)
- **Pathological Q waves** → late sign of infarction

Imaging:

- **Echocardiography**: wall motion abnormality
- **Stress testing**: assesses inducible ischemia
- **Coronary angiography**: visualizes artery occlusion

10. Complications of MI

Type	Examples
Arrhythmias	Ventricular fibrillation, heart block
Heart failure	Due to pump dysfunction
Cardiogenic shock	Severe hypotension, organ hypoperfusion
Mechanical complications	Ventricular rupture, septal defect, papillary muscle rupture
Pericarditis	Inflammation of pericardial lining
Aneurysm	Thinning and dilation of infarcted wall

11. Long-Term Consequences of IHD

- **Chronic heart failure**
- **Ventricular remodeling**
- **Risk of recurrent MI**
- Requires:
    - Lifestyle changes
    - Medication (antiplatelets, statins, beta-blockers, ACE inhibitors)
    - Regular follow-up

12. Psychosocial and Neural Factors

- Depression and stress worsen prognosis
- Sympathetic overactivation increases ischemic risk
- Heart-brain interaction is bidirectional
- Psychological therapy can improve long-term outcomes

13. Summary

- IHD results from oxygen supply-demand mismatch, primarily due to atherosclerosis.
- Angina is reversible; MI is irreversible.
- Timely diagnosis, intervention, and prevention are key.
- Multidisciplinary care including medical, interventional, and psychosocial strategies improves survival and quality of life.