


# Cardiovascular Pathology

## Ischemic Heart Disease (IHD)

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### Definition

Reduced coronary blood flow → myocardial ischemia →  
may progress to infarction 

Usually caused by:

- Atherosclerotic plaque narrowing
  - Acute thrombosis
  - Coronary vasospasm
- 

## Angina

### Definition

## Chest pain due to myocardial ischemia WITHOUT necrosis

⚠ Important:

- Ischemia = reversible
  - Infarction = irreversible cell death
- 

### Types of Angina

Type	Main Mechanism	Troponins	ECG Changes
Stable	Fixed atherosclerotic narrowing	Normal	± ST depression
Vasospastic	Coronary vasospasm	Normal	Transient ST elevation
Unstable	Partial thrombosis	Normal	ST depression/T inversion

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### Stable Angina


 Cause

Usually due to:

★  $\geq 70\%$  atherosclerotic narrowing



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### Pathophysiology Flowchart

Fixed coronary stenosis  $\rightarrow$  Increased  $O_2$  demand during exertion   $\rightarrow$  Coronary supply insufficient  $\rightarrow$  Myocardial ischemia  $\rightarrow$  Chest pain

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### Clinical Features

- Exertional chest pain
  - Predictable pattern
  - Relieved by:
    - Rest 
    - Nitroglycerin 
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### ECG

- May show ST depression

Why? → Subendocardial ischemia

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## 2 Vasospastic Angina (Prinzmetal Variant)

 Cause

Transient coronary artery vasospasm

 NOT primarily due to atherosclerosis


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 Pathophysiology Flowchart

Coronary vasospasm → Temporary coronary occlusion  
→ Transmural ischemia → Chest pain at rest





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 ECG

 Transient ST elevation

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## Risk Factors & Triggers

Factor	Association
Smoking 	Strong risk factor
Cocaine 	Vasospasm trigger
Alcohol 	Trigger
Triptans 	Trigger

### Important:

- HTN and hypercholesterolemia are NOT classic associations
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### Treatment

- Calcium channel blockers
  - Nitrates
  - Smoking cessation 
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## 3 Unstable Angina

## Cause

Partial coronary thrombosis

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## Pathophysiology Flowchart

Plaque rupture → Platelet aggregation → Incomplete coronary occlusion → Reduced coronary perfusion → Ischemia WITHOUT necrosis

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## Clinical Features

- Chest pain at rest
  - OR
  - Increasing frequency/severity ("crescendo angina")
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## ECG

- ST depression


- T-wave inversion

 KEY DISTINCTION

Feature	Unstable Angina	NSTEMI
Troponins	Normal	Elevated

 Stable vs Unstable vs NSTEMI vs STEMI

Feature	Stable Angina	Unstable Angina	NSTEMI	STEMI
Pain	Exertion	Rest/minimal exertion	Rest	Rest
Troponins	Normal	Normal	↑	↑
Infarction	None	None	Subendocardial	Transmural
ECG	± ST depression	ST depression/T inversion	ST depression/T inversion	ST elevation

 Coronary Steal Syndrome

## Core Principle

Occurs during pharmacologic vasodilation stress testing

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## Pathophysiology Flowchart

Distal coronary stenosis present → Diseased vessels already maximally dilated → Vasodilator given (dipyridamole/regadenoson) → Normal vessels dilate further → Blood preferentially flows to healthy regions → Ischemia worsens in stenosed territory

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## Key Drugs

- Dipyridamole
- Regadenoson

Used in:

Pharmacologic stress tests

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# ⚡ Sudden Cardiac Death (SCD)

## 📌 Definition

Unexpected cardiac death within:

★ 1 hour of symptom onset

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## 🌟 Most Common Mechanism

Lethal arrhythmia

Especially:

- Ventricular fibrillation
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## ⚠️ Major Associations

Condition	Importance
CAD	Up to 70% of cases
Hypertrophic cardiomyopathy	Young athletes

Dilated cardiomyopathy	HF-related
Long QT syndrome	Channelopathy
Brugada syndrome	Channelopathy

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## Prevention

Implantable cardioverter-defibrillator (ICD)

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## Chronic Ischemic Heart Disease

### Definition

Progressive heart failure due to chronic ischemic myocardial damage

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### Important Concepts

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## 1] Myocardial Hibernation

### Definition

Chronic ischemia causing:

- Reduced LV function
  - BUT potentially reversible after revascularization
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## 2] Myocardial Stunning

### Definition


Temporary LV dysfunction after brief acute ischemia

 Reversible

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## Myocardial Infarction (MI)

### Main Cause

 Plaque rupture → acute thrombosis

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## Pathophysiology Flowchart

Atherosclerotic plaque rupture → Platelet activation →  
Acute thrombus formation → Coronary occlusion →  
Myocyte necrosis 

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## Cardiac Biomarkers

Diagnostic markers:

- Troponins 
  - CK-MB
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## NSTEMI vs STEMI

Feature	NSTEMI	STEMI
Infarct Type	Subendocardial	Transmural
Layers Involved	Inner 1/3	Full thickness

ECG	ST depression/T inversion	ST elevation + Q waves
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## Why Subendocardium Is Vulnerable


The subendocardium:

- Is farthest from epicardial blood supply
- Experiences highest wall stress

→ Most susceptible to ischemia 

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## MI Pathogenesis Flowchart

Coronary thrombosis → Ischemia → ATP depletion →  
 Loss of contractility → Cell death → Inflammation →  
 Scar formation 

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## High-Yield ECG Concepts

ECG Finding	Meaning
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ST depression	Subendocardial ischemia
ST elevation	Transmural ischemia
Pathologic Q waves	Prior transmural infarction

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### Exam Pearls

- Stable angina → exertional pain relieved by rest
  - Vasospastic angina → transient ST elevation
  - Unstable angina → no troponin elevation
  - NSTEMI = subendocardial infarction
  - STEMI = transmural infarction
  - VFib = most common sudden cardiac death mechanism
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### High-Yield Summary


- Stable Angina = fixed stenosis + exertional ischemia
- Vasospastic Angina = coronary spasm + transient ST elevation

- Unstable Angina = partial thrombosis WITHOUT necrosis
  - NSTEMI = subendocardial infarction
  - STEMI = transmural infarction
  - Sudden Cardiac Death = lethal ventricular arrhythmia
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## Evolution of Myocardial Infarction (MI)


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### Core Concept

Myocardial infarction evolves through predictable morphologic and histologic stages over time 




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### Commonly Occluded Coronary Arteries


Artery	Frequency
LAD	 MOST COMMON

RCA	Second
Circumflex	Third

## Classic Symptoms of MI

Symptom	Mechanism
Severe retrosternal chest pain 	Myocardial ischemia
Left arm/jaw pain	Referred pain
Diaphoresis	Sympathetic activation
Nausea/vomiting 	Vagal stimulation
Dyspnea 	LV dysfunction
Fatigue	Reduced cardiac output

## Big Picture Flowchart

Coronary artery occlusion → Myocardial ischemia →  
 Coagulative necrosis (20-40 min) → Acute inflammation  
 (1-3 days) → Macrophage cleanup → Granulation tissue  
 formation → Scar formation 

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## Timeline of MI Evolution

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### 0-24 Hours

#### Gross Appearance

- Dark mottling
  - Pale with tetrazolium stain
- 

#### Histology

Early:

- Wavy fibers (0-4 hr)

Later:

- Early coagulative necrosis
- Edema

- Hemorrhage
- 


### Why Wavy Fibers Occur

Dead myocytes lose structural integrity → Pulled by adjacent viable myocardium → Wavy appearance

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### Reperfusion Injury

#### Pathophysiology Flowchart

Blood flow restored → Free radical generation  →  $\text{Ca}^{2+}$  influx ↑ → Hypercontraction of myofibrils → Additional injury

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#### Histology

- Dark eosinophilic contraction bands
-

## Major Complications (0-24 hr)

Complication	Mechanism
Ventricular arrhythmias ⚡	Electrical instability
Heart failure ❤️	Loss of contractility
Cardiogenic shock	Massive LV dysfunction

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## 1-3 Days

### Gross Appearance

- Hyperemia (red appearance)
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### Histology

- Extensive coagulative necrosis
  - Neutrophilic infiltration ★
- 

### Key Principle

Neutrophils dominate first

Acute inflammation phase 

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 Major Complication


Postinfarction fibrinous pericarditis

 Mechanism

Inflammation extends to pericardium

 Features

- Sharp pleuritic chest pain
  - Friction rub
- 

 3-14 Days

 Gross Appearance

- Yellow-brown soft center

- Hyperemic border
- 

## Histology

Early:

- Macrophages dominate 

Later:

- Granulation tissue forms
- 

## MOST DANGEROUS PERIOD

Why? → Macrophages digest dead tissue → Wall becomes structurally weak


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## Major Complications

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## 1] Free Wall Rupture

### Mechanism

Macrophage-mediated wall degradation → LV rupture →  
Cardiac tamponade 

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## 2] Papillary Muscle Rupture

Result:

Acute severe mitral regurgitation

### Presentation

- Pulmonary edema
  - New systolic murmur
- 

## 3] Interventricular Septal Rupture

Result:

Left → right shunt

## Presentation

- Acute HF
  - Harsh murmur
- 


## 4 LV Pseudoaneurysm

### Definition

Contained rupture of ventricular wall

 High risk of rupture

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 2 Weeks → Several Months

### Gross Appearance

- Gray-white scar
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### Histology

- Dense collagen scar formation 
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## Late Complications

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### 1 Dressler Syndrome (Postcardiac Injury Syndrome)

#### Mechanism

Autoimmune pericarditis after MI

#### Features

- Fever
  - Pericarditis
  - Friction rub
- 

### 2 Chronic Heart Failure

Due to loss of functional myocardium

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### 3 Arrhythmias

Scar tissue disrupts conduction ⚡

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### 4 True Ventricular Aneurysm

 Definition

Scarred ventricular wall bulges outward

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 Important

- LOW risk of rupture
  - HIGH risk of mural thrombus
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### Pseudoaneurysm vs True Aneurysm

Feature	Pseudoaneurysm	True Aneurysm
Cause	Free wall rupture contained by pericardium	Scarred ventricular wall bulging

Rupture Risk	HIGH 🚒	Low
Wall Composition	Pericardium/fibrous tissue	Myocardium + scar

## 🧠 Inflammatory Cell Timeline

Time	Dominant Cells
1-3 days	Neutrophils 🚒
3-14 days	Macrophages 🧹
Weeks onward	Fibroblasts/collagen 🩹


## 🧠 Easy Memory Flowchart

MI occurs → Necrosis → Neutrophils → Macrophages  
 → Granulation tissue → Scar


## ⚠️ MUST-KNOW EXAM PEARLS

🔥 0-24 hr

- Arrhythmias = most common cause of death
- 

 1-3 days

- Neutrophils dominate
- 

 3-14 days

- Greatest rupture risk 
- 

 Weeks later

- True ventricular aneurysm
  - Dressler syndrome
- 

### High-Yield Timing Summary

Time	Histology	Major Complication
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0-24 hr	Wavy fibers	Arrhythmia
1-3 days	Neutrophils	Fibrinous pericarditis
3-14 days	Macrophages	Rupture
Weeks-months	Scar	True aneurysm

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## ♥ High-Yield Summary

Early MI = Necrosis + arrhythmias

Intermediate MI = Macrophages weaken wall → rupture risk

Late MI = Scar formation → aneurysm/HF




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## 🌟 Diagnosis of Myocardial Infarction (MI)

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### 📌 Core Principle

Diagnosis of MI relies on:

1. Clinical presentation 
  2. ECG changes 
  3. Cardiac biomarkers 
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 Most Important Rule

 In the first 6 hours:

 ECG is the gold standard

Why? → Biomarkers may not yet be elevated early after infarction

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## Cardiac Biomarkers

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Troponin I — 

 Features

Property	Value
Begins rising	~4 hours
Peaks	~24 hours
Returns to normal	7-10 days

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### Why Troponin Is Best

- Highly sensitive
  - Highly specific for myocardial injury 
- 

### Pathophysiology Flowchart

Myocyte necrosis → Cell membrane breakdown → Troponin released into blood → Detectable serum elevation

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### High-Yield Point

Troponin remains elevated for:

★ 7-10 days

Useful for:

- Detecting recent MI

BUT:

- Not ideal for diagnosing reinfarction
- 

## 2 CK-MB

### Features

Property	Value
Begins rising	6-12 hours
Peaks	16-24 hours
Returns to normal	~48 hours

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### Key Clinical Use

★ Best marker for reinfarction

Why? → Returns to baseline quickly

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⚠ Important Limitation

CK-MB is:

- Mostly cardiac
- BUT
- Can also rise with skeletal muscle injury

→ Less specific than troponin

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Troponin vs CK-MB

Feature	Troponin I	CK-MB
Specificity	Higher ★	Lower
Rise	~4 hr	6-12 hr
Returns to normal	7-10 days	48 hr

Reinfarction diagnosis	Poor	Best ★
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## ECG Changes in MI

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### 🔥 ST Elevation

Indicates:

★ STEMI (transmural infarction)

Mechanism:

Transmural ischemia → Current shifts during repolarization → ST elevation

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### ▼ ST Depression

Indicates:

NSTEMI or subendocardial ischemia

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## Hyperacute T Waves

Earliest ECG change

- Tall, peaked T waves
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## T-Wave Inversion

Indicates:

- Ischemia/reperfusion
- 

## Pathologic Q Waves

Indicates:

- Evolving or old transmural infarction

Mechanism:

Dead myocardium becomes electrically silent

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 Poor R-Wave Progression

May indicate:

- Prior anterior MI
- 

 ECG Findings Summary

ECG Finding	Significance
Hyperacute T waves	Earliest ischemia
ST elevation	STEMI/transmural infarct
ST depression	NSTEMI/subendocardial ischemia
T-wave inversion	Ischemia
Q waves	Old/evolving transmural MI

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 ECG Localization of STEMI

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## 1] Anteroseptal MI

 Artery: LAD

 Leads

- V1-V2
- 


## 2] Anteroapical MI

 Artery: Distal LAD

 Leads

- V3-V4
- 

## 3] Lateral MI

 Artery: Left circumflex (LCX)

 Leads

- I, aVL
- 

#### 4 Inferior MI

 Artery: RCA

 Leads

- II, III, aVF
- 

#### 5 Posterior MI

 Artery: PDA (posterior descending artery)

 Findings

- V7-V9 ST elevation
- OR
- ST depression in V1-V3
- Tall R waves

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## Easy Localization Flowchart

Inferior MI: II, III, aVF → RCA

Septal/Anterior MI: VI-V4 → LAD

Lateral MI: I, aVL → LCX

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## High-Yield ECG Patterns

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Inferior MI

Think:

- RCA occlusion
  - Possible RV infarction
- 

Anterior MI

Think:

- LAD occlusion
  - High mortality risk ⚠️
- 

Posterior MI

Clue:

ST depression in VI-V3 is actually:

“Hidden posterior ST elevation”

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## Timeline of Diagnostic Findings



Early (<6 hr)

- ECG changes most important
- 



Hours Later

- Troponin rises
- 


 Days Later

- Troponin remains elevated
- 

 Memory Trick

Marker	Use
Troponin	BEST overall marker ★
CK-MB	Reinfarction ★

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 Exam Pearls

- Troponin rises earlier than CK-MB
- CK-MB normalizes faster
- ST elevation = transmural infarction
- ST depression = subendocardial ischemia
- Inferior MI → RCA

- Anterior MI → LAD
- 

## High-Yield Summary

Troponin I = most specific marker 

CK-MB = best for reinfarction 

STEMI = ST elevation + transmural infarction

NSTEMI = ST depression + subendocardial infarction

### ECG Localization

- LAD → anterior/septal
  - RCA → inferior
  - LCX → lateral
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-> The End <-