Category	Details
Lateral MI & ECG Findings	• ST Elevations in leads I & aVL suggest an acute lateral MI. • Left circumflex artery supplies the lateral aspect of the LV. • ECG localizes infarction: • **ST elevation** = acute MI • **Prominent Q waves** = old MI • Chest leads V5-V6 may also show ST elevation due to their lateral placement.
Coronary Arteries & Infarction Patterns	
Coronary Artery	**Supplies**
Left Circumflex (LCx)	Lateral aspect of LV
Left Anterior Descending (LAD)	Anterior LV, septum
Right Coronary Artery (RCA)	RV, inferior LV
Left Main Coronary Artery	LAD & Circumflex Artery
Inferior Wall Blood Supply & Coronary Dominance	
Right-Dominant Circulation (85%)	Inferior LV wall supplied by RCA → PDA
Left-Dominant Circulation (10%)	Inferior LV wall supplied by LCx → PDA
Codominant Circulation (5%)	Both RCA & LCx supply the inferior LV wall
Fibrinolytic Therapy in STEMI	• Used when **PCI is unavailable** • Agents: Alteplase, Tenecteplase, streptokinase • Dissolve fibrin clots & restore myocardial perfusion • **Complications**: ↑ risk of bleeding, contraindicated in recent hemorrhagic stroke
Inferior Wall MI with RV Involvement	**Key Findings Suggesting RV MI**: • Severe hypotension • Prominent jugular venous distension • Clear lungs (absence of pulmonary edema)
Right Ventricular MI Pathophysiology	• **RV dysfunction** leads to ↑ CVP • **Reduced RV output** decreases left atrial blood flow, lowering PCWP • **Hypotension** occurs due to low LV filling • **Increased SVR** compensates for low cardiac output
Beta Blocker Use in AMI	**Indications**: Reduce myocardial O2 demand, lower HR, decrease contractility, improve survival **Contraindications**: Bradycardia, heart block, hypotension, acute decompensated heart failure, asthma/COPD (for non-selective beta-blockers)

Category	Details
	Preferred Beta-Blockers: Cardioselective (metoprolol, atenolol, bisoprolol, nebivolol)
Myocardial Fibrosis Post-MI	
	Phases of Fibrosis: 1. **Inflammation** Phase (**Hrs to Days**): Neutrophils & macrophages remove necrotic tissue, release cytokines 2. **Proliferation** Phase (**Days to Wks**): Fibroblast proliferation, TGF-β stimulates collagen deposition 3. **Remodeling** Phase (**Wks to Months**): **Collagen cross-linking**, dense scar formation
Myocardial Ischemic Injury & Microscopic Changes	
Time-Based Light Microscopy Findings:	
0-4 hours	No visible changes
4-12 hours	Wavy fibers, elongated myocytes
12-24 hours	Myocyte hypereosinophilia, pyknosis (shrunken nuclei)
1-3 days	*Coagulative* necrosis, neutrophil infiltration
3-7 days	Dead neutrophils, macrophage infiltration at infarct border
7-10 days	Robust macrophage phagocytosis, granulation tissue formation
10-14 days	Well-developed granulation tissue, neovascularization
2-8 weeks	Progressive collagen deposition, scar formation
Mechanical Complications of MI	
Complication	**Time Course**
Papillary Muscle Rupture	3-5 days
Interventricular Septum Rupture	3-5 days
Free Wall Rupture	5 days - 2 wks
Left Ventricular Aneurysm	Wks - months
Right Ventricular Infarction & Shock	Hemodynamic Findings: • High RAP (right atrial pressure) • Low PCWP (left preload) • Low cardiac output • Severe hypotension & systemic hypoperfusion

Category	Details
Right Ventricular Protection in MI	RV function generally recovers after MI, due to: • Lower muscle mass & afterload reducing O2 demand at rest. • Lower systolic pressure (≤25 mmHg) enabling coronary perfusion during both systole & diastole. • More developed collateral circulation, allowing better perfusion from the left coronary arteries. • Heightened ischemic preconditioning, which improves O2 extraction & cellular adaptation to ischemia. • Acute RV infarction may cause transient contractile dysfunction, leading to hypotension & hemodynamic instability, but long-term RV dysfunction is rare.
STEMI (ST-Elevation MI) Pathophysiology	
	Cause: Atherosclerotic plaque rupture, thrombus formation, complete coronary occlusion
	Symptoms: Persistent substernal chest pain, not relieved by nitrates or rest
	ECG Changes: Peaked T waves, ST elevations, Q wave formation
Histological Changes Over Time in MI	
Time Post-MI	**Histological Findings**
0-4 hours	Minimal change
4-12 hours	Early coagulation necrosis, wavy fibers
12-24 hours	Coagulative necrosis, contraction band necrosis
1-5 days	Neutrophilic infiltrate
5-10 days	Macrophage phagocytosis
10-14 days	Granulation tissue, neovascularization
2 weeks - 2 months	Collagen deposition, scar formation
STEMI & Fibrinolytic Therapy Complications	
	Fibrinolytic Therapy Risks: **Intracerebral Hemorrhage (ICH):** Most serious complication, signs: neurological deterioration, asymmetric pupils, irregular breathing
Cardiac Ischemia & Ion Pump Failure	Key Pathophysiology: • ATP depletion leads to Na^+/K^+-ATPase & Ca^{2+}-ATPase pump failure

Category	Details
	• Intracellular Na^+ & Ca^{2+} accumulation causes increased solute concentration & water influx
Right Ventricular Infarction & Shock	Hemodynamic Findings: • High RAP (right atrial pressure) • Low PCWP (left preload) • Low cardiac output • Severe hypotension & systemic hypoperfusion