COMPLICATIONS OF MYOCARDIAL INFARCTION

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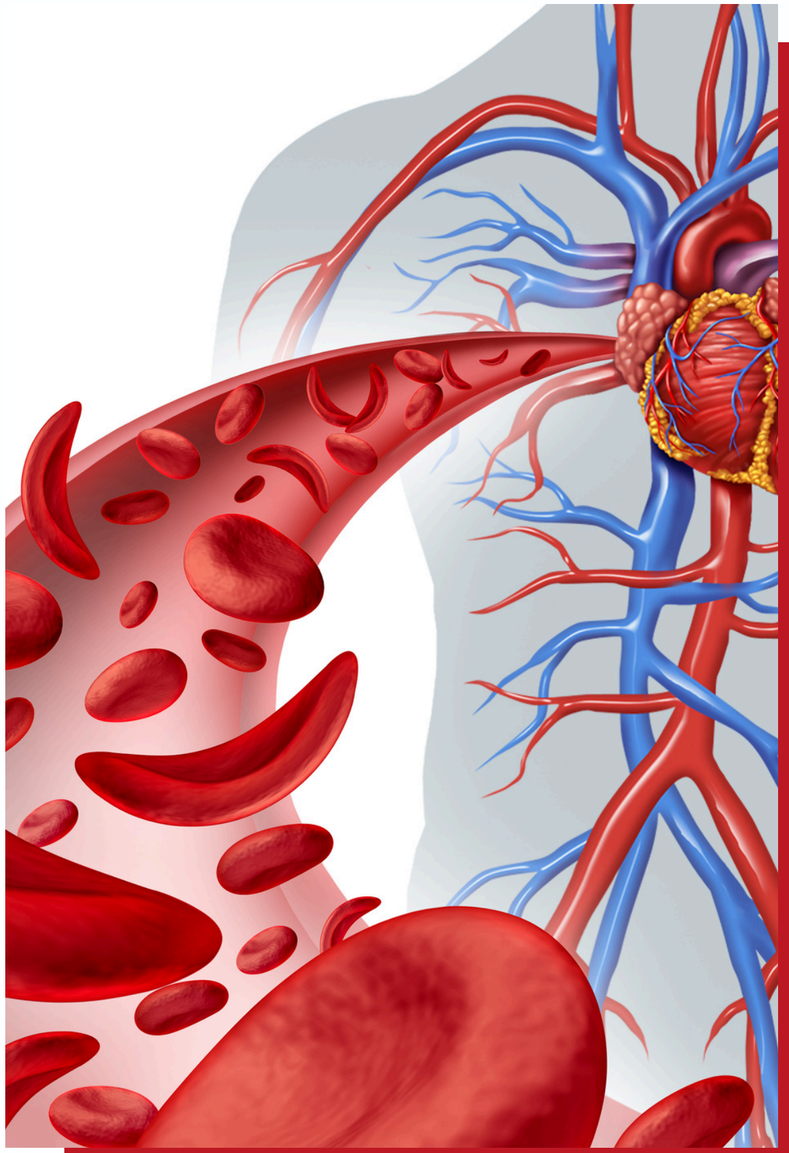
CONTENT



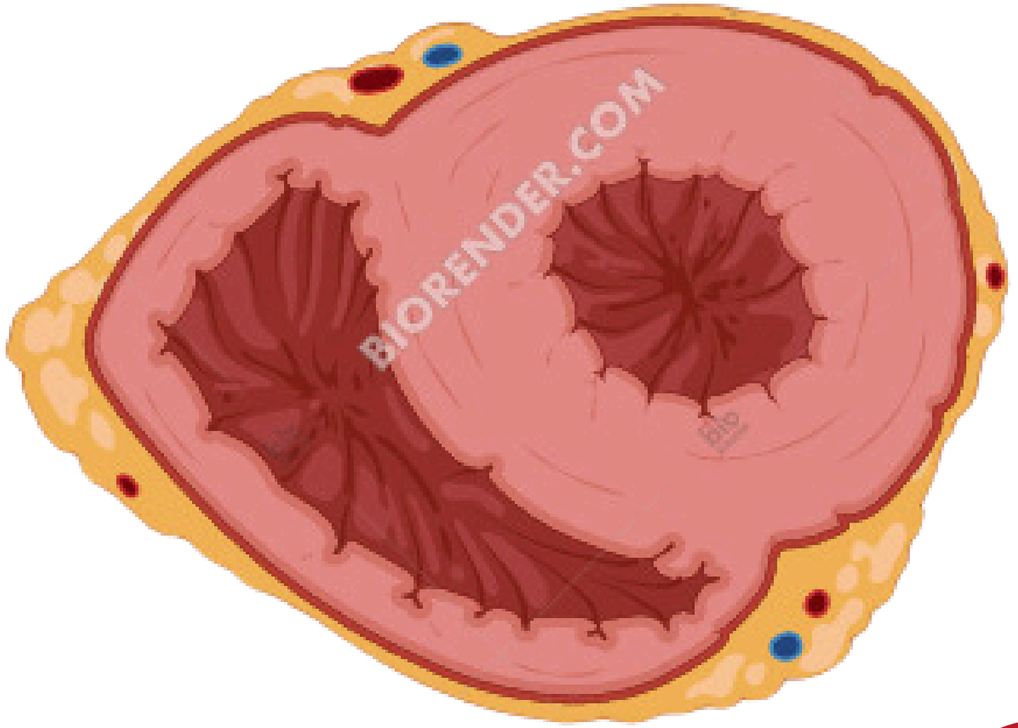
INTRODUCTION

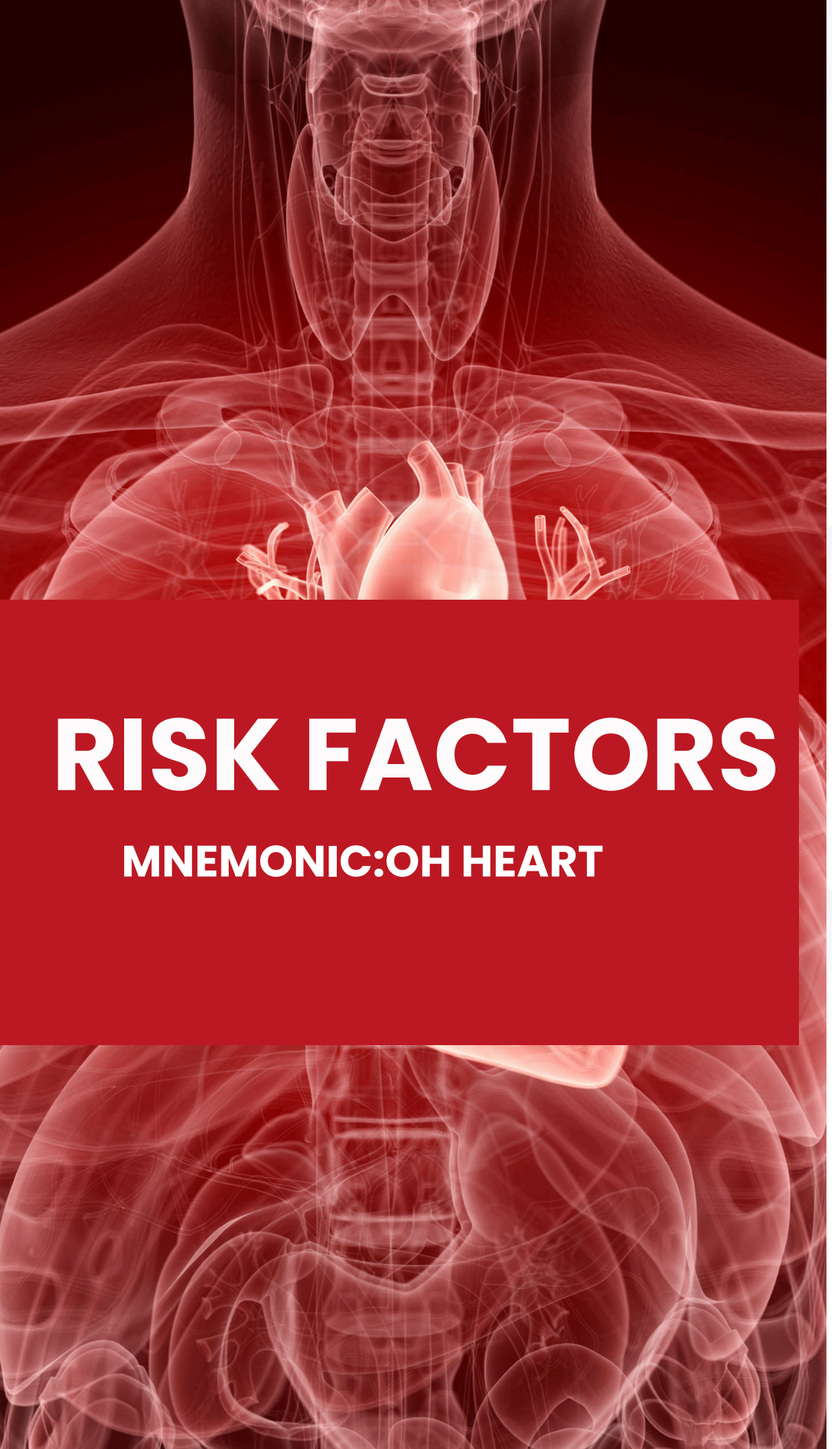
Myocardial infarction (MI) refers to ischemic necrosis of myocardial tissue or acute myocardial injury with clinical and diagnostic evidence of acute ischemia.

The most common underlying cause is **coronary artery disease.**

MI manifests clinically with acute coronary syndrome (ACS), a potentially **lethal condition.**

Diagnosis is based on typical clinical features, ECG findings, and elevation of cardiac biomarkers
All patients suspected of having ACS should be **considered for emergency revascularization**





RISK FACTORS

MNEMONIC:OH HEART

Any condition that causes occlusion of the coronary arteries, reduces myocardial oxygen supply, or increases oxygen demand can potentially lead to myocardial ischemia and infarction

Risk factors is

1. **O** - Obesity (BMI ≥ 30)
2. **H** - Hypertension (most common risk factor)
3. **H** - Hyperlipidemia (↑LDL,↓HDL)
4. **E** - EtOH (alcohol abuse)
5. **A** - Age (≥ 65, non-modifiable)
6. **R** - Relatives (repeating for emphasis, or CAD risk in family)
7. **T** - Tobacco use

UNSTABLE ANGINA

BLOOD FLOW

Patients with unstable angina also have subendocardial ischemia

PATHOPHYSIOLOGY

Unstable plaque, with a weak fibrous cap that is prone to rupture
—›Fatty center of the plaque is exposed; platelets attach to the center, leading to thrombus formation

▼Occludes ≥ 90% of the lumen (near total occlusion)

CLINICAL FEATURES

Chest pain occurs at rest and worsens with exertion

SUBENDOCARDIAL INFARCTS / NSTEMI

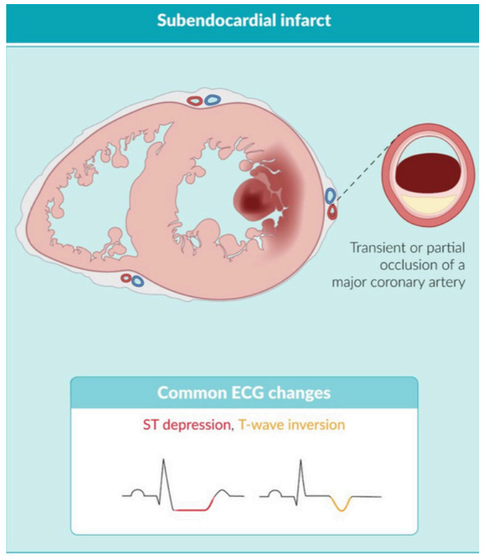
PATHOPHYSIOLOGY

Unstable plaque, with a weak fibrous cap that is prone to rupture —>Fatty center of the plaque is **exposed**; platelets attach to the center, leading to **thrombus formation**

▼ **Occludes ≥ 90%** of the lumen (**near total occlusion**)

BLOOD FLOW

Chest pain occurs at rest and **worsens with exertion**

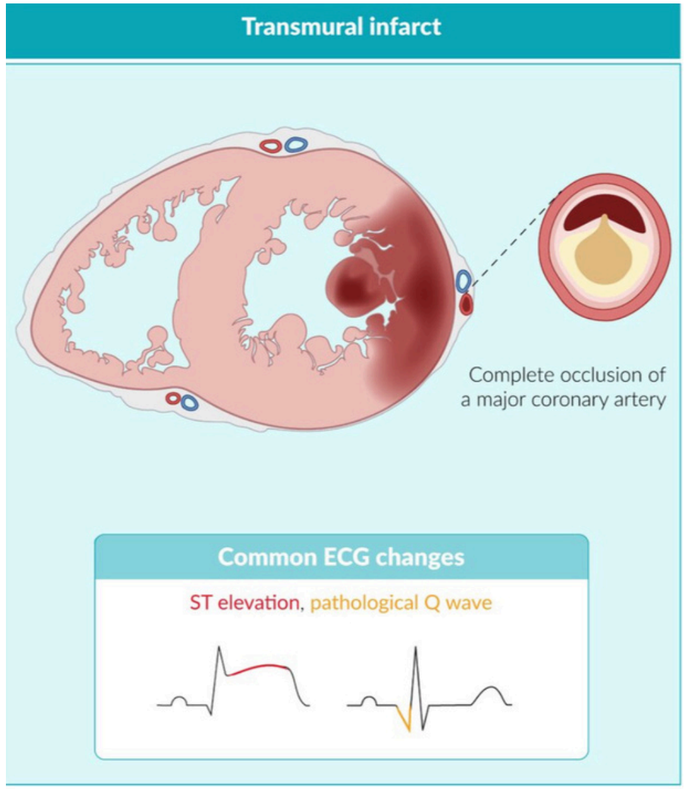


CLINICAL FEATURES

In subendocardial infarction, there is significant **reduction of blood flow leading to infarction (cell death)**

Infarction results from at least **30 minutes of unreversed ischemia**

TRANSMURAL INFARCT/STEMI



Patho physiology

Unstable plaque, with a weak fibrous cap that is prone to rupture Fatty center of the plaque is exposed; platelets attach to the center, leading to thrombus formation
Occludes100% of the lumen (total occlusion)

Clinical features

Chest pain occurs at rest
Severe chest pain with exertion

Blood Flow

There is complete loss of oxygen supply The entire myocardium (from the endocardium to the epicardial portion) will infarct ("transmural")

Complication in THE FIRST 24 HOURS

Sudden cardiac death

Ventricular fibrillation can result in Sudden Cardiac Death

Acute heart failure → Pulmonary Edema

- Blood backs up into the pulmonary circulation leading to flash pulmonary edema
- Shortness of breath, hypoxia

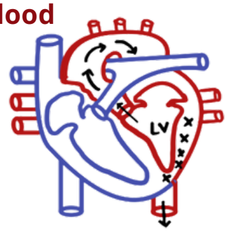
o reflex tachycardia to maintain a decent cardiac output

Cardiogenic shock

- If there is a significant infarct in a major vessel (e.g LADA occlusion) especially in the left ventricle

→ The heart would not be able to push blood into the systemic circulation resulting in hypotension

- Hypotension causes cold extremities due to hypoperfusion
- This can progress to cardiogenic shock



- HOTN → Shock
- Cold Ext.
- Flash Pulmonary Edema
- Reflex ↑HR

Arrhythmia

- A common cause of death in MI patients in the first 24 hours

▼Ventricular tachyarrhythmias : If there is infarct in a major vessel (LADA OR LCX) re entrance circuit VT → VF → SCD →

▼Atrioventricular block (e.g., complete heart block):Right coronary artery supplies the SA and AV node

- If the right coronary artery is occluded (as in IHD) SA and AV node (especially the AV node) do not receive proper blood supply

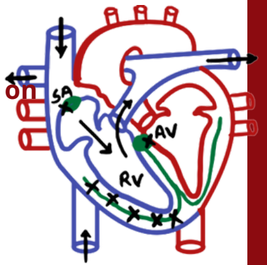
→ This affects the conduction through the heart

→ Can lead to decrease in heart rate

- Sinus bradycardia
- AV block (it can be 1st, 2nd or 3rd degree, it depends on the severity of the AV dysfunction)

▼Asystole

▼Atrial fibrillation



1-3 DAYS POST-INFARCTION

EARLY INFARCT-ASSOCIATED PERICARDITIS

Typically occurs within the first week of a large infarct close to the pericardium

CLINICAL FEATURES OF ACUTE PERICARDITIS, INCLUDING:

01 Friction rub

02 Pleuritic chest pain , dry cough

03 Diffuse ST elevations on ECG

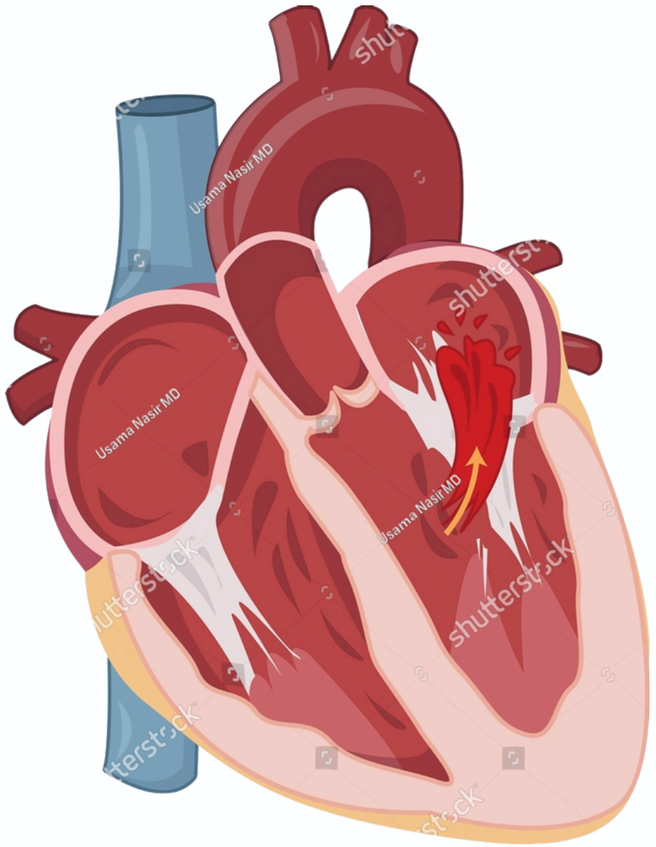
04 Pericardial effusion

- Treatment: supportive care
- Complications (rare): hemopericardium, pericardial tamponade
- Prognosis: usually self-limiting
- Prevention: early coronary reperfusion therapy

3-14 DAYS POST-INFARCTION

PAPILLARY MUSCLE RUPTURE

Usually occurs 2-7 days after myocardial infarction
Can lead to acute mitral regurgitation



Location

More often: posteromedial papillary muscle rupture due to occlusion of the posterior descending artery (single supply)
Less often:anterolateral papillary muscle rupture due to occlusion of LAD and/or LCx (double supply

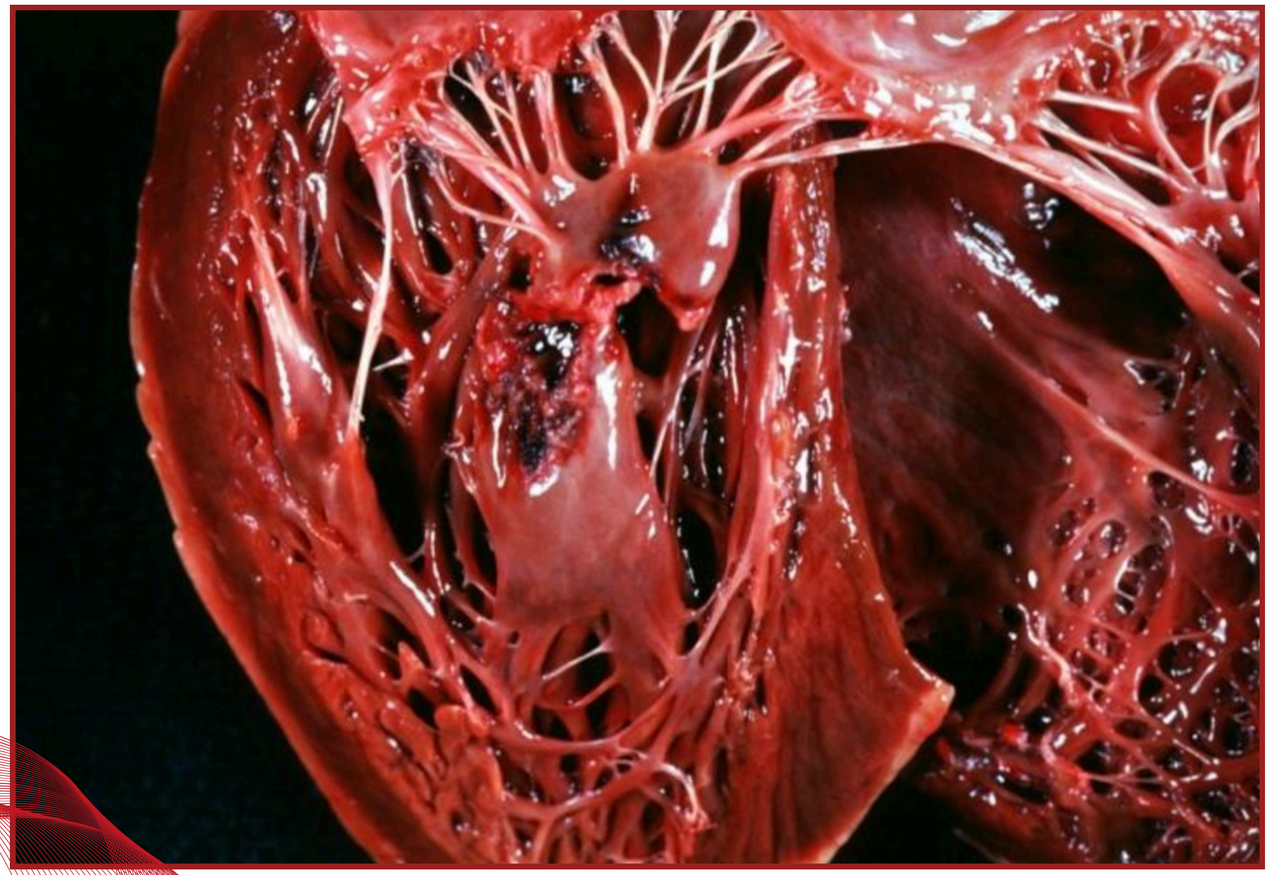
Clinical features

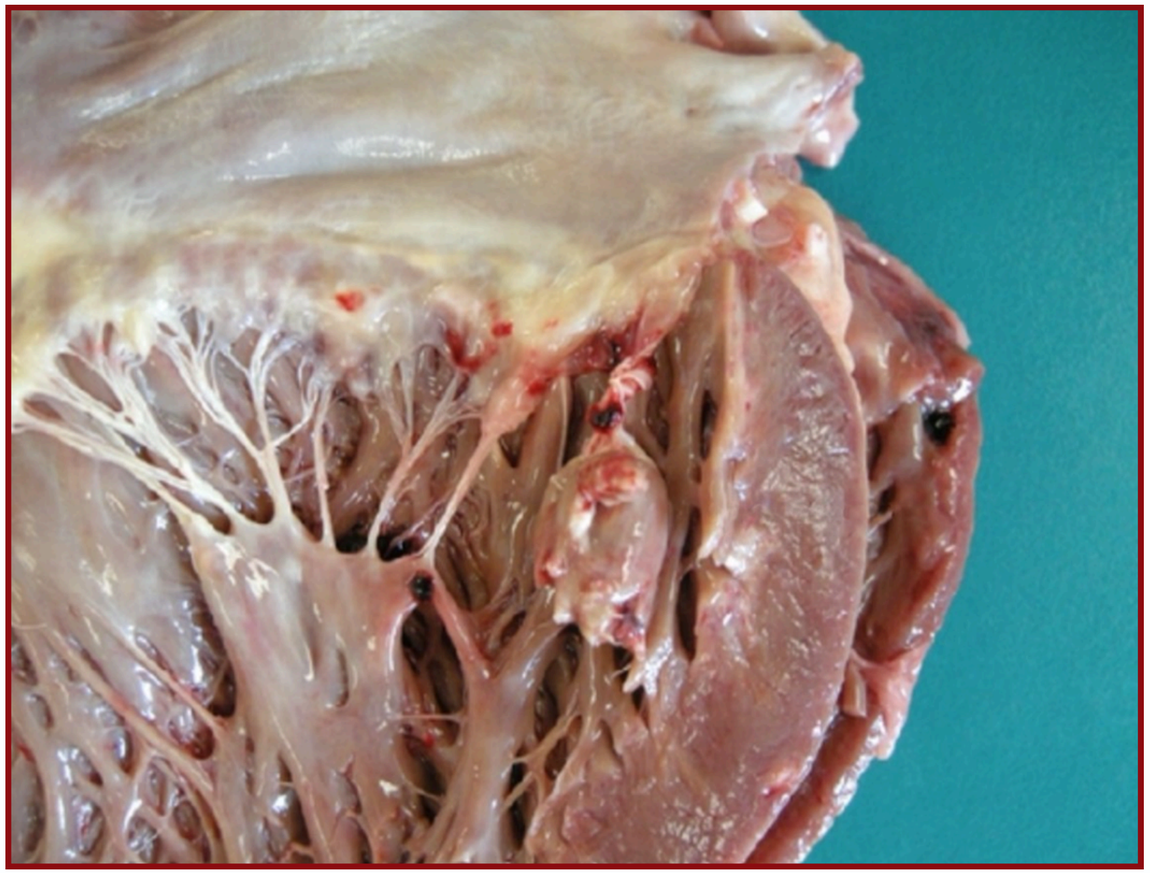
New holosystolic, blowing murmur over the 5 thICS on the midclavicular line
Signs of acute mitral regurgitation: dyspnea, cough, bilateral crackles, hypotension

Complications

Mitral regurgitation can lead to severe pulmonary edema and/or cardiogenic shock.

PAPILLARY MUSCLE RUPTURE





3-14 DAYS POST-INFARCTION

VENTRICULAR SEPTAL RUPTURE

About

- Usually occurs 3-5 days after myocardial infarction
- Most commonly due to LAD infarction (septal arteries arise from LDA)

Pathophysiology

macrophagic degradation of the septum → ventricular septal defect → blood flow from LV to RV following the pressure gradient
(left-to-right shunt) → ↑ pressure in RV and ↑ O 2 content in the venous blood

Complications

can progress to cardiogenic shock and severe pulmonary edema

Clinical features

- New holosystolic murmur over the left sternal border
- Acute-onset right heart failure (jugular venous distention, peripheral edema)

LEFT VENTRICULAR FREE WALL RUPTURE

USUALLY OCCURS 5–14 DAYS AFTER MYOCARDIAL INFARCTION

Greatest risk during macrophage-mediated removal of necrotic tissue

LV hypertrophy and tissue fibrosis from previous MI decrease the risk of free wall rupture.

Clinical features

- Chest pain
- Dyspnea
- Signs of cardiac tamponade (e.g., Beck triad)

Complications: cardiac tamponade , sudden cardiac death (if the rupture occurs acutely)

3–14 DAYS POST-INFARCTION

LEFT VENTRICULAR PSEUDOANEURYSM

refers to the outpouching of the ventricular wall rupture that is contained by either the pericardium, a thrombus, or scar tissue
Usually occurs 3–14 days after myocardial infarction

Clinical features

Can be asymptomatic

If symptomatic:

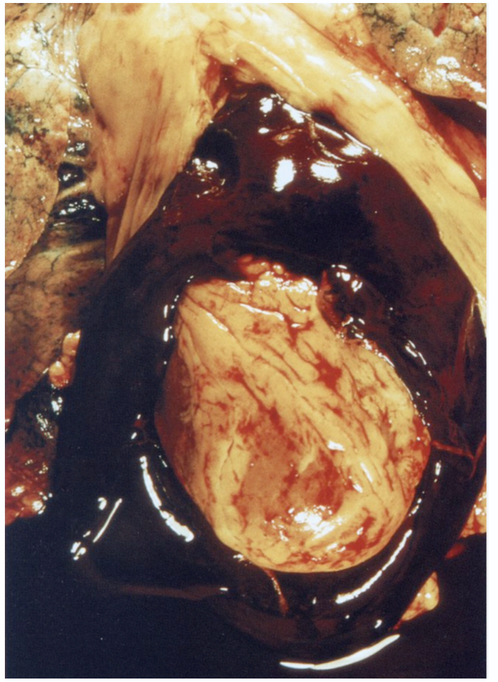
- New heart murmur
- Chest pain
- Heart failure
- Syncope

Complications

Rupture → cardiac tamponade (risk of rupture is higher than in a true aneurysm)

Associated with:

- mural thromboembolism
- decreased cardiac output
- increased risk of arrhythmia



2 weeks to months post-infarction

01 Atrial and ventricular aneurysms

02 Dressler syndrome

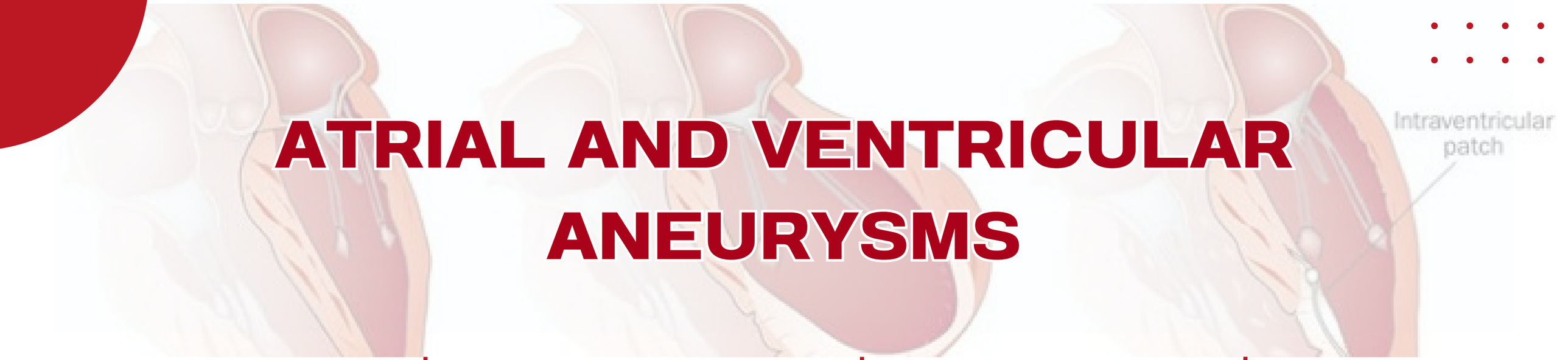
03 Mural thrombuformation

04 Congestive heart failure

05 Arrhythmias

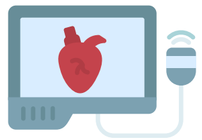
06 Reinfarction

ATRIAL AND VENTRICULAR ANEURYSMS



Clinical features

- LV is the MC
- Double Apex on exam
- Persistent (> 3 weeks post-MI) ST elevation and T-wave inversions
- Systolic murmur, S3 (V dysfunction and HF)and/or S4(Stiffness of V wall)
- New heart murmur(indicate thrombus or rupture), chest pain, heart failure, and/or syncope



Diagnosis

ECHOCARDIOGRAPHY

- Visualization of the pathological myocardial wall protrusion
- Detection of dyskinetic movements of the thinned aneurysmal wall (uncoordinated contraction occurs due to fibrotic changes of the myocardium)

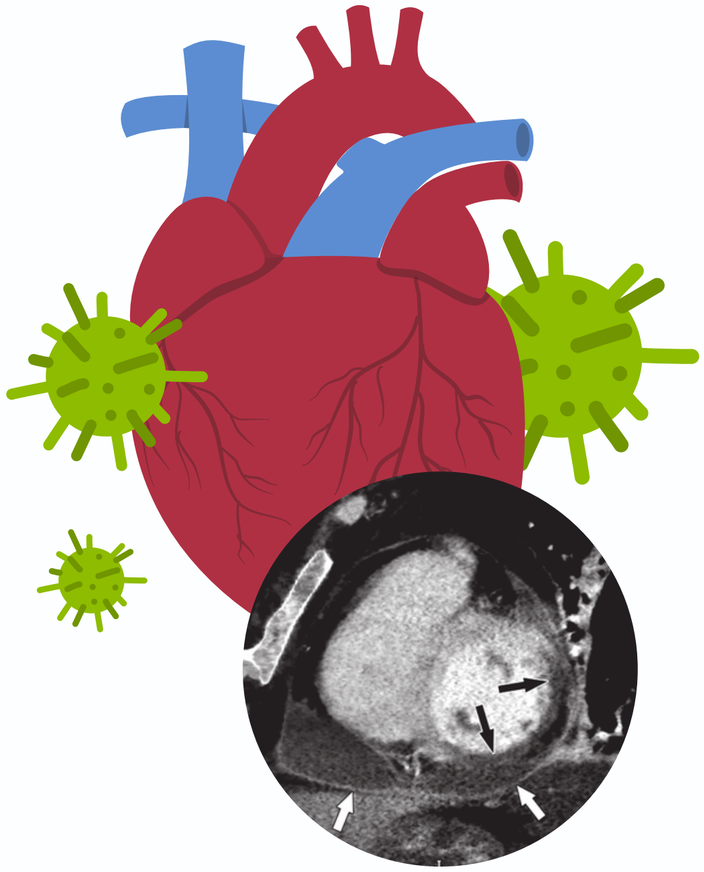
Complications

- Cardiac arrhythmias (risk of ventricular fibrillation)
- Rupture → cardiac tamponade
- Left ventricular thrombus

Treatment

- Anticoagulation if is present
- Possibly surgery

DRESSLER'S SYNDROME



"Postmyocardial infarction syndrome "

pericarditis occurring 2-10 weeks post-MI without an infective cause

Pathophysiology

thought to be due to circulating antibodies against cardiac muscle cells (autoimmune etiology) →immune complex deposition →inflammation

Clinical features

Signs of acute Pericarditis:Pleuritic chest pain , dry cough , friction rub,Fever

Investigations

ECG: diffuse ST elevations
LAB:leukocytosis, ↑ serum troponin

Treatment

NSAIDs (e.g., aspirin), colchicine
Aspirin 1st\ Ibuprofen 2ed choeice of ttt.

Complications (rare)

hemopericardium, pericardial tamponade

CONTINUE

Mural thrombus formation

thromboembolism (stroke, mesenteric ischemia, renal infarction, acute obstruction of peripheral arteries)

Congestive heart failure

- (e.g., due to ischemic cardiomyopathy)
- Can occur at any time after an ischemic event
- Treatment: for patients with LVEF < 40% or signs of heart failure, ACE inhibitor/Angiotension receptor BLOCKERS “ARB” and aldosterone antagonists have been shown to confer a mortality benefit.

Reinfarction

- Causes :

1. Incomplete revascularization
2. Recurrent atherosclerosis
3. Stent thrombosis
4. Non-compliance with medications
5. Uncontrolled risk factors
6. Embolism

CK-MB is the best here cuz it returns to its normal level faster

Arrhythmias

SUMMARY

24h

- Sudden cardiac death (SCD)
- Arrhythmias
- Acute leb heart failure
- Cardiogenic shock

1–3d

Early infarct-associated pericarditis

3–14d

- Papillary muscle rupture
- Ventricular septal rupture
- Left ventricular free wall rupture
- Left ventricular pseudoaneurysm

2w–m

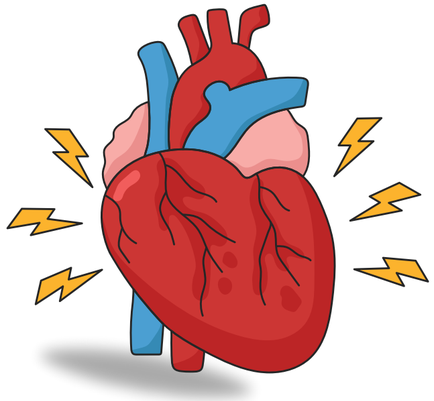
Atrial and ventricular aneurysms
Dressler syndrome
Mural thrombus formation
Arrhythmias
Congestive heart failure
Reinfarction

USEFUL LINKS

Quiz Time

Mind Map

Ninja Nerd



" وَيَومَئذ يَفرَح ٱلمُؤمِنُونَ
بِنَصرِ اللّهِ "