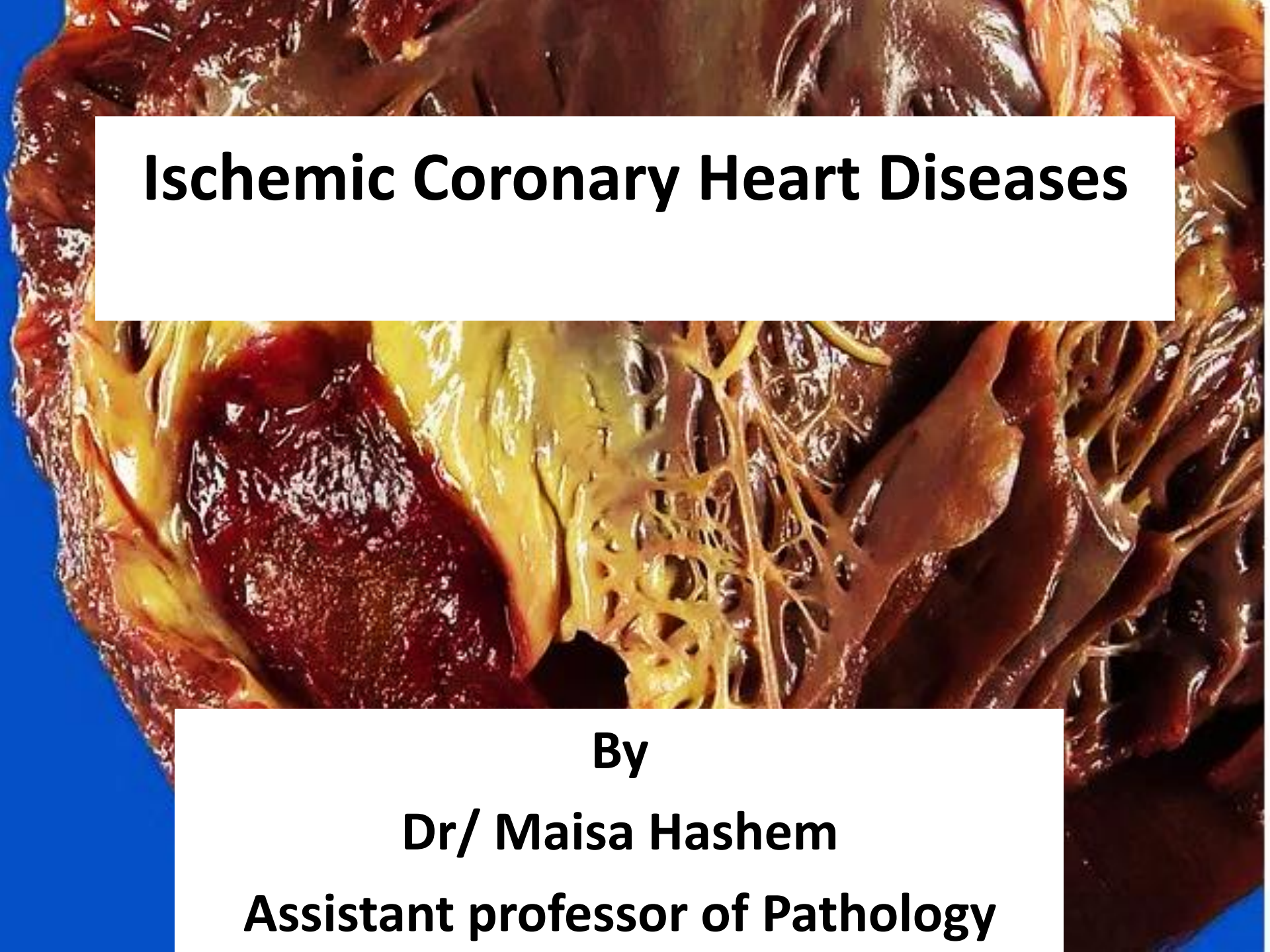

Ischemic Coronary Heart Diseases

By

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Learning objectives

After the lecture, students should be able to:

- List the risk factors for ischemic coronary heart disease.
- Enumerate cause and effects of coronary occlusion.
- Discuss sites, macroscopic changes and histological changes of myocardial infarction.
- List the possible complications of myocardial infarction.

Ischemic Heart Disease (IHD)

- It is a condition in which there is imbalance between myocardial oxygen supply and demand.
- Most often caused by atherosclerosis of the coronary arteries.

Risk Factors for Ischemic Coronary Heart Diseases

I: Non-modifiable risk factors:

- Age: between ages of 40 to 60 years, the incidence of IHD increases five folds. Also. Death rates from IHD rises with advanced ages.
- Genetic factors: familial hypercholesterolemia predispose to atherosclerosis and IHD.
- Sex: premenopausal women are relatively protected against atherosclerosis and its related consequences as IHD, compared to age-matched men (in absence of other risk factors as D.M., HTN and hyperlipidaemia.
- After menopause; the incidence of IHD exceeds in women than in men.

II- Modifiable risk factors:

- Hyperlipidaemia and increased serum level of LDL are associated with increased risk for atherosclerosis and IHD
- Hypertension increases risk for IHD by about 60%.
- Cigarette smoking.
- Diabetes mellitus: as it induces hypercholesterolemia and increases risk for IHD.

Causes of Coronary Occlusion

I- Gradual incomplete coronary occlusion:

Causes: gradual reduction of coronary blood flow due to *coronary atherosclerosis.*

Effects:

- Abnormal cardiac rhythm.
- Angina pectoris: Angina pectoris is sudden attacks of chest pain, discomfort, or pressure occurring when the heart muscle does not receive enough oxygen-rich blood, typically caused by coronary artery disease. It feels like squeezing, heaviness, or burning, often radiating to the arms, neck, or back, and is a key symptom of reduced blood flow.
- Left sided heart failure.
- Superimposed sudden coronary occlusion

II- Sudden complete coronary artery occlusion

Causes of coronary occlusion:

- Coronary atherosclerosis: it is the main cause of sudden coronary occlusion (> 90%). Coronary atherosclerosis may result in thrombosis or sub intimal hemorrhage with subsequent elevation of the sub endothelial connective tissue (rupture of atheromatous plaque.
- Coronary embolism: rare, as coronary filling occurs in the diastole. The embolus may arise from vegetations of subacute bacterial endocarditis, mural thrombosis over left ventricular infarction.

Results of sudden coronary occlusion:

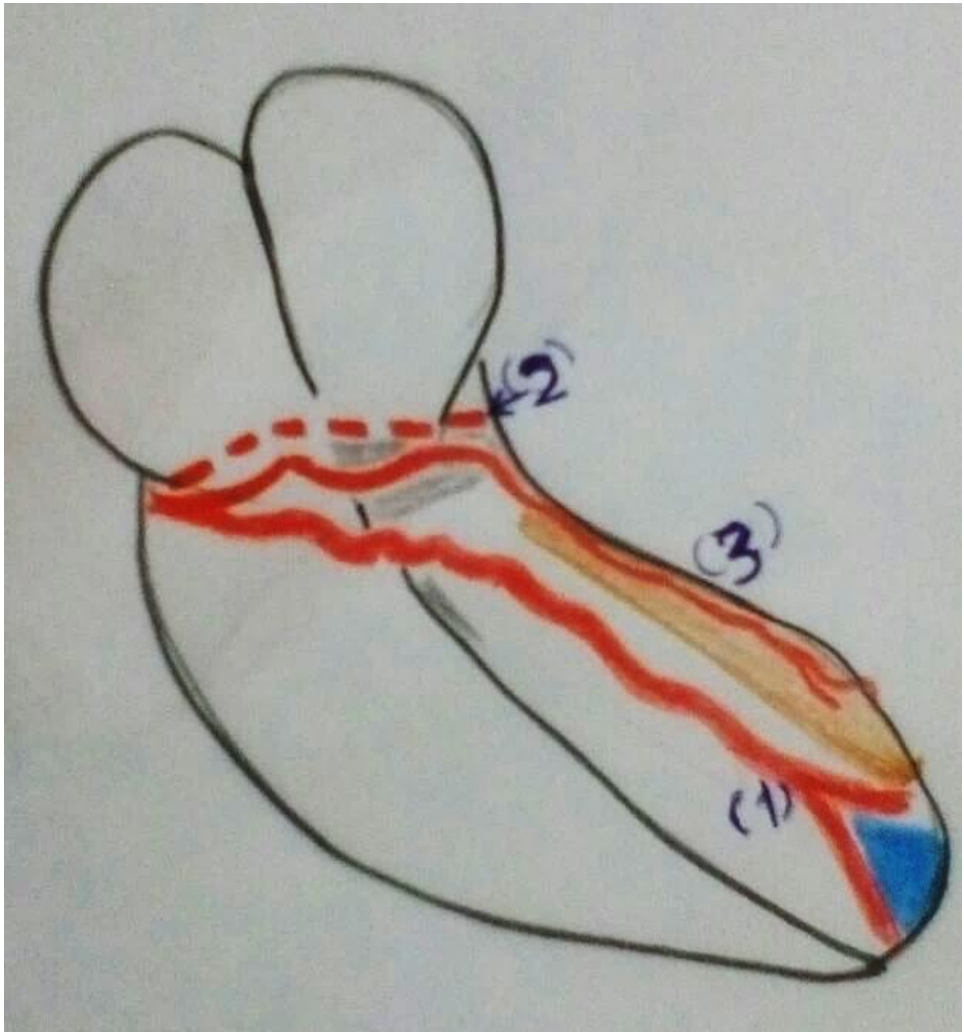
- Sudden death due to ventricular fibrillation.
- Myocardial infarction.

Myocardial infarction

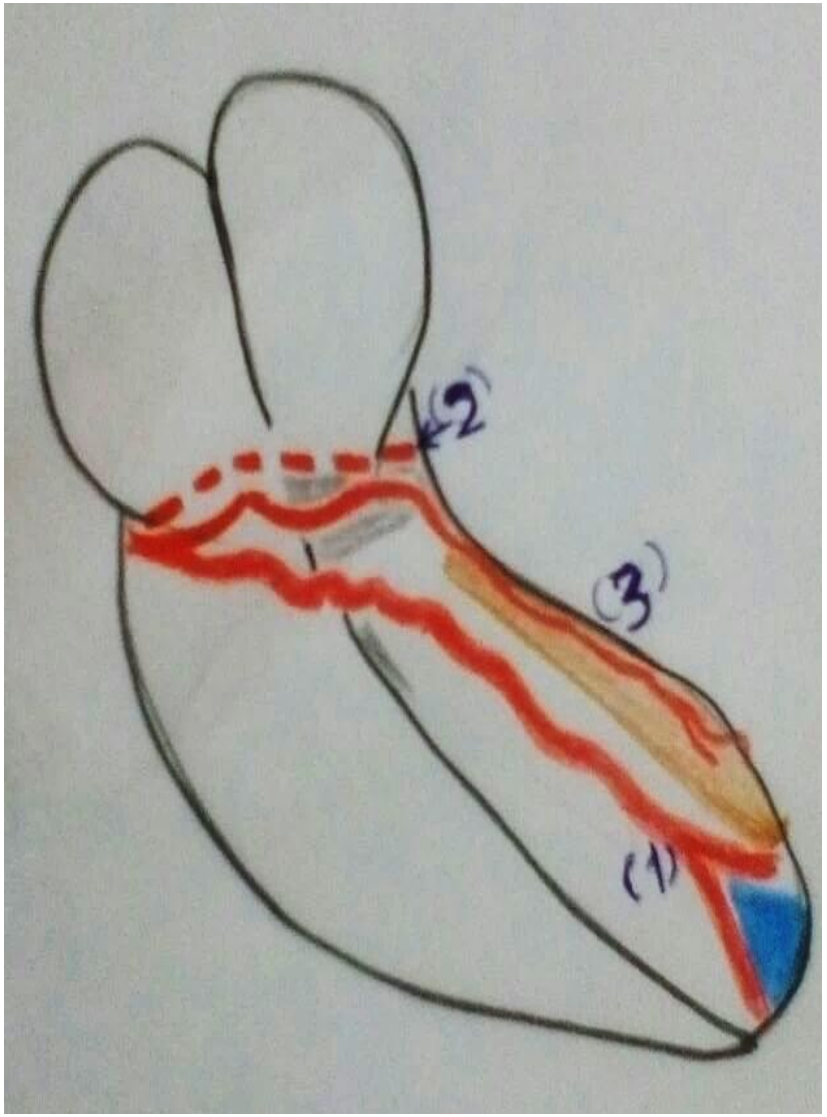
Sites of infarction

1) Occlusion of the anterior descending branch of the left coronary (This is the commonest site):

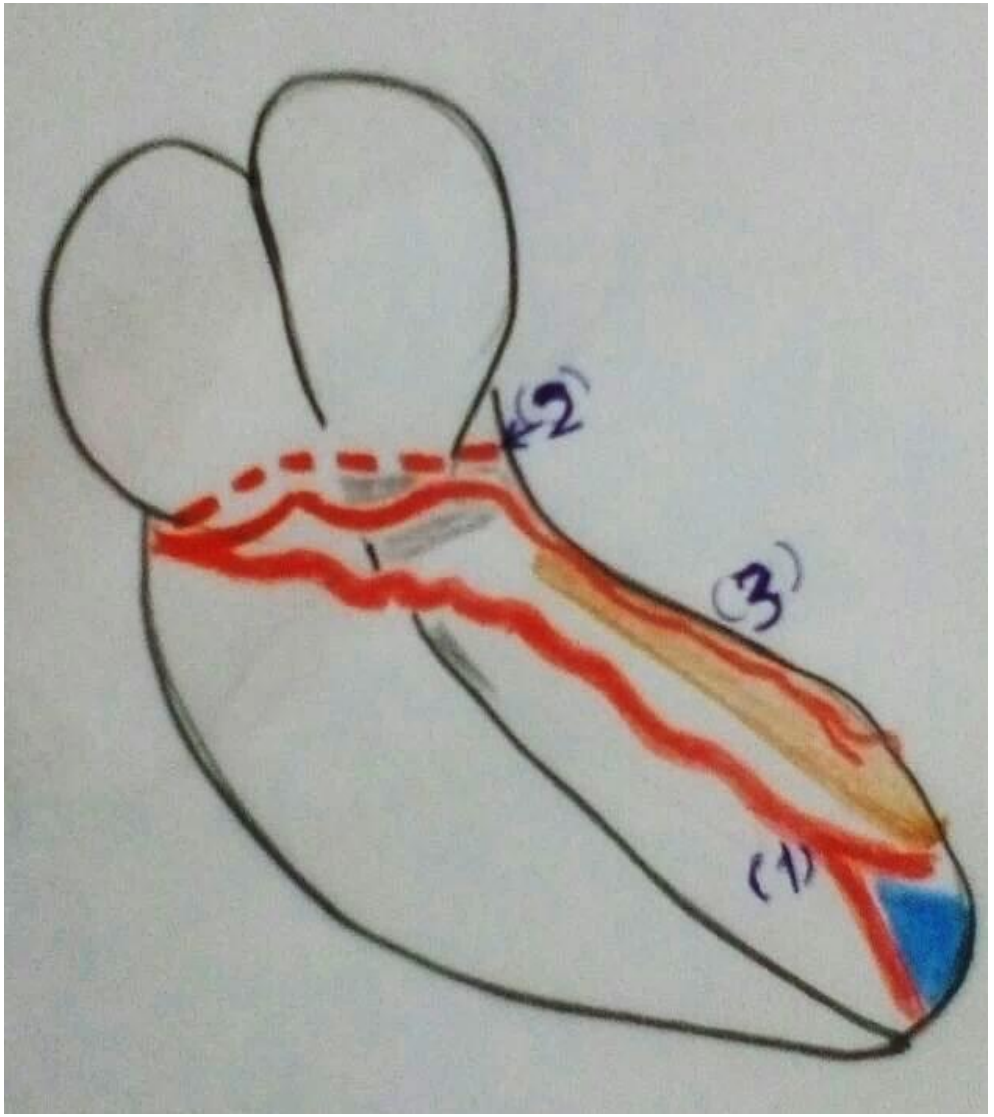
Causes infarction in the anterior wall of the left ventricle towards the apex and anterior portion of the interventricular septum.



2) Occlusion of the right coronary causes infarction in the posterior wall of the left ventricle near the base and the posterior part of the septum. Affection of right ventricle is minimal or absent.



3) Occlusion of the left circumflex artery cause infarction in the lateral wall of the left ventricle.

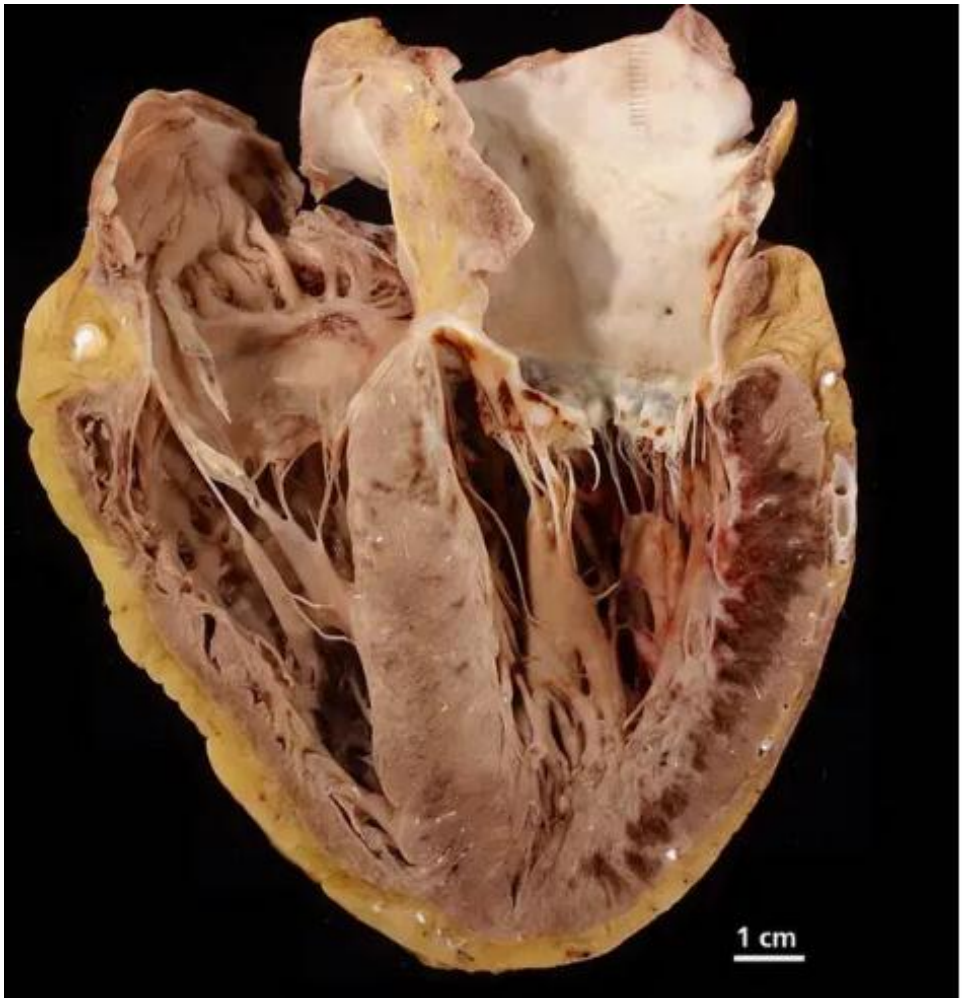


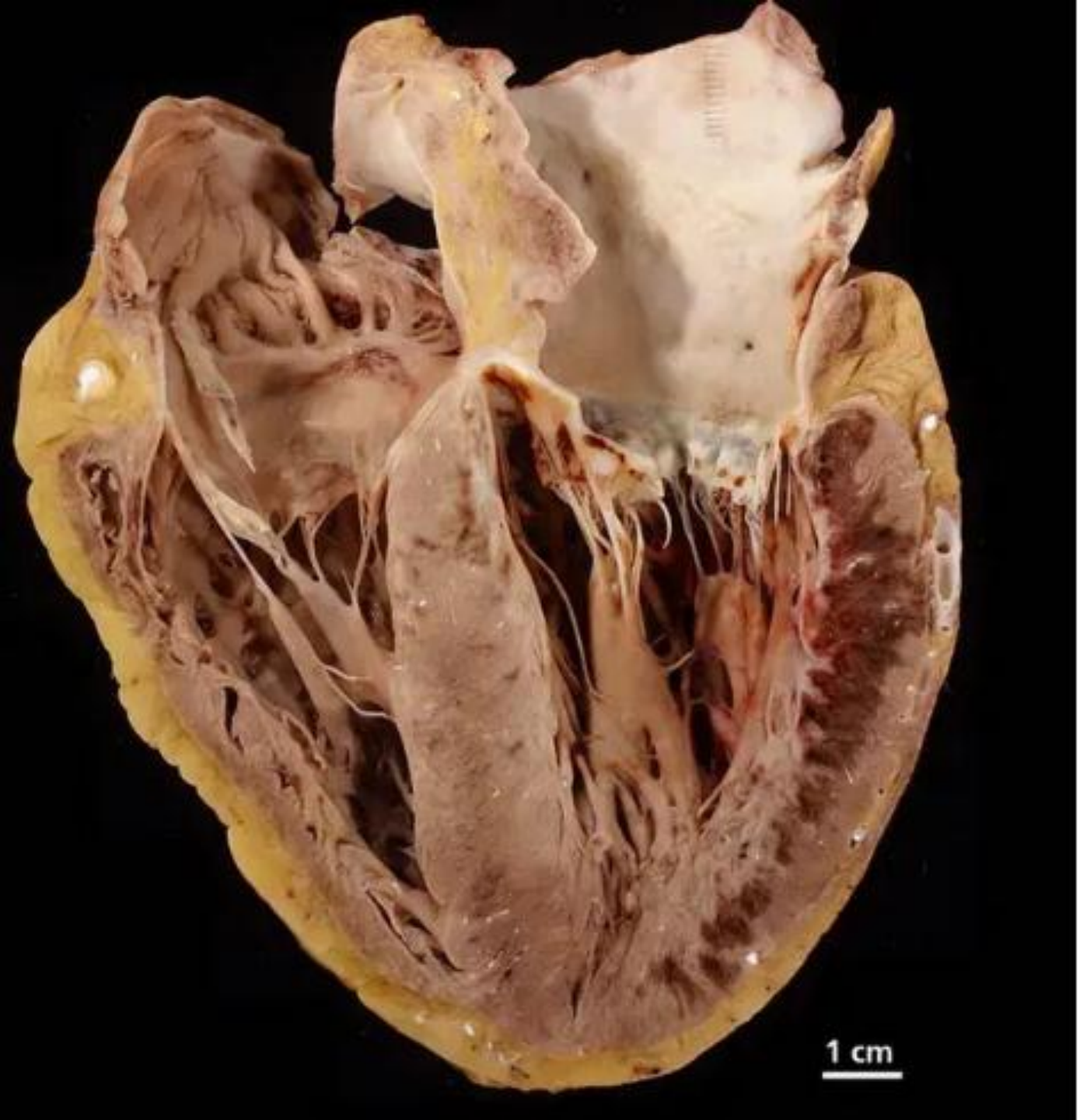
Gross picture

Recent infarction:

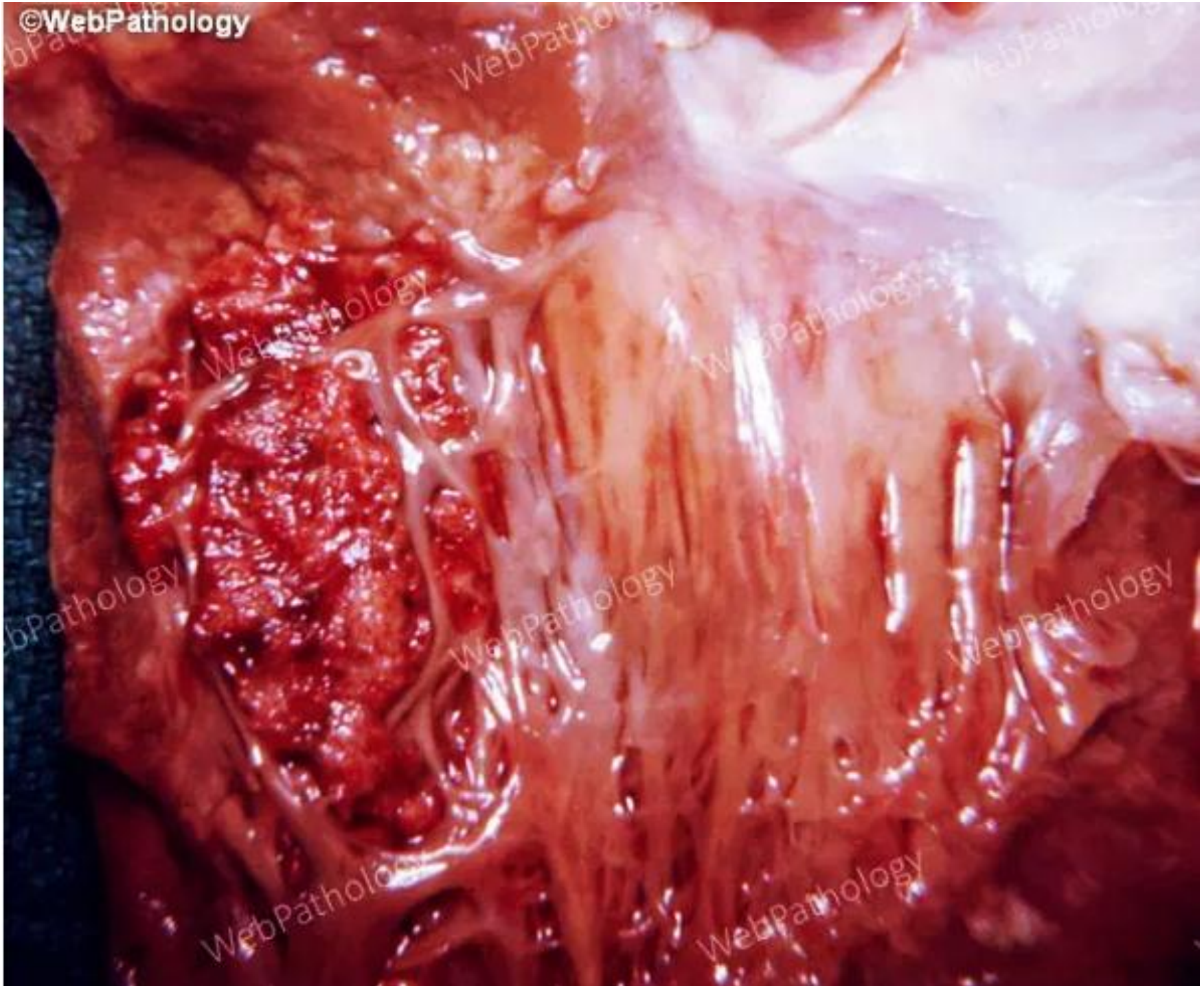
Appears swollen, dark red and friable. Later the color changes to yellowish brown with remains of hemorrhage at the edges.

The infarct is covered on the endocardial surface by a mural thrombus which may fragment forming emboli. The infarct area is covered on the pericardial surface by a patch of fibrinous pericarditis.





Acute infero-lateral infarction with hemorrhage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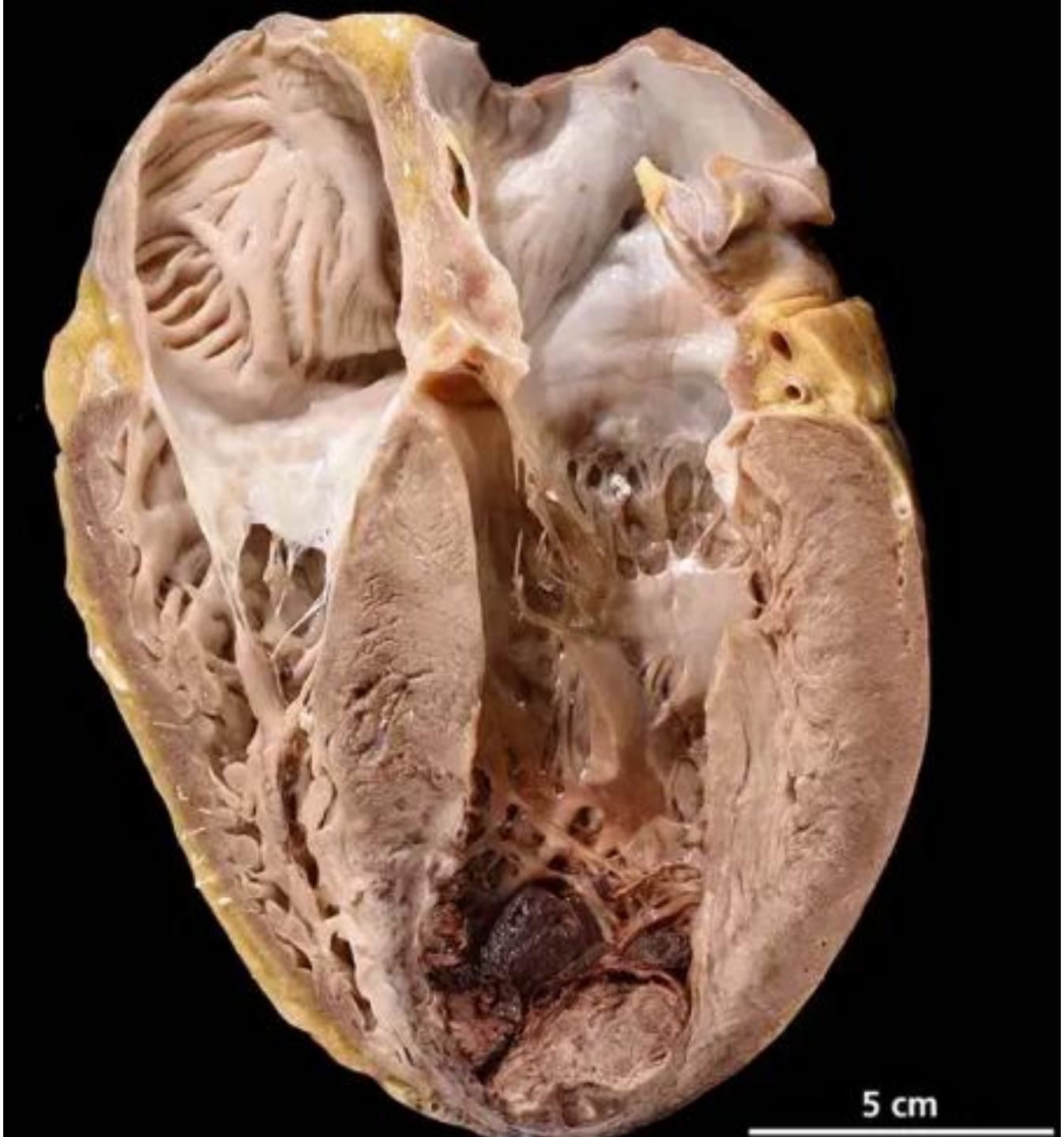
Healed infarct:

Appear as a thin grayish white fibrous patch which is weak and nonelastic. The rest of the myocardium shows compensatory hypertrophy. The pericardium covering the infarct shows fibrosis and adhesions.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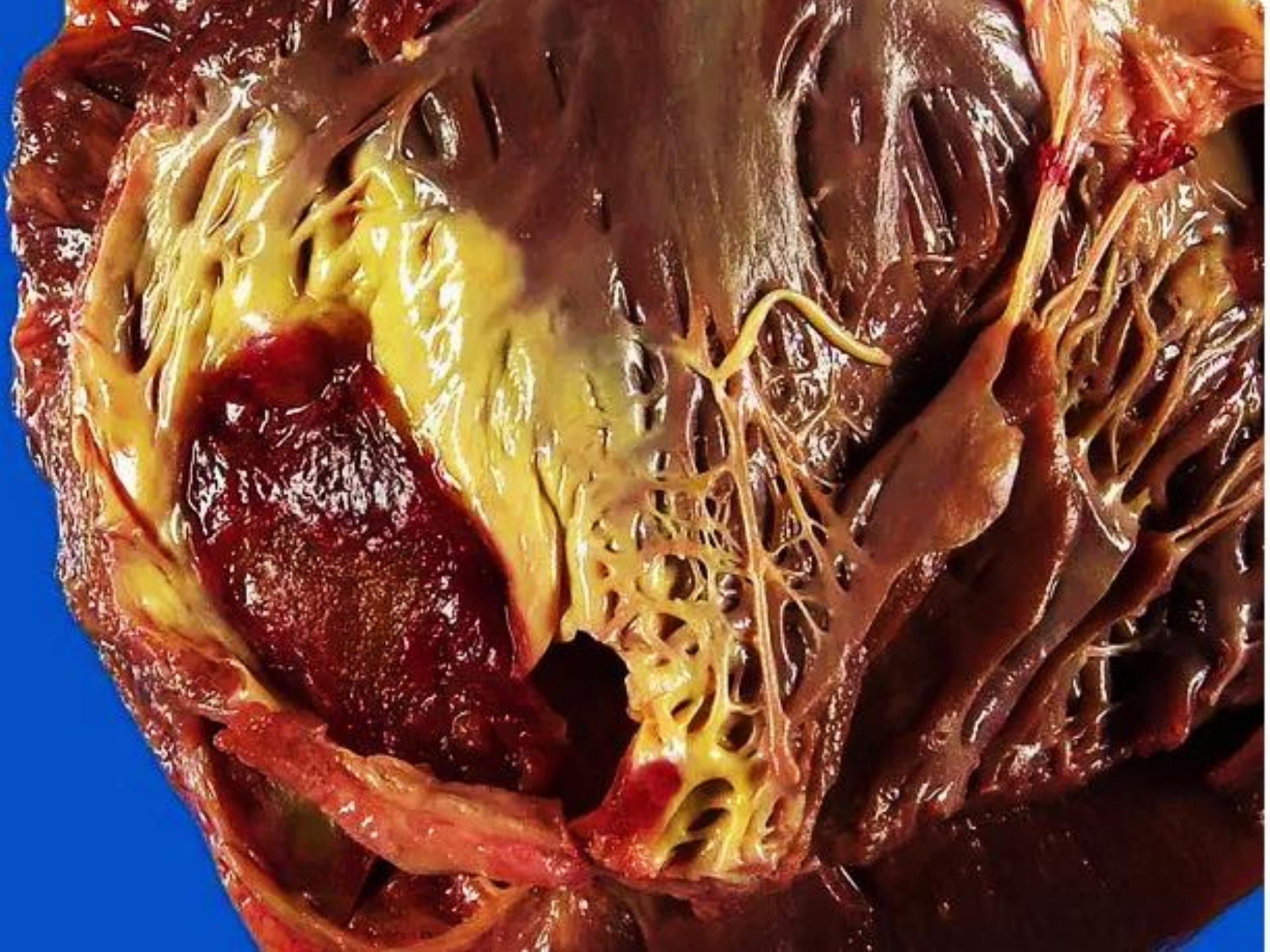
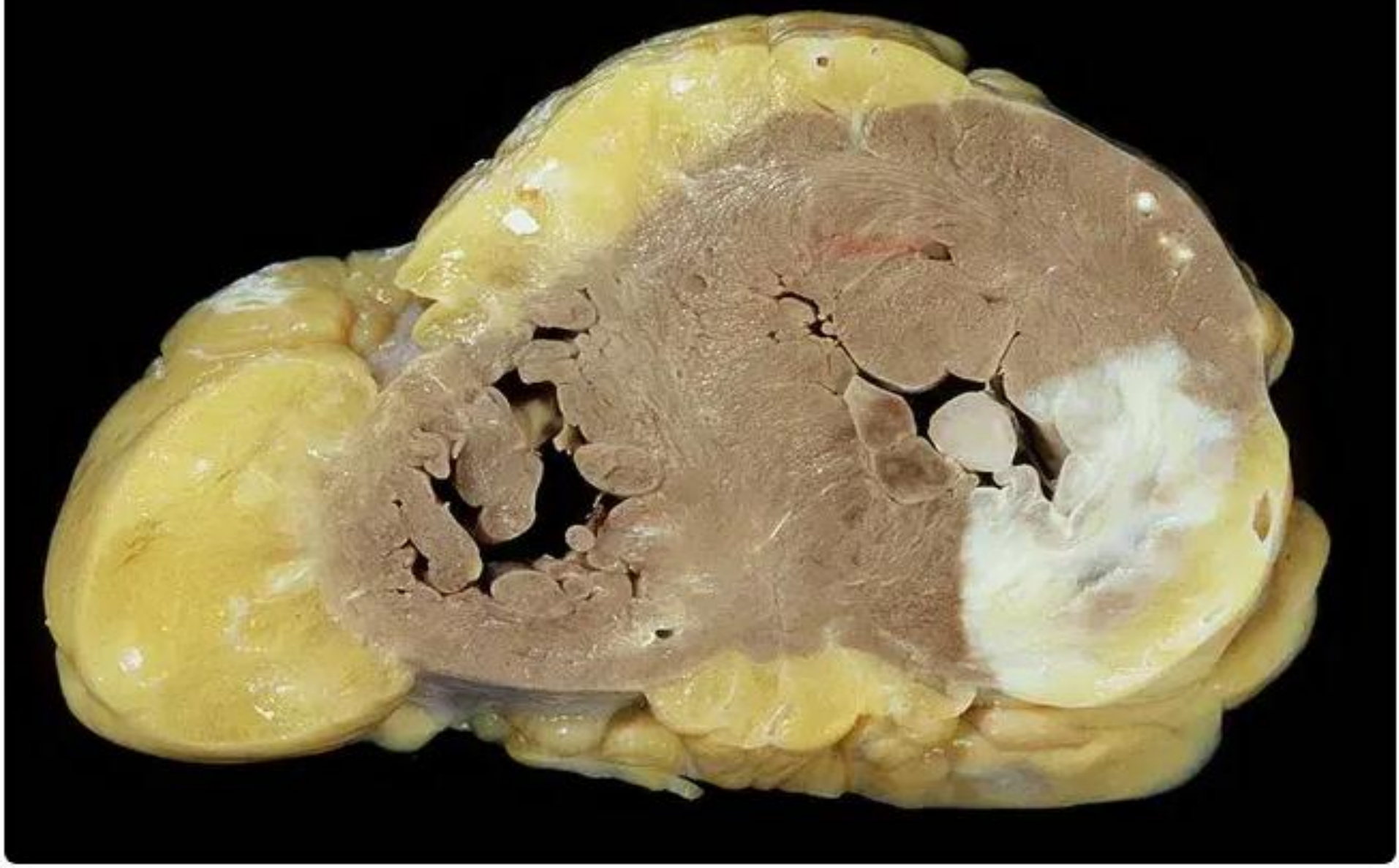
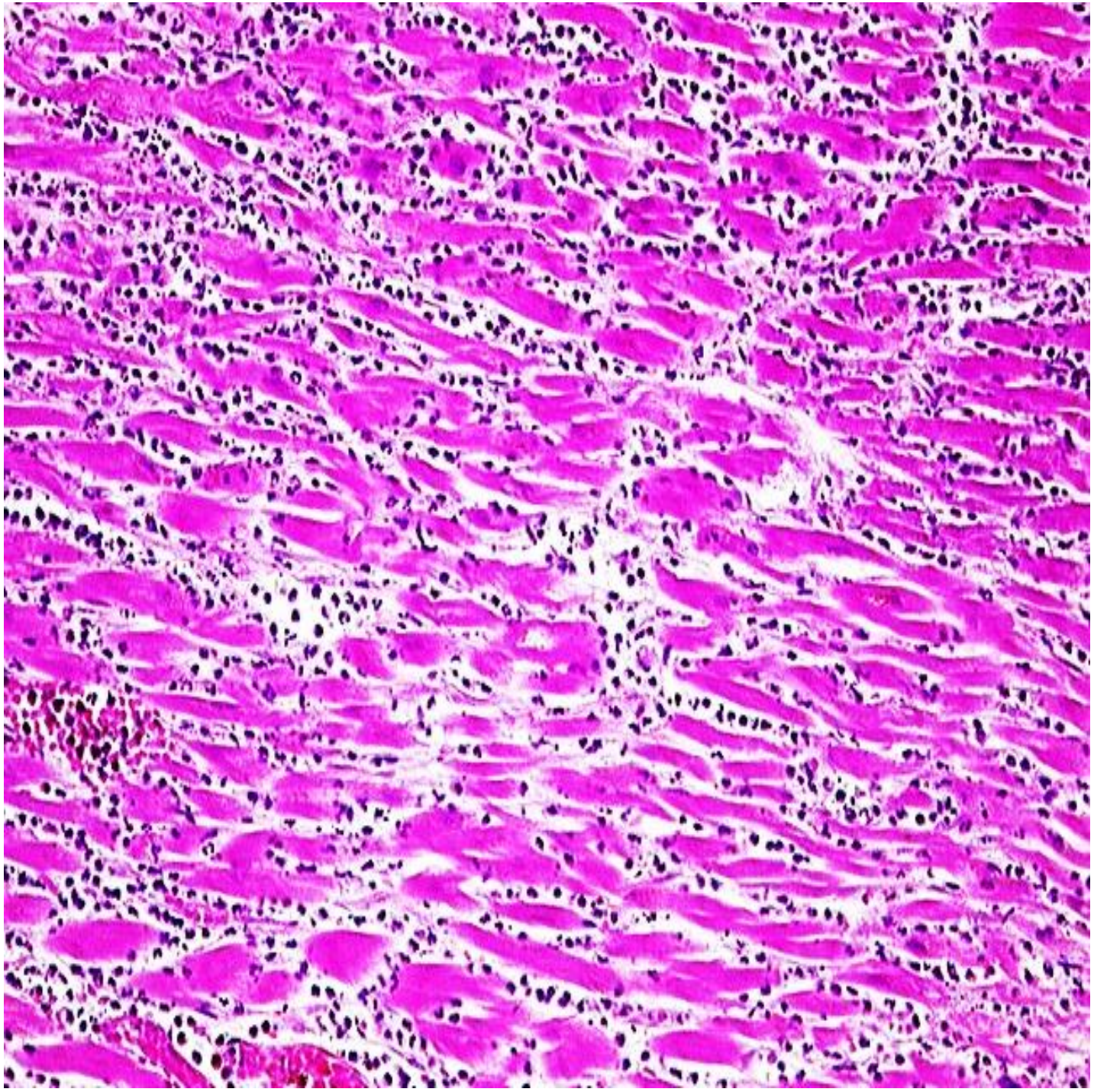




Healed myocardial infarction of the posterior and lateral walls in a 70 y/o male. There is biventricular concentric myocardial hypertrophy.

Microscopic picture:

Microscopic features of coagulative necrosis appear by the end of 6 hours. The cross striations of the muscles and the nuclei disappear. Leucocytic infiltration followed by fibrosis.



Acute inflammatory response is seen within the damaged myocardium in 3 to 4 days.

Complications of Myocardial infarction

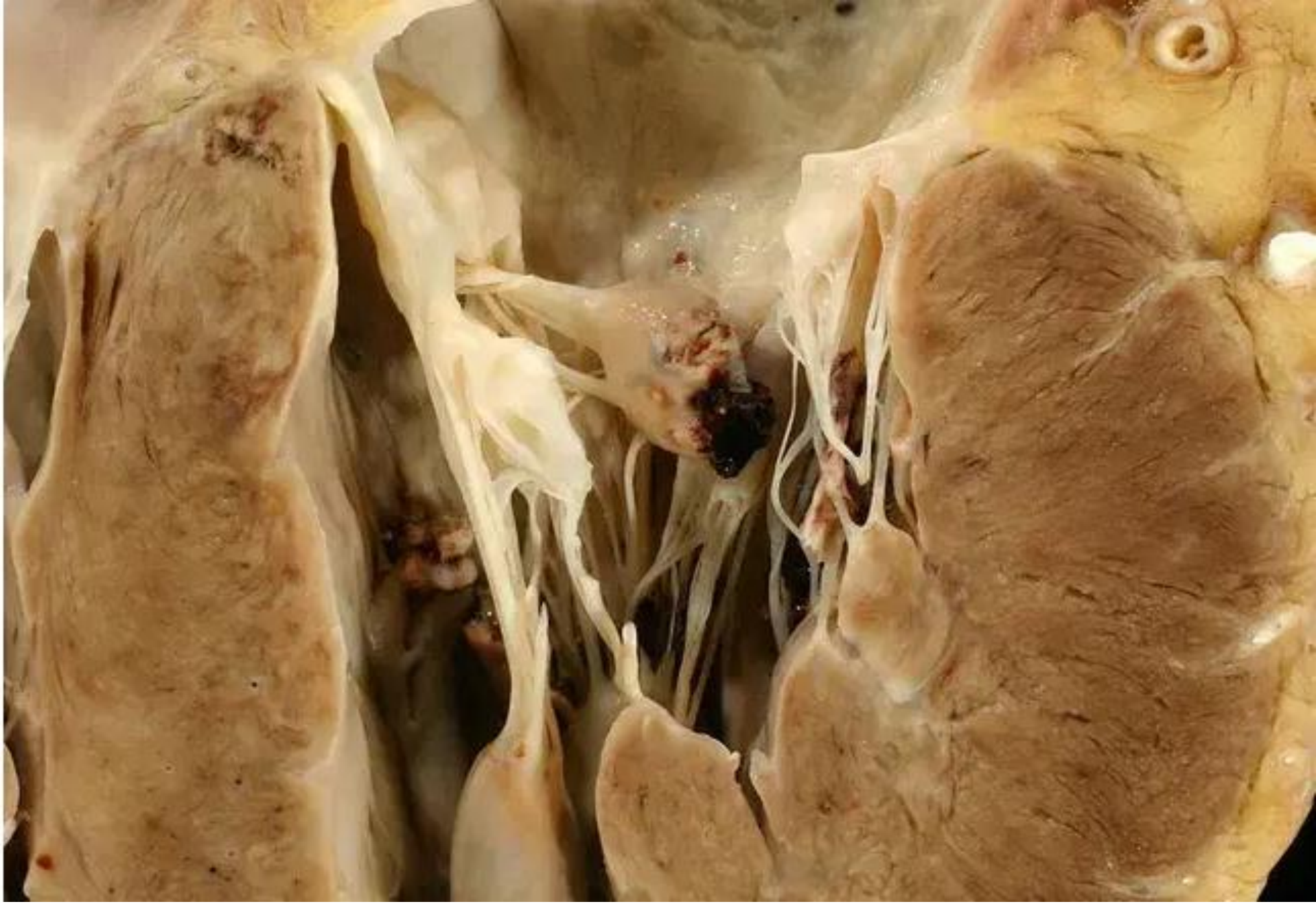
1) **Acute complications (Hours- Days):**

➢ Arrhythmia;

- Ventricular tachycardia.
- Ventricular fibrillation causing sudden death.

➢ Mechanical disruption:

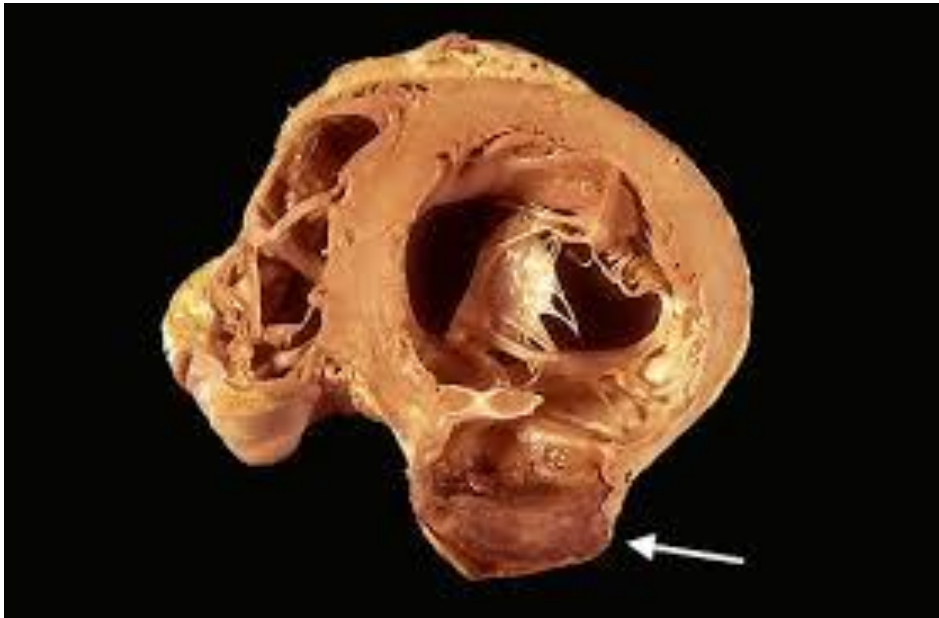
- Ventricular wall rupture causing cardiac tamponade.
- Ventricular septal defect.
- Papillary muscle rupture cause acute mitral regurgitation.



Rupture of the papillary muscle is the **least common form of myocardial rupture following an acute myocardial infarction (MI)**. It results in **severe acute mitral regurgitation**.

2) Chronic/ subacute complications (weeks to months):

- Left ventricular aneurysm.
- Chronic heart failure.
- Dressler syndrome: immune-mediated pericarditis occur weeks to months post myocardial infarction.
- Post infarction angina: recurrent chest pain due to ongoing ischaemia.
- Ventricular pseudoaneurysm: ventricular rupture contained by surrounding adhesions.




Thank You