Ischemic Heart Disease

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General Consideration

- Results from partial or complete interruption of arterial blood flow to the myocardium.
- Most occurs because of atherosclerotic plaque with in one or more coronary arteries
- Ischaemia maybe clinically silent, manifest as angina pectoris or MI.

Oxygen Carrying Capacity

- The oxygen carrying capacity relates to the content of hemoglobin and systemic oxygenation
- When atherosclerotic disease is present, the artery lumen is narrowed and vasoconstriction is impaired
- Coronary blood flow cannot increase in the face of increased demands and ischemia may result

Angina Pectoris

- Angina Pectoris: uncomfortable sensation in the chest or neighboring anatomic structures produced by myocardial ischemia

Types of Angina

- **Stable angina**: most common. Pain is precipitated by exertion. Relieved by rest or vasodilators.
- **Unstable angina**: prolonged or recurrent pain at rest. Often indicative of imminent MI.
  - Generally caused by disruption of an atherosclerotic plaque with superimposed thrombosis.
- **Prinzmetal (variant) angina**: intermittent chest pain at rest. Cause by vasospasm.

Angina: cont

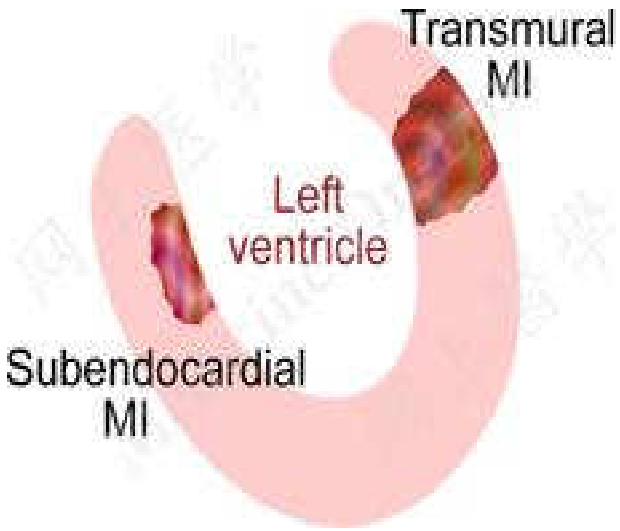
- Patients with mild obstruction coronary lesions can also experience unstable angina
- >90% of Acute MI result from an acute thrombus obstructing a coronary artery with resultant prolonged ischemia and tissue necrosis

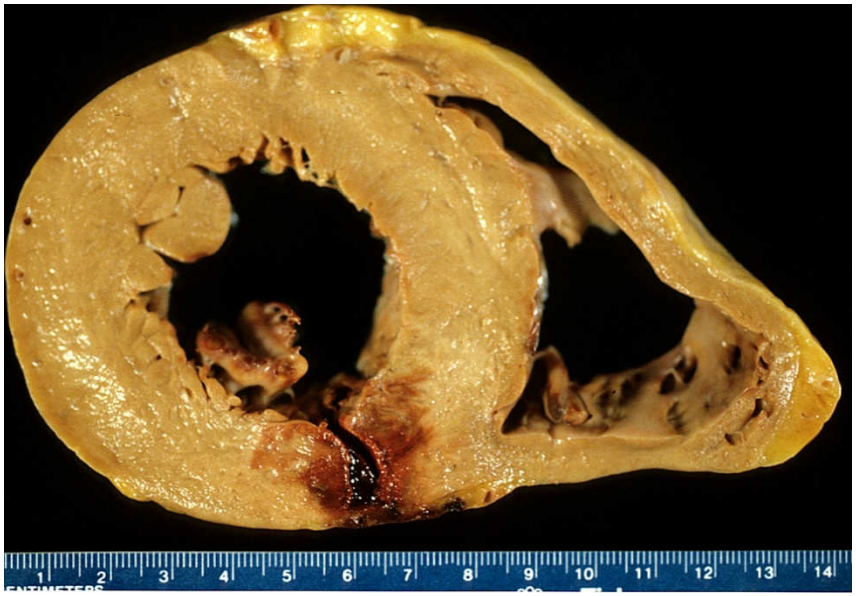
Treatment of Angina

- Treatment of Chronic Angina is directed at minimizing myocardial oxygen demand and increasing coronary flow
- Where as in the acute syndromes of unstable angina or MI primary therapy is also directed against platelet aggregation and thrombosis

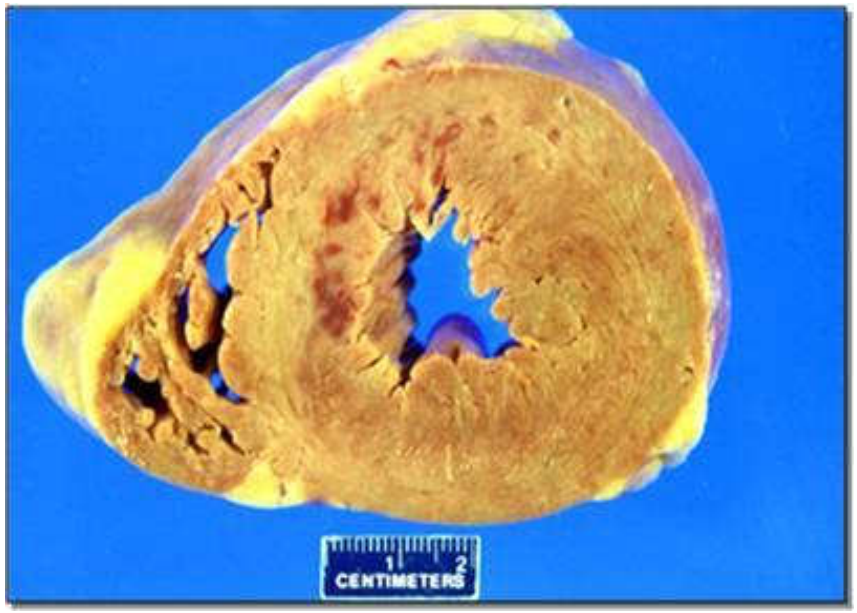
Myocardial Infarction

- MI is ischaemic necrosis of myocardium.
- MI – can be transmural or subendocardial.
- Necrosis evokes a PMN response 1-3 days after infarction.
  - Macrophages replace PMN after 3-7 days.
  - Granulation tissue forms 2-3 weeks after infarction.

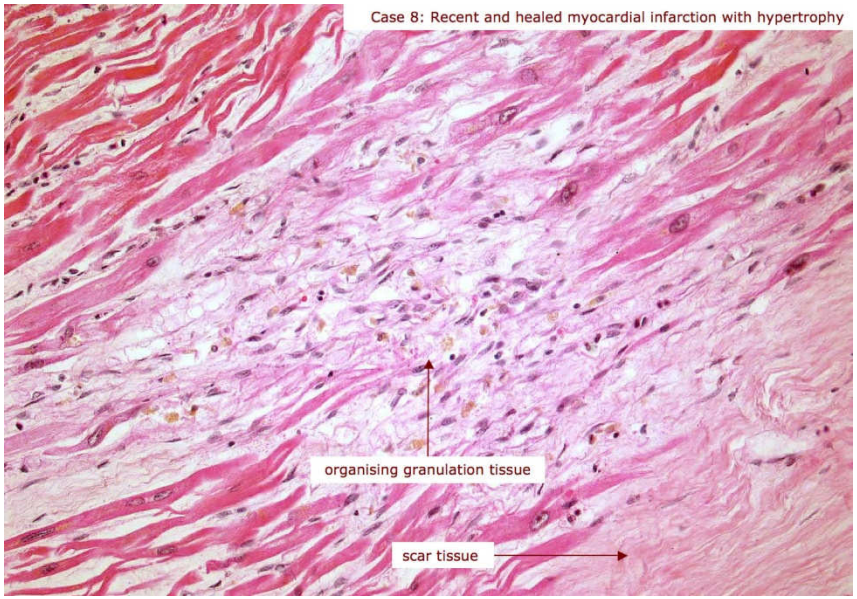




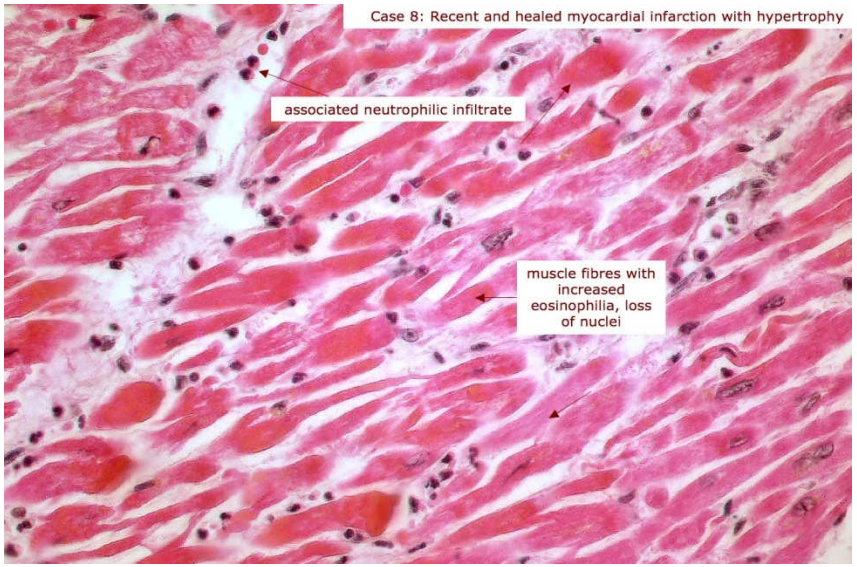
Transmural



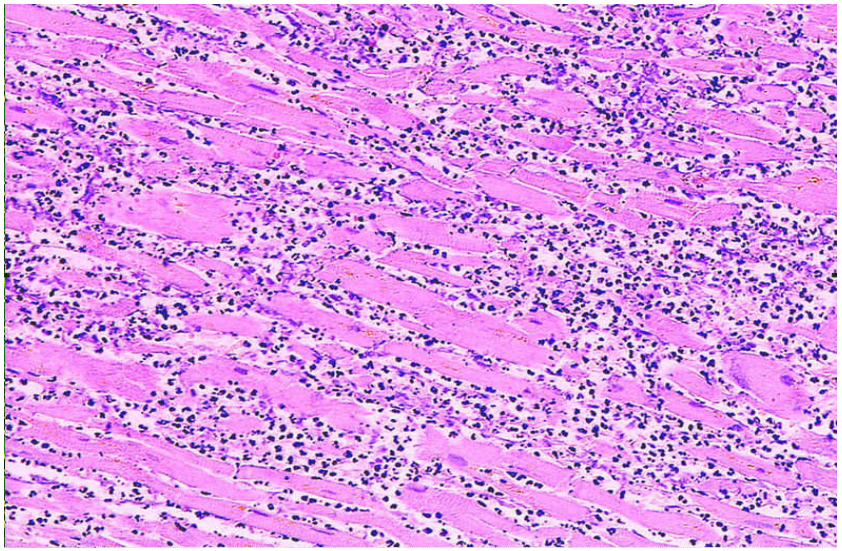
Subendocardial



3 weeks old infact



2 day old infarct



7 day old infarct

Diagnostic Tests

- Blood tests include serum lipids, fasting blood sugar, Hematocrit, thyroid (anemias and hyperthyroidism can exacerbate myocardial ischemia
- Resting Electrocardiogram:

Electrocardiogram

- Electrocardiogram: is useful in diagnosis when pt has chest pain
- When ischemia results in transient horizontal or downsloping ST segments or T wave inversions which normalize after pain resolution
- ST elevation suggest severe transmural ischemia or coronary artery spasm which is less often

Exercise Stress Test

- Used to confirm diagnosis of angina

Other Diagnostic Tests

- Radionuclide studies
- Myocardial perfusion scintigraphy
- Exercise radionuclide ventriculography
- Echocardiography
- Ambulatory ECG monitoring
- Coronary arteriography

Serum Markers of Infarction

- Certain proteins are released into circulation during an MI
- Creatine kinase CK rises in plasma within 4 to 8 hours, peaks at 24 hours, returns to normal by 48 hours to 72 hours
- Not specific for myocardial damage: skeletal muscle trauma and IM injection, and hypothyroidism

CK-MB

- CK-MB isoenzyme is more specific for diagnosis of AMI
- Not influenced by skeletal muscle injuries
- CK-MB rises and peaks slightly earlier than total CK and returns to normal within 36 – 72 hours
- May be elevated in: myocarditis after surgery, hypothyroidism, repetitive cardioversion

Enzymes

- Acute MI: CK-MB is greater than 2.5% of total serum CK
- Serum CK and CK-MB isoenzyme should be measured on admission, then 12 and 24 hours later in diagnostic evaluation of an acute MI

Troponin

- Troponin I and T are sensitive and highly specific markers of acute MI
- Levels begin to rise within 3 hours after onset of infarction and remain elevated for several days
- Higher Troponin I levels or early (+) of Troponin T assay correlate with greater short-term mortality

Myoglobin

- Myoglobin is released into circulation very early after myocardial injury and detected within 2 hours of infarction
- Rapid renal clearance and low specificity limit it s diagnostic role

Lactate Dehydrogenase (LDH)

- Rises within 24 to 48 hours of MI
- Peaks at 3 – 5 days and returns to baseline by 7-10 days
- Usefulness in patients who are admitted to hospital 2 – 3 days after onset of symptoms
- Level of LDH-1 greater than LDH-2 = myocardial necrosis

Complications of MI

- Hemopericardium – usually in the first week
- Arterial emboli – may arise from a mural thrombus
- Pericarditis – seen in association with transmural MI
- Ventricular aneurysm – late complication.
- Dressler syndrome – late complication. An autoimmune pericarditis.

END

PDF file notes available at: www.pathologyatsmhs.wordpress.com

Other notes & study guides on:

- Heart failure
- Cardiomyopathy
- Endocarditis
- Rheumatic valvular heart diseases will be made available at website: www.pathologyatsmhs.wordpress.com

References: Robins Pathological Basis of Diseases, 6th & 7th Ed.