Cardiac pathology IV

Ischemic heart disease

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Angina Pectoris

- Angina pectoris is intermittent chest pain caused by transient, reversible myocardial ischemia.
- There are three variants:

1. ***Typical* or *stable angina***
2. ***Prinzmetal, or variant angina***
3. ***Unstable angina* (also called *crescendo angina*)**

- ***Typical* or *stable angina*** is episodic chest pain associated with exertion or some other form of increased myocardial oxygen demand (e.g., tachycardia or hypertension due to fever, anxiety, fear).
- The pain is classically described as a crushing or squeezing substernal sensation, which can radiate down the left arm or to the left jaw *(referred pain).*
- Stable angina pectoris is usually associated with a fixed atherosclerotic narrowing (≥75%) of one or more coronary arteries.

- With this degree of critical stenosis, the myocardial oxygen supply may be sufficient under basal conditions but cannot be adequately augmented to meet any increased requirements.
- The pain is usually relieved by rest (reducing demand) or by administering agents such as nitroglycerin; such drugs cause peripheral vasodilation and thus reduce venous blood delivered to the heart (hence reducing cardiac work); in larger doses, nitroglycerin also increases blood supply to the myocardium by direct coronary vasodilation

- ***Prinzmetal, or variant angina*** is angina occurring at rest due to coronary artery spasm.
- Although such spasms typically occur on or near an existing atherosclerotic plaque, completely normal vessels can be affected.
- The etiology is not clear, but Prinzmetal angina typically responds promptly to the administration of vasodilators such as nitroglycerin or calcium channel blockers.

- ***Unstable angina*** is characterized by increasing frequency of pain, precipitated by progressively less exertion; the episodes also tend to be more intense and longer lasting than stable angina.
- unstable angina is associated with plaque disruption and superimposed partial thrombosis, distal embolization of the thrombus, and/or vasospasm.
- Unstable angina is the harbinger of more serious, potentially irreversible ischemia (due to complete luminal occlusion by thrombus) and is therefore sometimes called ***pre-infarction angina***.

- **Myocardial Infarction**
- MI *is necrosis of heart muscle resulting from ischemia.*
- Men are at significantly greater risk than women, although the gap progressively narrows with age.

- Pathogenesis
- *most MIs are caused by acute coronary artery thrombosis.*
- Sometimes, particularly with infarcts limited to the innermost (subendocardial) myocardium, thrombi may be absent. In these cases, severe diffuse coronary atherosclerosis significantly limits coronary vessel perfusion, and a prolonged period of increased demand (e.g., due to tachycardia or hypertension) may be sufficient to cause necrosis of myocytes most distal to the epicardial vessels.

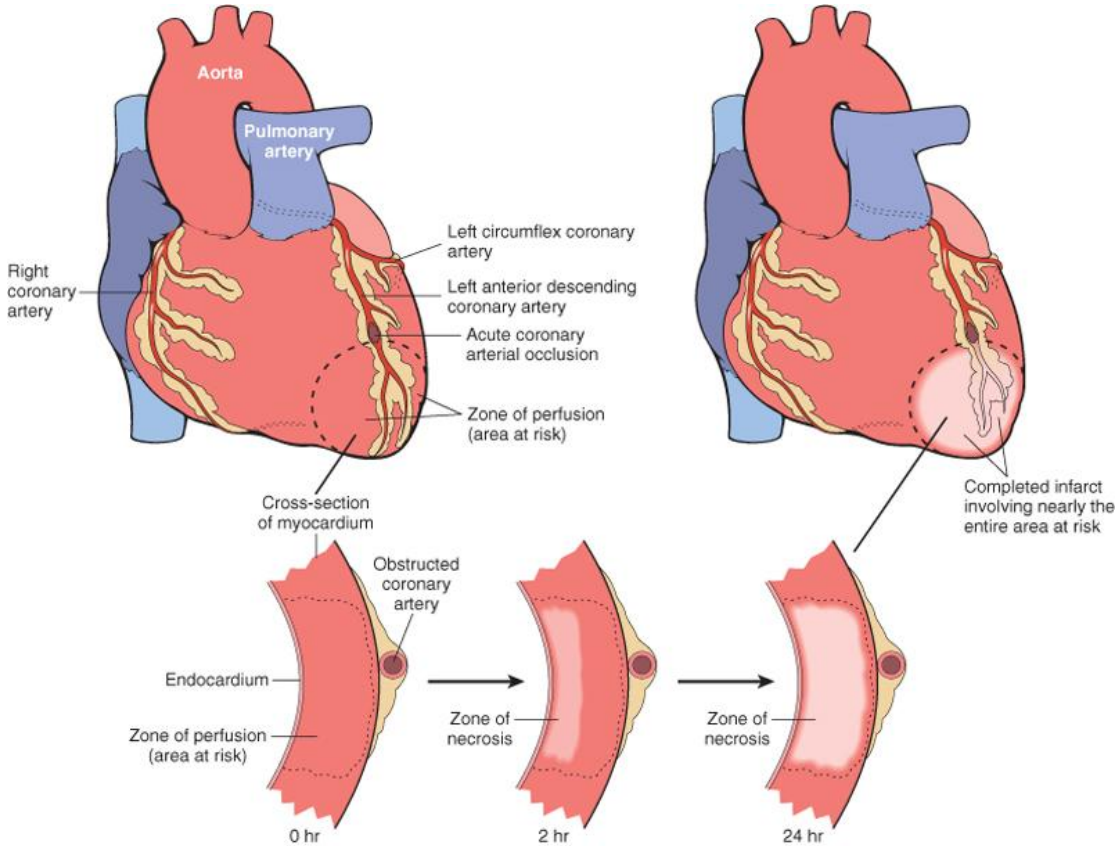
- Only severe ischemia lasting at least 20 to 40 minutes causes *irreversible* injury and myocyte death; the predominant pattern is coagulation necrosis . With longer periods of ischemia, microvasculature injury ensues.
- If myocardial blood flow is restored anywhere along this timeline (*reperfusion*), cell viability may be preserved.
- This provides the rationale for early clinical detection of acute MI, and prompt intervention by angioplasty or thrombolysis, to restore blood flow to the at-risk areas.

- Myocardial ischemia also contributes to arrhythmias, probably by causing *electrical instability (irritability)* of ischemic regions of the heart.
- Although massive myocardial damage can clearly cause a fatal mechanical failure, SCD in the setting of myocardial ischemia is most often (80% to 90% of cases) due to ventricular fibrillation caused by myocardial irritability.

- Irreversible injury of ischemic myocytes first occurs in the subendocardial zone???
- an infarct usually reaches its full size within 3 to 6 hours.

- The final location, size, and specific morphologic features of an acute MI depend on:

- Location, severity, and rate of development of the coronary occlusion
- Size of the vascular bed perfused by the obstructed vessels
- Duration of the occlusion
- Metabolic demands of the myocardium (affected, e.g., by blood pressure and heart rate)
- Extent of collateral supply.



Clinical Features

- An MI is usually heralded by severe, crushing substernal chest pain or discomfort that can radiate to the neck, jaw, epigastrium, or left arm.
- In contrast to the pain of angina pectoris, the pain of an MI typically lasts from 20 minutes to several hours and is not significantly relieved by nitroglycerin or rest.

- In a substantial minority of patients (10% to 15%) MIs can be entirely asymptomatic. Such "silent" infarcts are particularly common in patients with underlying diabetes mellitus (with peripheral neuropathies) and in the elderly.

- Dyspnea is common and is caused by impaired myocardial contractility and dysfunction of the mitral valve apparatus, with resultant pulmonary congestion and edema.
- With massive MIs (>40% of the left ventricle) cardiogenic shock develops.

- *Electrocardiographic abnormalities* are important markers of MIs; these include changes such as **Q waves** (indicating transmural infarcts), and **ST-segment** abnormalities and **T-wave** inversion (representing abnormalities in myocardial repolarization).

- Arrhythmias caused by electrical abnormalities of the ischemic myocardium and conduction system are common, and indeed, SCD due to a lethal arrhythmia accounts for the vast majority of deaths occurring before hospitalization.

- *Laboratory evaluation* of MI is based on measuring the blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membranes;

- **Troponins** and **CK-MB** have high specificity and sensitivity for myocardial damage.


Thank
you