

MI

*33% -50% die before hospital → lethal arrhythmia → Sudden Cardiac Death .

* Arrhythmias are caused by electrical abnormalities of the ischemic myocardium and conduction system.

***Acute occlusion of the proximal left anterior descending (LAD) artery is the cause of 40% to 50% of all MIs.**

* Frequency of MIs rises with increasing age and presence of risk factors such as hypertension, smoking, and diabetes.

*** Cardiac troponins T and I (TnT, TnI), are the best markers for acute MI.**

*** Creatine kinase CK-MB is the second best marker after the cardiac-specific troponins.**

***Microscopic changes of MI and its repair:**

- (<24H) → coagulative necrosis and wavy fibers. Necrotic cells are separated by edema fluid.

- 2- to 3-day old- infarct → Dense neutrophil infiltrate, **in case of reperfusion** → contraction bands

-(7 to 10 days) → complete removal of necrotic myocytes by **phagocytic macrophages**

-up to 14 days → **Granulation tissue** characterized by loose connective tissue and abundant capillaries.

-several weeks → Healed myocardial infarct consisting of a **dense collagenous scar**.

***Consequences and complications of MI:**

1- Death: 50% of the deaths associated with acute MI occur in individuals who never reach the hospital.

- Extraordinary progress has been made in patient outcomes subsequent to acute MI (their-hospital) death rate has declined from approximately 30% to an overall rate of between 10% and 13%).

2- Cardiogenic shock.

- (10% to 15%) of patients after acute MI - with a large infarct (>40% of the Left ventricle).

- 70% mortality rate; 2/3 of in-hospital deaths.

3-Myocardial rupture

a- rupture of the ventricular free wall → hemopericardium and cardiac tamponade (usually fatal)

b- rupture of the ventricular septum → VSD and left-to-right shunt

c- papillary muscle rupture → severe mitral regurgitation

4-Pericarditis.

- Fibrinous or hemorrhagic pericarditis, usually 2 to 3 days of a transmural MI

- Typically spontaneously resolves with time (immunologic mechanism)

5-Infarct expansion : Because of the weakening of necrotic muscle, there may be disproportionate stretching, thinning, and dilation of the infarct region (especially with anteroseptal infarcts).

6- Mural thrombus

- The combination of a local loss of contractility (causing stasis) + endocardial damage (causing a thrombogenic surface) → thromboembolism

7- Ventricular aneurysm :

- Most commonly result from a large transmural anteroseptal infarct that heals with the formation of thin scar tissue.

8- Papillary muscle dysfunction (post-infarct mitral regurgitation)

- Dysfunction of a papillary muscle after MI occurs due to: rupture, ischemic dysfunction , fibrosis and shortening , ventricular dilation.

9- Progressive late heart failure

***long term prognosis after MI:** depends on many factors, the most important of which are left ventricular function and the severity of atherosclerotic narrowing of vessels perfusing the remaining viable myocardium.

*Chronic IHD usually results from post-infarction cardiac decompensation that follows exhaustion of the hypertrophic viable myocardium.

***Coronary artery disease is the most common underlying cause of sudden cardiac death .**

* In many adults SCD is the first clinical manifestation of IHD.

TUMORS OF BLOOD VESSELS

1) Benign tumors (common) → Hemangioma .

- Contain vascular channels lined by normal-appearing endothelial cells.
- Common tumors composed of blood-filled vessels, most common infancy and childhood , most common head and neck, most are present from birth .
- Many regress spontaneously , possible internal organs (1/3 → liver), malignant transformation rare.

* HISTOLOGIC AND CLINICAL VARIANTS:

1- Capillary hemangiomas (most common type): skin and mucous membranes of oral cavity & lips.

2- Juvenile hemangiomas (strawberry hemangiomas) of newborn .

3- Pyogenic granulomas: rapidly growing pedunculated lesions on gingival mucosa .

4- Cavernous hemangiomas : large, dilated vascular channels; deep organs, do not spontaneously regress.

2) Borderline (locally aggressive)→ kaposi sarcoma .

- A vascular neoplasm caused by human herpesvirus- 8 = HHV-8
- Most common in patients with AIDS , used as a criterion for diagnosis of AIDS , The most common HIV-related malignancy.
- Multiple red-purple skin plaques or nodules, usually on the distal lower extremities; progressively increase in size and number and spread proximally.

3) Malignant (rare, mets) → angiosarcoma.

- more cellular, cytologic atypia, proliferative, do not form well-organized vessels.
 - Lesions can occur at any site, but **most often involve the skin, soft tissue, breast, and liver.**
 - A latent period between exposure and tumor development
- *Risk factors:** Chemical carcinogens(liver angiosarcoma), Irradiation , Lymphedema, foreign bodies

CARDIAC TUMORS

- Very rare
 - **Metastatic Neoplasms are the most common** malignancy of heart (5% of patients dying of cancer).
(lung cancer→ most common source)
 - **Angiosarcomas →most common primary malignant tumor of heart.**
 - Benign tumors are also very rare but important for their critical location.
- * Clinical features and significance** : "ball-valve" obstruction , Embolization ,fever and malaise→ tumor elaboration of interleukin-6.

Valvular Heart Disease

- * **Stenosis** is always due to a chronic process (e.g., calcification or valve scarring).
- * **Insufficiency** : failure of a valve to close completely → regurgitation (backflow) of blood.
- * **It can result from disease of either: valve cusps** (e.g., endocarditis) Or **supporting structures** (e.g. mitral annulus, tendinous cords, papillary muscles)
- * **It can be either: Acute** → e.g. chordal rupture Or **chronic** → e.g. scarring and retraction
- * **The most common congenital valvular lesion is bicuspid aortic valve.**
- * **Mitral valve is the most common target of acquired valve diseases.**
- * **Most important causes of acquired valvular diseases are postinflammatory scarring of the mitral valves and aortic valve due to (rheumatic fever) → 2/3 of all**
- * **bicuspid aortic valve disease:**
 - only two functional cusps instead of the normal
 - **early life** → Asymptomatic , **Later** → early and progressive degenerative calcification.
- * **Rheumatic fever -Rheumatic Valvular Disease.**
 - **immune- mediated inflammatory disease following group A β -hemolytic streptococcal infections.**
 - Hypersensitivity reaction due to antibodies against group A streptococcal antigens that are crossreactive with host antigens.
 - Major organs involved in **Rheumatic fever** : heart; joints; skin; and brain.
 - * **Acute rheumatic fever- clinical picture:** 80% of cases are children , Elevated serum titers of streptococcal antigens (streptolysin O; DNA-ase) , cultures for streptococci are (-) at the time of symptom onset.
 - **Aschoff bodies** are pathognomonic for rheumatic fever (collections of T lymphocytes).
- * **Chronic rheumatic carditis- clinical picture**
 - Onset: years or decades after initial acute episode.
 - **Symptoms:** cardiac murmurs ,CHF , arrhythmias (esp. Atrial fibrillation) , thromboembolism (mural thrombi).
 - Aschoff bodies rarely seen.
 - **Stenosis** is the most important functional consequence of chronic RHD.
 - **Mitral valve alone: 70% of cases (most common)**
 - combined mitral and aortic disease: 25%
 - tricuspid valve: less frequent, less severe
 - pulmonary valve: rarely involved

*** Infective endocarditis (IE)**

- Microbial (mostly bacterial) invasion of heart valves and endocardium.
- bulky, friable vegetations (necrotic debris+ thrombus+ organisms)
- **Most common: aortic and mitral valves , tricuspid valve common in I.V. drug abusers**

Features	Acute endocarditis	Subacute endocarditis
Virulence	a highly virulent organism	low virulent organism
Most common organism	Staph. aureus	Streptococcus viridans
underlying cardiac disease	previously normal valve	previously abnormal valve (scarred or deformed)
Clinical course	rapidly developing	Insidious disease
Outcome	High morbidity and mortality	most patients recover after appropriate antibiotic therapy

- Fever is the most consistent sign of infective endocarditis.
- Diagnosis = (positive blood cultures + echocardiographic (echo) findings).