MYOCARDIAL INFARCTION

INTRODUCTION

Acute myocardial infarction (MI) is the most important and feared consequence of coronary artery disease.

Many patients may die within the first few hours of the onset, while remainder suffer from effects of impaired cardiac function.

A significant factor that may prevent or diminish the myocardial damage is the development of collateral circulation through anastomotic channels over a period of time.

A regular and well-planned exercise programme encourages good collateral circulation and improved cardiac performance.

- Myocardial infarction is the myocardial necrosis occurring as a result of a critical imbalance between coronary blood supply and myocardial demand.
- It is usually due to the formation of an occlusive thrombus at the site of rupture of arthermatous plague in a coronary artery.

ETIOLOGY

- Tobacco smoking
- Hypertension
- Drug abuse
- Obesity
- Stress
- Alcohol
- Age
- Gender
- Diabetes
- Hyperlipoproteinaemia
- Family history of Ischaemic Heart Disease
- Hyperhomocysteinemia
- Chronic kidney disease

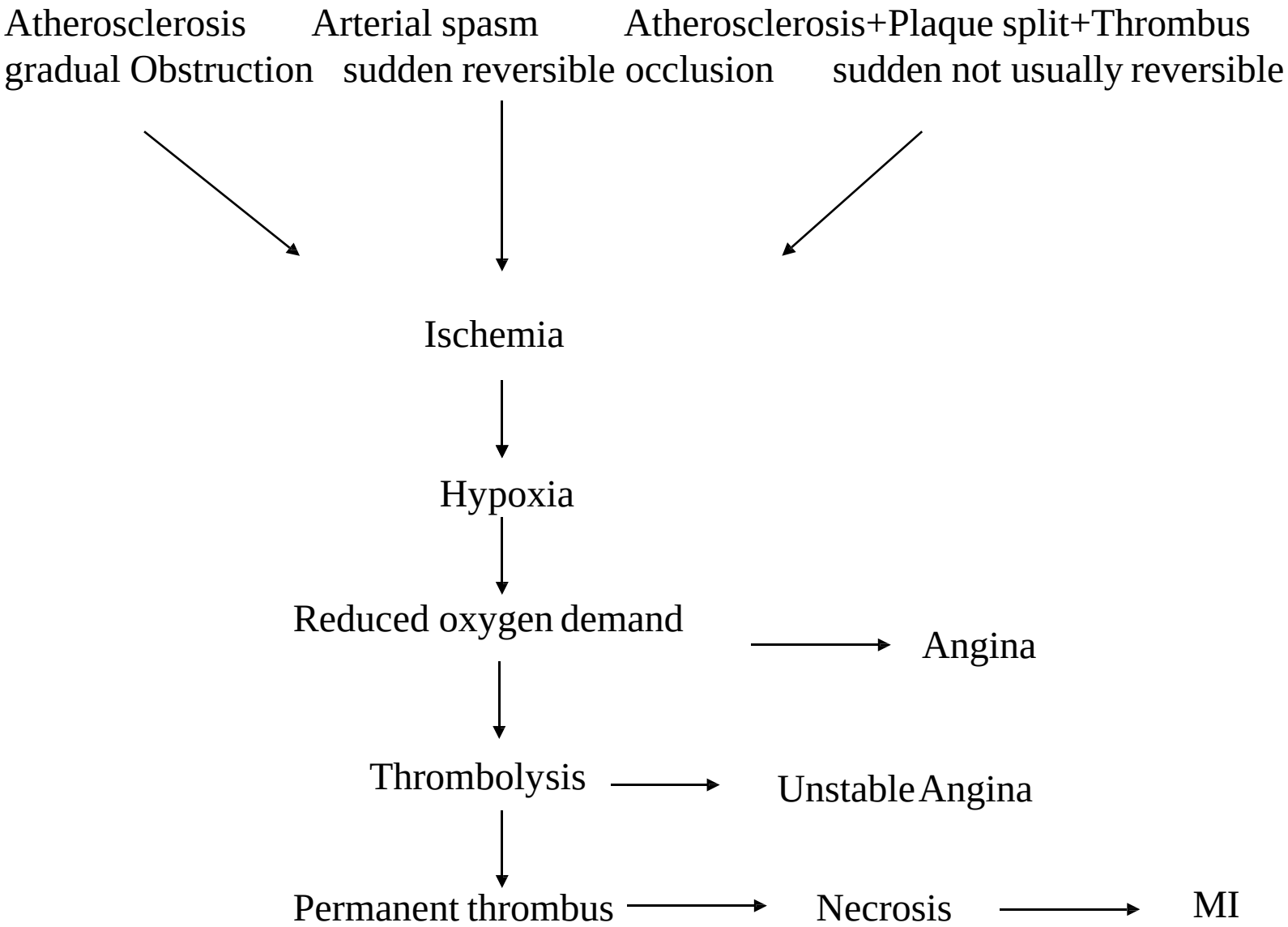
TYPES OF INFARCTS

1. According to anatomic region of left ventricle invoved:
- Anterior
- Posterior
- Lateral
- Septal
- Circumferential
- Combinations- Anterolateral, Posterolateral,Anteroseptal

2. According to degree of thickness of ventricular wall involved:
- Transmural (full thickness)
- Laminar (subendocardial)

3. According to age of infarcts:
- Newly formed (acute, recent, fresh)
- Advanced infarcts (old, healed, organised)

PATHOPHYSIOLOGY



A few notable features in the development of acute MI are

1. Myocardial ischaemia

Myocardial ischaemia is brought about by one or more of the following mechanisms:

i) Diminised coronary blood flow e.g. in coronary artery disease, shock.

ii) Increased myocardial demand e.g. in exercise, emotions.

iii) Hypertrophy of the heart without simultaneous increase of coronary blood flow e.g. in hypertension, valvular heart disease.

2. Role of platelets

Rupture of an atherosclerotic plaque exposes the subendothelial collagen to platelets which undergo aggregation, activation and release reaction. These events contribute to the build-up of the platelet mass that may give rise to emboli or initiate thrombosis.

3. Acute plaque rupture

Acute complications in coronary atherosclerotic plaques in the form of superimposed coronary thrombosis due to plaque rupture and plaque haemorrhage is frequently encountered in cases of acute MI:

i) Superimposed coronary thrombosis due to disruption of plaque is seen in about half the cases of acute MI. Infusion of intracoronary fibrinolysins in the first half an hour of development of acute MI in such cases restores blood flow in the blocked vessel in majority of cases.

ii) Intramural haemorrhage is found in about one-third cases of acute MI. Plaque haemorrhage and thrombosis may occur together in some cases.

4. Non-atherosclerotic causes

About 10% cases of acute MI are caused by non-atherosclerotic factors such as coronary vasospasm, arteritis, coronary ostial stenosis, embolism, thrombotic diseases, trauma and outside compression.

5. Transmural versus subendocardial infarcts

There are some differences in the pathogenesis of the transmural infarcts involving the full thickness of ventricular wall and the subendocardial (laminar) infarcts affecting the inner subendocardial one-third to half.

Contrasting features of subendocardial and transmural infarcts

	Feature	Transmural Infarct	Subendocardial Infarct
1.	*Definition*	Full-thickness, solid	Inner third to half, patchy
2.	*Frequency*	Most frequent (95%)	Less frequent
3.	*Distribution*	Specific area of coronary supply	Circumferential
4.	*Pathogenesis*	> 75% coronary stenosis	Hypoperfusion of myocardium
5.	*Coronary thrombosis*	Common	Rare
6.	*Epicarditis*	Common	None

CLINICAL FEATURES

Typically, acute MI has a sudden onset.

i) Pain: Usually sudden, severe, crushing and prolonged, substernal or precordial in location, unrelieved by rest or nitroglycerin, often radiating to one or both the arms, neck and back.

ii) Indigestion: Pain is often accompanied by epigastric or substernal discomfort interpreted as ‘heartburn’ with nausea and vomiting.

iii) Apprehension: The patient is often terrified, restless and apprehensive due to great fear of death.

iv) Shock: Systolic blood pressure is below 80 mmHg; lethargy, cold clammy limbs, peripheral cyanosis, weak pulse, tachycardia or bradycardia are often present.

v) Oliguria: Urine flow is usually less than 20 ml per hour.

vi) Low grade fever: Mild rise in temperature occurs within 24 hours and lasts up to one week, accompanied by leucocytosis and elevated ESR.

vii) Acute pulmonary oedema: Some cases develop severe pulmonary congestion due to left ventricular failure and develop suffocation, dyspnoea, orthopnoea and bubbling respiration.

COMPLICATIONS

- Arrhythmias
- CHF
- Mural thrombosis and thromboembolism
- Cardiogenic shock
- Rupture
- Cardiac aneurysm
- Pericarditis
- Dressler’s syndrome

DIAGNOSIS

The diagnosis of acute MI is made on the observations of 3 types of features—clinical features, ECG changes, and serum enzyme determinations.

ECG changes

The ECG changes are one of the most important parameters. Most characteristic ECG change is ST segment elevation in acute MI (termed as STEMI); other changes inlcude T wave inversion and appearance of wide deep Q waves.

Serum cardiac markers

Certain proteins and enzymes are released into the blood from necrotic heart muscle after acute MI. Measurement of their levels in serum is helpful in making a diagnosis and plan management.

Cardiac markers

- Creatinine phosphokinase (CPK)
- Lactate dehydrogenase(LDH)
- Cardiac-specific troponins (cTn)
- Myoglobin