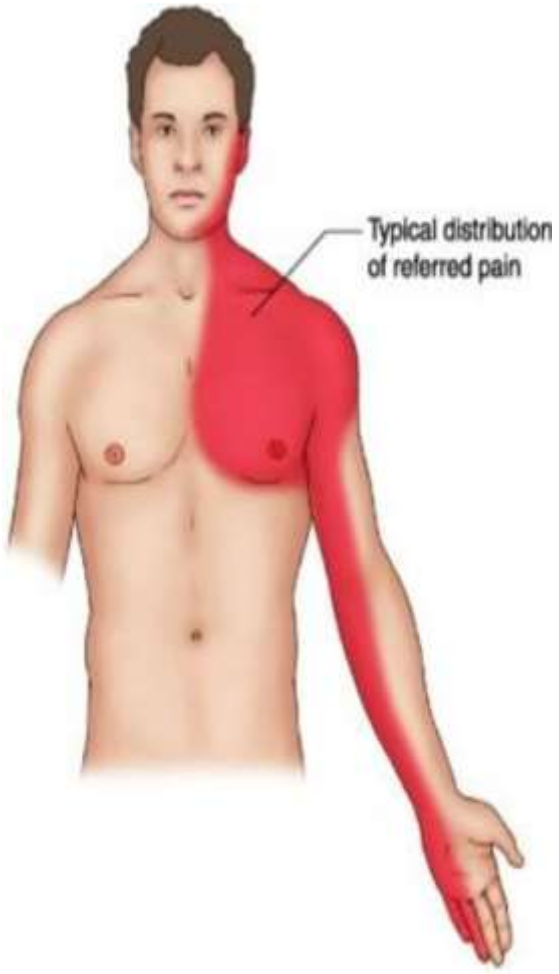

ANGINA PECTORIS

INTRODUCTION

Angina pectoris is a clinical syndrome of IHD resulting from transient myocardial ischaemia.

It is characterised by paroxysmal pain in the substernal or precordial region of the chest which is aggravated by an increase in the demand of the heart and relieved by a decrease in the work of the heart.

Often, the pain radiates to the left arm, neck, jaw or right arm. It is more common in men past 5th decade of life.



PATHOPHYSIOLOGY

Myocardial ischemia develops when coronary blood flow becomes inadequate to meet myocardial oxygen demand.

Heart rate, myocardial inotropic state, and myocardial wall tension are the major determinants of myocardial metabolic activity and myocardial oxygen demand. Increases in the heart rate and myocardial contractile state result in increased myocardial oxygen demand.

Increases in both afterload (ie, aortic pressure) and preload (ie, ventricular end-diastolic volume) result in a proportional elevation of myocardial wall tension and, therefore, increased myocardial oxygen demand.

This causes myocardial cells to switch from aerobic to anaerobic metabolism, with a progressive impairment of metabolic, mechanical, and electrical functions.

Angina pectoris is the most common clinical manifestation of myocardial ischemia. It is caused by chemical and mechanical stimulation of sensory afferent nerve endings in the coronary vessels and myocardium.

These nerve fibers extend from the first to fourth thoracic spinal nerves, ascending via the spinal cord to the thalamus, and from there to the cerebral cortex.

Adenosine may be the main chemical mediator of anginal pain. During ischemia, ATP is degraded to adenosine, which, after diffusion to the extracellular space, causes arteriolar dilation and anginal pain. Adenosine induces angina mainly by stimulating the A1 receptors in cardiac afferent nerve endings.

TYPES OF ANGINA

There are 3 overlapping clinical patterns of angina pectoris with some differences in their pathogenesis:

i) Stable or typical angina

ii) Prinzmetal’s variant angina

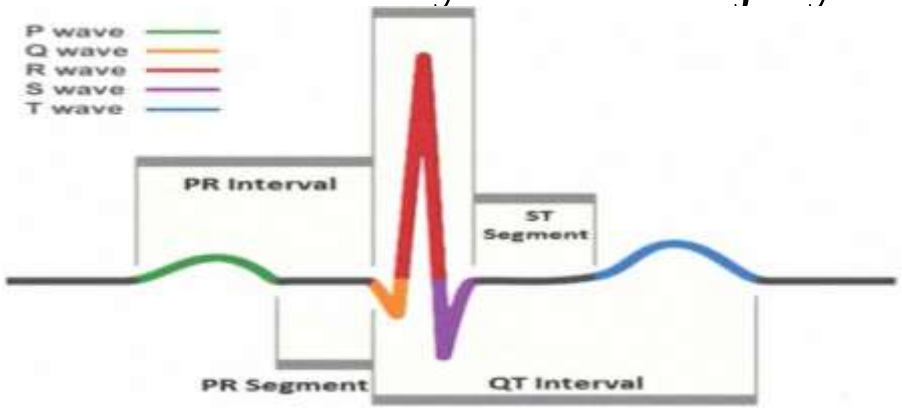
iii) Unstable or crescendo angina

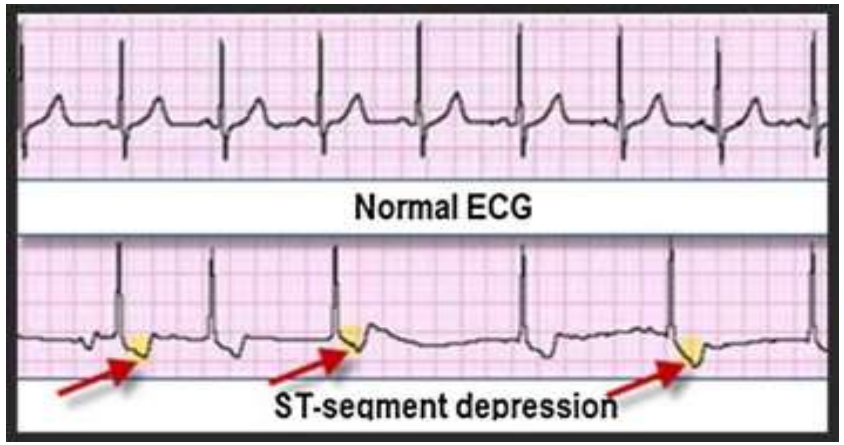
STABLE OR TYPICAL ANGINA

This is the most common pattern. Stable or typical angina is characterised by attacks of pain following physical exertion or emotional excitement and is relieved by rest.

The pathogenesis of condition lies in chronic stenosing coronary atherosclerosis that cannot perfuse the myocardium adequately when the workload on the heart increases.

During the attacks, there is depression of ST segment in the ECG due to poor perfusion of the sub endocardial region of the left ventricle but there is no elevation of enzymes in the blood as there is no irreversible myocardial injury.





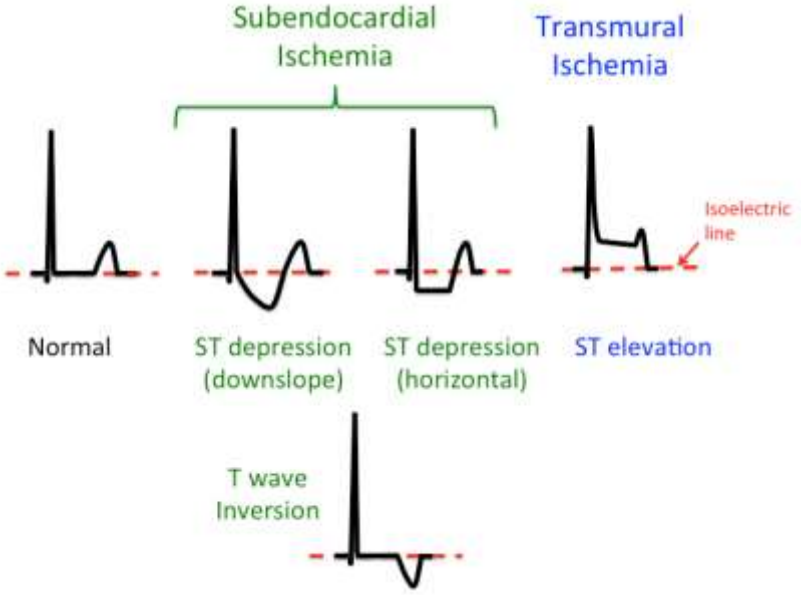
PRINZMETAL'S VARIANT ANGINA

This pattern of angina is characterised by pain at rest and has no relationship with physical activity.

The exact pathogenesis of Prinzmetal's angina is not known. It may occur due to sudden vasospasm of a coronary trunk induced by coronary atherosclerosis, or may be due to release of humoral vasoconstrictors by mast cells in the coronary adventitia.

ECG shows ST segment elevation due to transmural ischaemia.

These patients respond well to vasodilators like nitroglycerin.



UNSTABLE OR CRESCENDO ANGINA

Also referred to as ‘pre-infarction angina’ or ‘acute coronary insufficiency’, this is the most serious pattern of angina.

It is characterized by more frequent onset of pain of prolonged duration and occurring often at rest. It is thus indicative of an impending acute myocardial infarction.

Distinction between unstable angina and acute MI is made by ST segment changes on ECG— acute MI characterised by ST segment elevation while unstable angina may have non-ST segment elevation MI.

Multiple factors are involved in the pathogenesis of unstable angina which include: stenosing coronary atherosclerosis, complicated coronary plaques (e.g. superimposed thrombosis, haemorrhage, rupture, ulceration etc), platelet thrombi over atherosclerotic plaques and vasospasm of coronary arteries.

More often, the lesions lie in a branch of the major coronary trunk so that collaterals prevent infarction.

ANGINA DECUBITUS

Angina decubitus is a variant of angina pectoris that occurs at night while the patient is recumbent. Some have suggested that it is induced by an increase in myocardial oxygen demand caused by expansion of the blood volume with increased venous return during recumbency.

SIGNS AND SYMPTOMS

- Retrosternal chest discomfort (pressure, heaviness, squeezing, burning, or choking sensation) as opposed to frank pain
- Pain localized primarily in the epigastrium, back, neck, jaw, or shoulders
- Pain precipitated by exertion, eating, exposure to cold, or emotional stress, lasting for about 1-5 minutes and relieved by rest or nitroglycerin
- Pain intensity that does not change with respiration, cough, or change in position

DIAGNOSIS

- Electrocardiogram
- Exercise tolerance test
- Cardiac catheterization
- ECHO
- Cardiac imaging