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A COMPREHENSIVE REVIEW OF MYOCARDIAL INFARCTION

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ABSTRACT:

Myocardial infarction (MI), commonly known as a heart attack, is a leading cause of death and disability worldwide, predominantly resulting from coronary artery disease. MI occurs due to the interruption of blood flow to the myocardium, leading to myocardial cell death and necrosis. This comprehensive review explores the etiology, including modifiable and non-modifiable risk factors, and provides an epidemiological overview, highlighting prevalence rates across different demographics. The pathophysiology of MI is detailed, focusing on the mechanisms of coronary artery occlusion and subsequent myocardial damage.

KEYWORDS: Reperfusion Therapy, Risk Factors, Epidemiology, Pathophysiology, Complications

INTRODUCTION:

Myocardial infarction (MI), commonly known as a heart attack is the disease of the blood vessels supplying the heart muscle (Myocardium) i.e. coronary heart disease. The area of heart muscle that has either zero flow or so little flow that it cannot sustain cardiac muscle function is said to be infracted and the overall process is called a myocardial infarction.

Soon after the onset of the infarction, the area to become overfilled with stagnant blood as a combined effect of collateral blood flow through anastigmatic channel to the infracted area and progressive dilation of local blood vessels. Simultaneously the muscle fibres use the last vestiges of the oxygen in the blood, causing the haemoglobin to become totally de-oxygenated. Therefore, the infracted area takes on a bluish-brown. In later stages, the vessel walls become highly permeable and leak fluid; the local muscle tissue becomes edematous, and the cardiac muscle cells begin to swell because of diminished cellular metabolism.

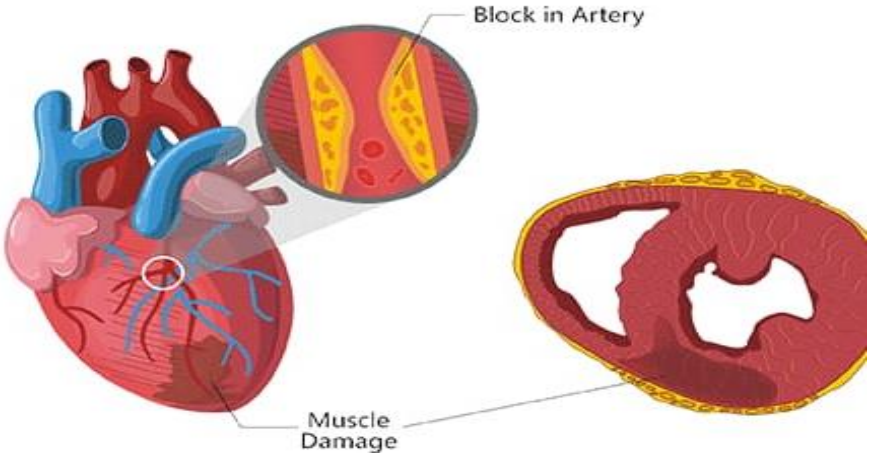


Figure.no :01 Overview of myocardial infarction

Within a few hours of almost no blood supply, the cardiac muscle cells die. Cardiac muscle requires about 1.3 milliliters of oxygen per 100 grams of muscle tissue per minute just to remain alive.The majority of myocardial infarctions are attributed to underlying coronary artery disease f(CAD), which remains the leading cause of death in the United States. In CAD, the coronary arteries that supply blood to the heart muscle become narrowed or blocked. When a coronary artery becomes occluded, the myocardium is deprived of oxygen. If this oxygen deprivation is prolonged, it can result in myocardial cell death and necrosis, significantly damaging the heart muscle[1].

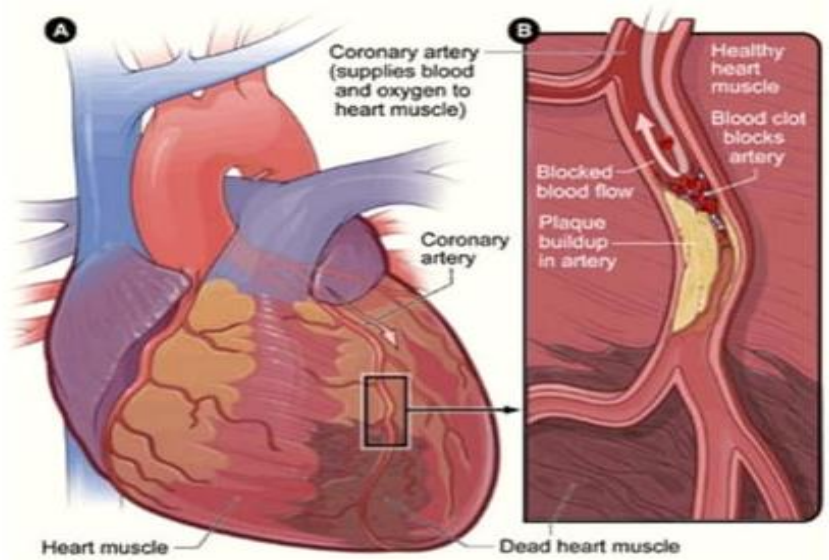


Figure.no:02 Blood clot blocks artery

TYPES OF MYOCARDIAL INFARCTION:

Type 1: Spontaneous myocardial infarction: Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis.

Type 2: Myocardial infarction secondary to an ischaemic imbalance: In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or

demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LVH.

Type 3: Myocardial infarction resulting in death when biomarker values are unavailable: Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.

Type 4a: Myocardial infarction related to percutaneous coronary intervention (PCI): Myocardial infarction associated with percutaneous coronary intervention is arbitrarily defined by elevation of cardiac troponin values >5 x 99th percentile URL in patients with normal baseline values (<=99th percentile URL) or a rise of cardiac troponin values >20% if the baseline values are elevated and are stable or falling.

Type 4b: Myocardial infarction related to stent thrombosis: Myocardial infarction associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/ or fall of cardiac biomarkers values with at least one value above the 99th percentile URL.

Type 5: Myocardial infarction related to coronary artery bypass grafting (CABG): Myocardial infarction associated with CABG is arbitrarily defined by elevation of cardiac biomarker values >10 x 99th percentile URL in patients with normal baseline cTn values (<=99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormalities[2].

STAGES OF MYOCARDIAL INFRACTION:

- **Stage 1: Aborted MI (no/minimal myocardial necrosis):**

No or minimal damage to the heart muscle. In the best case the entire area of myocardium at risk may be salvaged.

- **Stage 2: MI with significant cardiomyocyte necrosis, but without microvascular injury:**

Damage to the heart muscle and no injury to small blood vessels in the heart. Revascularization therapy will result in restoration of normal coronary flow.

- **Stage 3: MI with cardiomyocyte necrosis and microvascular dysfunction leading to microvascular obstruction (i.e., "no-reflow"):**

Damage to the heart muscle and blockage of small blood vessels in the heart. The major adverse cardiac event rate is increased 2- to 4-fold at long-term follow-up.

- **Stage 4: MI with cardiomyocyte and microvascular necrosis leading to reperfusion hemorrhage:**

Damage to the heart muscle, blockage and rupture of small blood vessels resulting in bleeding into the heart muscle. This is a more severe form of microvascular injury, and the most severe form of ischemia-reperfusion injury. It is associated with a further increase in adverse cardiac event rate of 2- to 6-fold at long-term follow-up[3]

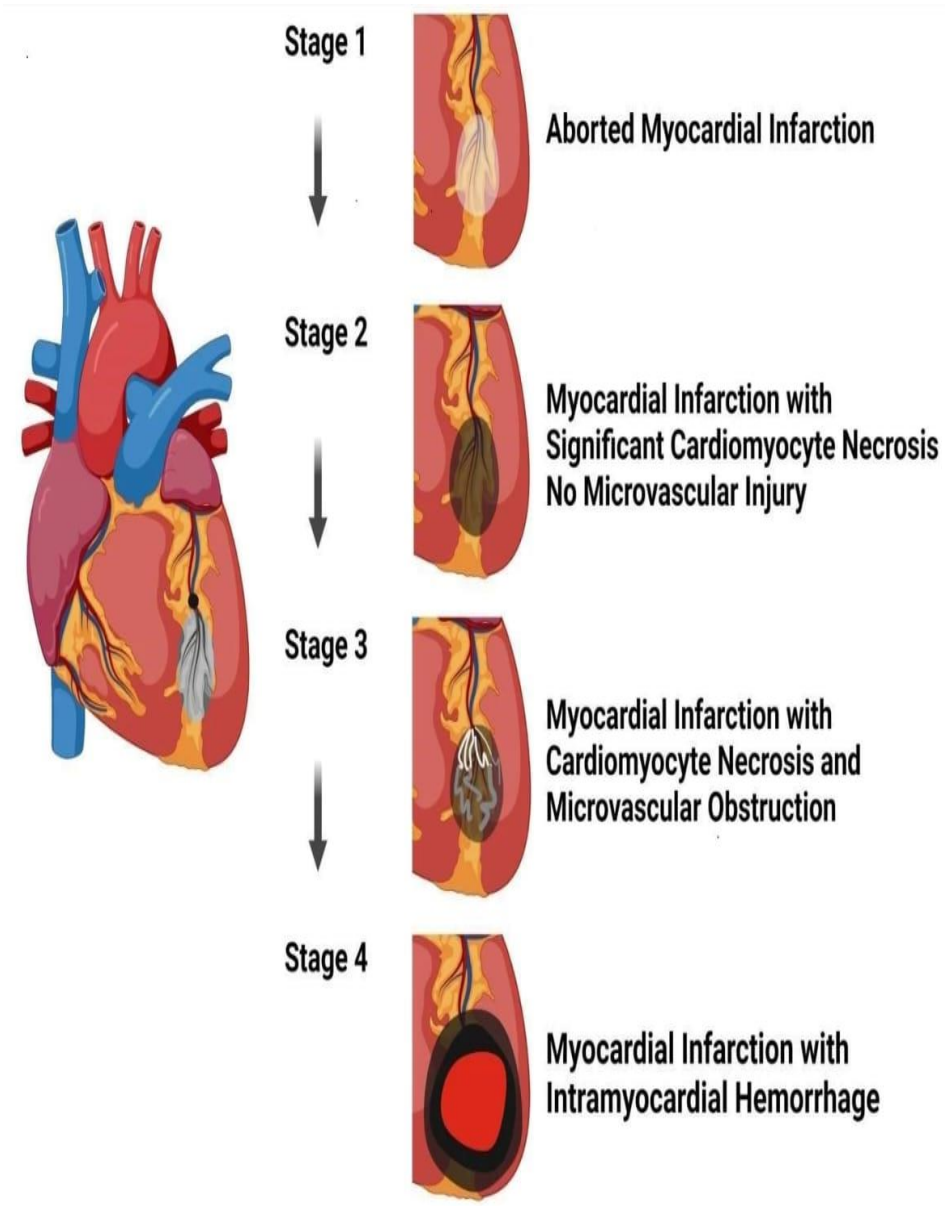


Figure.no:05 Stages of myocardial infarction

EPIDEMIOLOGY:

MI is a common presentation of ischemic heart diseases. In industrial countries MI accounts for 10-25% of all deaths. Incidence is higher in elderly people, about 5% occurs at people under age 40. Males have higher risk. Women during reproductive period have low risk. In 2006, studies revealed a prediction that would account for 40-60% of cardiovascular diseases burden within next 10-15 years. Over last 30 years, the rate of diseases increased from 2-6% in rural population and 4-12%in urban population.

The WHO estimated that in 2002, 12.6 percent of deaths worldwide were from ischemic heart disease. In the United States, diseases of the heart are the leading cause of death, causing a higher mortality than cancer (malignant neoplasm). Coronary heart disease is responsible for 1 in 5 deaths in the U.S. This means that roughly every 65 seconds, an

American dies of a coronary event. In India, cardiovascular disease (CVD) is the leading cause of death. The deaths due to CVD in India were 32% of all deaths in 2007 and are expected to rise from 1.17 million in 1990 and 1.59 million in 2000 to 2.03 million in 2010[4].

ETIOLOGY:

As stated above, myocardial infarction is closely associated with coronary artery disease. INTERHEART is an international multi-center case-control study which delineated the following modifiable risk factors for coronary artery diseases:

1. Smoking
2. Abnormal lipid profile/blood apolipoprotein (raised ApoB/ApoA1)
3. Hypertension
4. Diabetes mellitus
5. Abdominal obesity (waist/hip ratio) (greater than 0.90 for males and greater than 0.85 for females)
6. Psychosocial factors such as depression, loss of the locus of control, global stress, financial stress, and life events including marital separation, job loss, and family conflicts
7. Lack of daily consumption of fruits or vegetables
8. Lack of physical activity
9. Alcohol consumption (weaker association, protective)

The INTERHEART study showed that all the above risk factors were significantly associated with acute myocardial infarction except for alcohol consumption, which showed a weaker association. Smoking and abnormal apolipoprotein ratio showed the strongest association with acute myocardial infarction. The increased risk associated with diabetes and hypertension were found to be higher in women, and the protective effect of exercise and alcohol was also found to be higher in women.

Other risk factors include a moderately high level of plasma homocysteine, which is an independent risk factor of MI. Elevated plasma homocysteine is potentially modifiable and can be treated with folic acid, vitamin B6, and vitamin B12[5].

ETIO-PATHOGENESIS:

Atherosclerotic causes

- Accounts for 75% of cases
- Fatty streak deposits on the coronary artery
- Endothelium develop into an atherosclerotic plague depending on the presence of risk factors
- Risk factors – HT, DM, Smoking, Hyperlipidaemia
- Plaque progression, proliferation and disruption of integrity of blood vessel
- Results in narrowing off coronary artery & MI

Non atherosclerotic causes

- Accounts for 10% of the causes of MI
- Coronary vasospasm
- Inflammation of arteries
- Coronary embolism
- Development of thrombosis
- Injury[6]

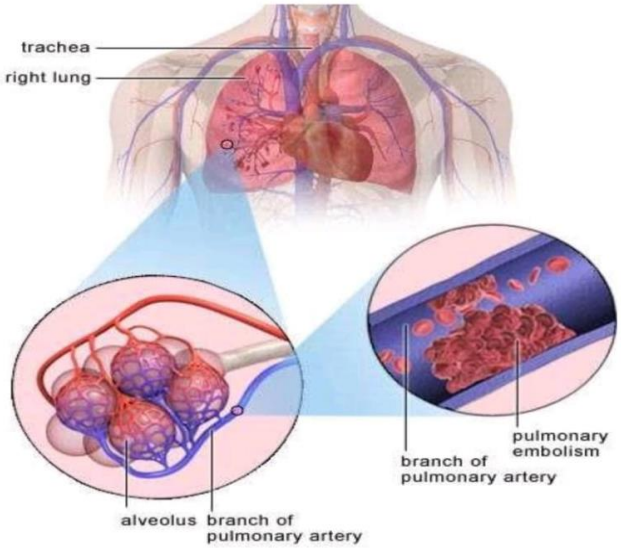
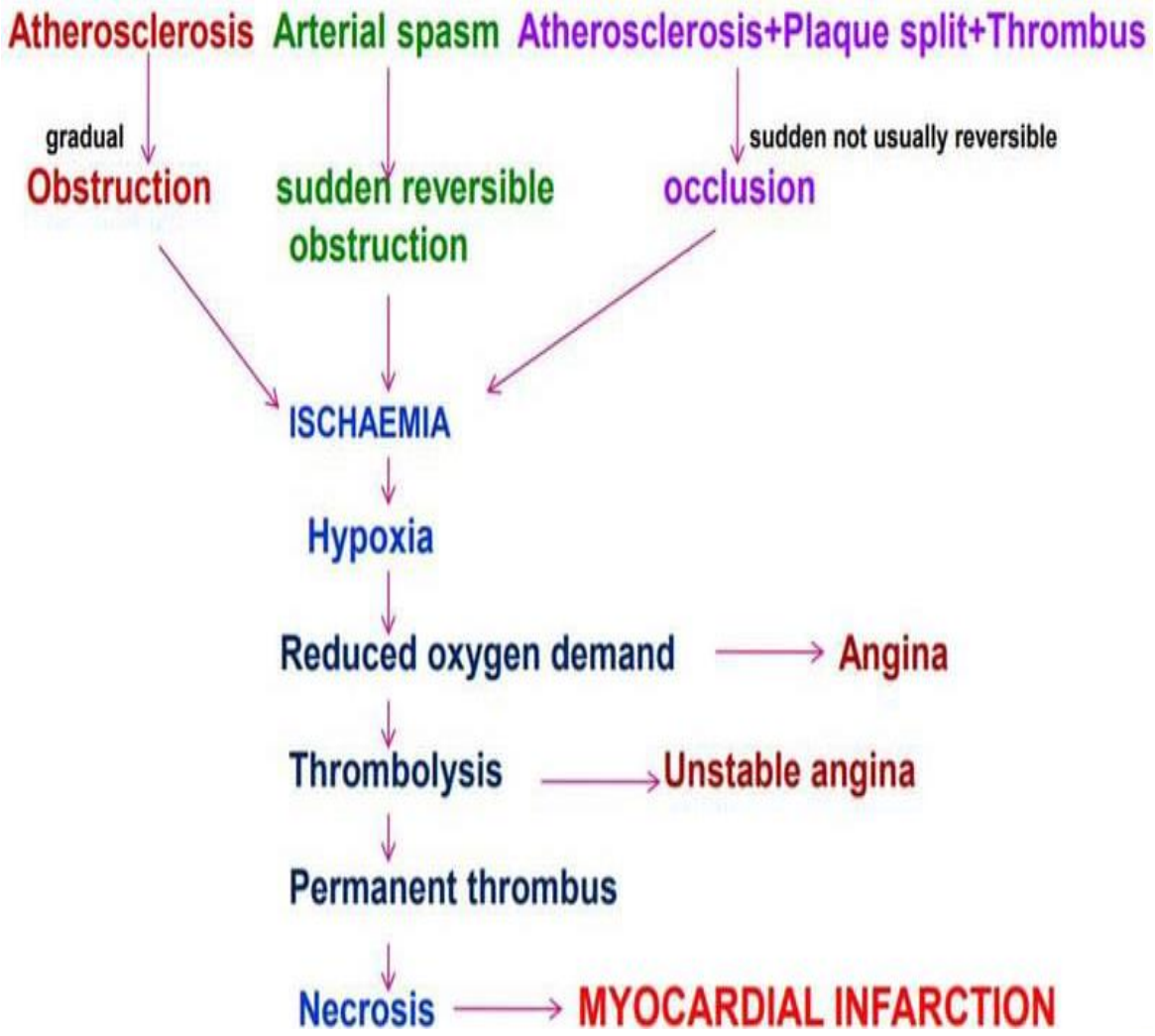


Figure.no:06 Etio-Pathogenesis of myocardial infarction

PATHOPHYSIOLOGY:

- The acute occlusion of one or multiple large epicardial coronary arteries for more than 20 to 40 minutes can lead to acute myocardial infarction.
- The occlusion is usually thrombotic and due to the rupture of a plaque formed in the coronary arteries. The occlusion leads to a lack of oxygen in the myocardium, which results in sarcolemmal disruption and myofibril relaxation.
- These changes are one of the first ultrastructural changes in the process of MI, which are followed by mitochondrial alterations.
- The prolonged ischemia ultimately results in liquefactive necrosis of myocardial tissue.
- The necrosis spreads from sub-endocardium to sub-epicardium.
- The subepicardium is believed to have increased collateral circulation, which delays its death[7].

Figure.no:07



Pathophysiology of myocardial infarction

CLINICAL MANIFESTATIONS:

Common warning signs of a heart attack include:

- Chills or cold sweat
- Chest discomfort or pain that feels like pressure, squeezing or clenching, or fullness
- Feeling dizzy or light-headed
- Nausea or vomiting
- Pain or discomfort in the chest or upper body that may spread to the jaw or arms
- Prolonged and severe central chest pain (heavy and crushing sensation)
- Shortness of breath, which may occur on its own or with chest pain

While chest discomfort or pain is the most common symptom, women are more likely to experience shortness of breath, nausea, vomiting, and back or jaw pain[8].

RISK FACTORS:

There are modifiable and non-modifiable risk factors associated with a heart attack.

Modifiable risk factors include:

- **Cigarette smoking.** Smoking and exposure to second-hand smoke damages the inner walls of the arteries, allowing plaque to accumulate.
- **Obesity.** A sedentary lifestyle or a lack of physical exercise contributes to obesity and high cholesterol levels.
- **Chronic conditions**

such as:

- High blood pressure, which causes the heart to work harder and the heart muscles to thicken and harden. This accelerates the process of atherosclerosis.
- High cholesterol, which is a major component of plaque.
- Diabetes, which is linked to an increased risk of heart attack and other heart problems, especially if blood sugar levels are poorly controlled.

Non-modifiable risk factors include:

- **Age.** Men aged 45 or older and women aged 55 or older are more likely to have a heart attack than younger Adults.

- **Strong family history of the disease.** If one of your immediate family members, such as a parent or sibling, has had a heart attack, or was diagnosed with heart disease before the age of 60, this may indicate a family history of premature heart disease. This means that your chances of developing heart disease may be higher than the normal population.
- **Ethnicity.** African Americans, Hispanics, Latinos and Southeast Asians are ethnic groups with an increased risk of CAD morbidity and mortality.
- **Gender.** Men are 3 – 5 times more prone to having a heart attack than women.
- **Menopause.** Loss of natural oestrogen increases a woman's risk of heart disease.

If you have any of the risk factors, it is important to go for regular heart screening with a specialist to ensure a healthy heart. Talk to your cardiologist to find out more[9].

DIAGNOSIS:

Electrocardiogram:

Electrocardiogram (ECG) is a valuable tool in the diagnosis and management of myocardial infarction (MI). ECG changes can help identify the location and extent of myocardial damage, and guide the selection of appropriate treatment strategies. Here are some of the common ECG changes seen in MI:

• **ST-segment elevation:** ST-segment elevation is a hallmark of acute MI and indicates transmural myocardial damage. ST-segment elevation is most commonly seen in ST-segment elevation MI (STEMI) and is usually accompanied by T-wave inversion and Q-wave formation.

• **ST-segment depression:** ST-segment depression is seen in non-ST-segment elevation MI (NSTEMI) and indicates subendocardial myocardial damage.

• **T-wave inversion:** T-wave inversion is a common ECG finding in MI and can be seen in both STEMI and NSTEMI. T-wave inversion usually reflects the presence of ischemia or injury in the affected myocardium.

• **Q-wave formation:** Q-wave formation is a late ECG finding in MI and indicates irreversible myocardial damage. Q-wave formation is seen in the leads that face the affected myocardium and can persist for several months or years after the MI.

• **Other changes:** Other ECG changes seen in MI include atrial and ventricular arrhythmias, conduction disturbances, and ST-segment elevation or depression in leads opposite to the affected myocardium. The ECG changes in MI can evolve over time, and serial ECGs can provide valuable information on the progression and resolution of myocardial damage[10].

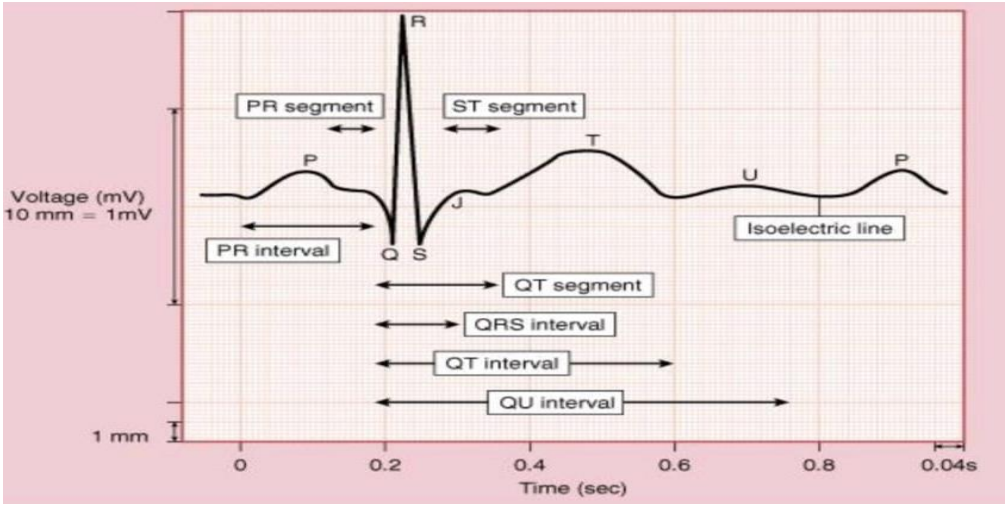


Figure.no:09 Electrocardiogram graph-1

Magnetic resonance imaging(MRI):

MR imaging is valuable in establishing a diagnosis of chronic MI and distinguishing this condition from nonischemic cardiomyopathies, mainly through use of delayed-enhancement patterns. MR imaging also provides clinicians with several prognostic indicators that enable risk stratification, such as scar burden, microvascular obstruction, hemorrhage, and peri-infarct ischemia. The extent and transmurality of scar burden have been shown to have independent and incremental prognostic power over a range of left ventricular function. The extent of scarring at MR imaging is an important predictor of successful outcome after revascularization procedures, and extensive scarring in the lateral wall indicates poor outcome after cardiac resynchronization therapy[11].

Cardiac imaging:

Current diagnostic criteria for MI include the presence of new regional wall motion abnormalities in echocardiography, myocardial scars in MRI or in nuclear tests or an intracoronary thrombus during coronary angiography combined with a significant rise and/or fall of cardiac troponin. Echocardiography is also an important

tool for diagnosis of non-ischemic causes of chest pain such as myocarditis, valvular disease, cardiomyopathies, pulmonary embolism or aortic dissection. Furthermore, echocardiography is the method of choice for detection of complications such as ventricular wall rupture or secondary mitral valve regurgitation after papillary muscle rupture or ischemia.

Coronary angiography (also called a cardiac catheterization):

Is a minimally invasive procedure that uses a catheter (a long, thin flexible tube) inserted into a blood vessel in the leg, arm, or neck to take pictures of the coronary artery opening. This test allows doctors to measure the width of the artery and rate of blood flow. Contrast dye is used to make it easier to see and evaluate the artery opening. Your doctor may go ahead and perform a procedure called an angioplasty, or stent, if a blockage is found during angiography[12].

Echocardiogram:

Is a non-invasive test using ultrasound (sound waves) and a device called a transducer — which is placed on the surface of the chest — to create a moving picture of the heart. It shows areas of the heart muscle or valves damaged by the heart attack[13].

Positron Emission Tomography (PET):

PET imaging was performed with the ECAT II tomography (CT! Knoxville, TN). Regional myocardial blood flow was assessed with N-13 ammonia (20 mCi) and exogenous glucose utilization with F-18 2-deoxyglucose (10 mCi) after an overnight fast as previously described. Cross-sectional imaging following each tracer injection was performed at corresponding levels. The images were analyzed with an operated-interactive program using circumferential profile techniques. Observed normalized counts in each PET scan were compared to the limits established from a normal database. Regional reductions in N-13 ammonia uptake below 2 SD identified segments of reduced blood flow.

Persistence or absence of metabolic activity in these segments were defined by regional F-18 2-deoxyglucose uptake. Scar tissue was defined as concordant segmental reduction of N-13 and F-18 counts of at least 2 SD below normal in two or more adjacent segments. Tissue viability was defined as the F-18/N-13 count ratio greater than 2 SD above normal in two or more adjacent segments[14].

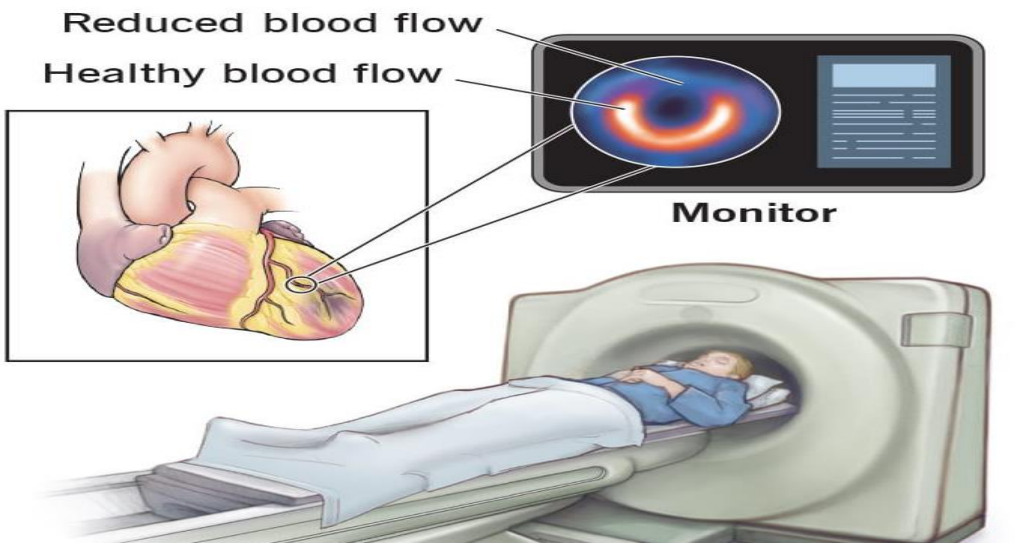


Figure.no:10 positron emission tomography imaging test

TREATMENT:

The treatment of myocardial infarction (MI) aims to restore blood flow to the affected part of the heart, relieve symptoms, prevent complications, and reduce the risk of future cardiovascular events. The treatment of MI involves a combination of pharmacological and interventional therapies, as well as lifestyle modifications. Here are the different treatment options for MI with sources:

❖ **Reperfusion therapy:**

Reperfusion therapy is the restoration of blood flow to the affected part of the heart and is the cornerstone of the treatment of MI. Reperfusion therapy can be achieved either by pharmacological thrombolysis or by percutaneous coronary intervention (PCI), also known as angioplasty. Both thrombolysis and PCI have been shown to improve outcomes in patients with ST-segment elevation MI (STEMI).

➢ **Thrombolytic agent:** also at high risk. All of these patient groups are at high absolute risk and are likely to benefit substantially from thrombolysis, which reduces mortality and preserves left ventricular function.24 A patent infarct-related artery has the potential to provide collaterals to another infarct zone in the event of subsequent coronary occlusion and can decrease arrhythmogenesis and remodeling of the left ventricle.

➢ **Fibrinolytic agent:** Four fibrinolytic agents are approved for the treatment of STEMI in the United States-streptokinase, alteplase, reteplase, and tenecteplase. Several clinical trials have demonstrated the beneficial effects of these therapies in reducing mortality rates in patients with suspected acute myocardial infarction[15].

❖ **Antiplatelet therapy:**

Antiplatelet therapy, such as aspirin and P2Y12 inhibitors (clopidogrel, ticagrelor, and prasugrel), is used to prevent further thrombotic events and reduce the risk of recurrent MI. Dual antiplatelet therapy (DAPT) with aspirin and a P2Y12 inhibitor is recommended for at least 12 months after PCI in patients with STEMI.

➢ **Clopidogrel:** Clopidogrel is a prodrug of a platelet inhibitor used to reduce the risk of myocardial infarction and stroke. Clopidogrel is indicated to reduce the risk of myocardial infarction for patients with non-ST elevated acute coronary syndrome (ACS), patients with ST-elevated myocardial infarction, and in recent MI, stroke, or established peripheral arterial disease.

❖ **Anticoagulant therapy:**

Anticoagulant therapy, such as unfractionated heparin or low molecular-weight heparin, is used to prevent thrombus formation and reduce the risk of recurrent MI.

➢ **Heparin:** Heparin is an anticoagulant medication that is commonly used in the treatment of myocardial infarction (MI). When intravenous heparin is administered for myocardial infarction with non-ST elevation and unstable angina, an initial bolus of 60 to 70 U/kg (maximum, 5000 U) followed by a 12- to 15-U/kg/h infusion is recommended[16].

❖ **Beta-blockers:**

Beta-blockers are used to reduce the workload on the heart, decrease myocardial oxygen demand, and improve survival in patients with MI. Beta-blockers are recommended for all patients with MI, unless contraindicated.

➢ **Metoprolol:** Metoprolol is a beta-blocker medication that is commonly used in the treatment of myocardial infarction (MI). Here are some key points about metoprolol in the treatment of MI: Acute MI is a metoprolol is indicated for the treatment of acute MI, particularly in patients with ST-elevation MI (STEMI) or non-ST-elevation MI (NSTEMI). Secondary prevention is a Metoprolol is also used for secondary prevention of MI, to reduce the risk of recurrent MI and death.

❖ **ACE inhibitors or ARBs:**

Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) are used to reduce mortality and prevent heart failure in patients with MI. ACE inhibitors or ARBs are recommended for all patients with MI, unless contraindicated.

➢ **Captopril**: Is also used to help treat heart failure. It is also used in some patients after a heart attack. After a heart attack, some of the heart muscle becomes damaged and weak. The heart muscle may continue to weaken as time goes by. This makes it more difficult for the heart to pump blood. Captopril may be started within the first few days after a heart attack to increase survival rate.

❖ **Statins:**

Statins are used to reduce cholesterol levels and prevent future cardiovascular events in patients with MI. Statins are recommended for all patients with MI, regardless of their cholesterol levels[17].

CONCLUSION:

Myocardial infarction (MI) is a serious and potentially life-threatening condition that occurs when there is a blockage in one or more of the coronary arteries, which supply blood and oxygen to the heart muscle. MI can lead to significant morbidity and mortality if not treated promptly and appropriately. This new classification includes several types of MI, based on the underlying mechanism and clinical presentation. MI is a major public health problem globally, with a significant burden on healthcare resources and the economy. In India, MI is a leading cause of mortality and morbidity. The pathophysiology of MI involves a complex interplay of factors, including atherosclerosis, thrombosis, and inflammation.

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