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## Myocardial Infraction: Etiology, Risk Factors, Pathophysiology, Diagnosis and Management

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### ABSTRACT

Cardiovascular disease is considered the major cause of morbidity and mortality throughout the world. Also myocardial infraction is the main health problem. In 2015, about 15.9 million myocardial infraction occurred throughout the world. In the United States about one million people have an MI each year. Modifiable risk factors include high blood pressure, smoking, diabetes, lack of exercise, obesity, depression, high blood cholesterol, poor diet, life style and excessive alcohol intake. Family history is also responsible for cardiovascular disease. Reperfusion injury include coronary thrombus formation followed by thrombolytic therapy. By the physical examination with electrocardiogram findings and cardiac markers MI is diagnosed. Therapeutic intervention such as pharmacologic, non-pharmacologic and combination therapy improves the clinical outcomes in MI patient. This review focus on the risk factors, pathophysiology in relation to produce myocardial injury and on the cardioprotective treatment.

**Keywords:** Myocardial infraction, Pathophysiology, risk factors, Cardiac injury, Management.

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## INTRODUCTION

Cardiovascular Disease (CVD) is a global health problem having high mortality and morbidity rate. As per World Health Organization reports there were 17,700,000 deaths due to CVD in 2015 [1]. Myocardial infraction (MI) is defined as the necrosis in the myocardium due to the lack of the oxygen supply of heart which cannot be supplied by the coronary artery [2]. It is also known as a heart attack that is sudden block in blood flow in the coronary arteries. If the block is severe, the heart can stop beating. It is characterized by chest pains or discomfort which may travel into the shoulder, arm, back, neck or jaw [3,4,5]. This type of pain always starts from the center or left side of the chest where heart is present and remains for few minutes. We distinguish between two types of MI on which clinical decision-making is based : ST-Elevation Myocardial Infraction (STEMI) and Non ST-Elevation Myocardial infraction (NSTEMI). Furthermore, MI is classified into 5 types based on its pathophysiology, clinics and prognostics :[6]

### Type I : Spontaneous MI

Atherosclerotic plaque disturbance resulting in thrombus formation and decreased myocardial blood flow or distal platelet emboli with ensuring myocyte necrosis.

### Type II : MI secondary to an ischemic imbalance

Imbalance between myocardial oxygen supply and demand due to other conditions eg. Coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy-/brady-arrhythmias, heart failure, anemia, respiratory failure, hypotension, hypertension, renal failure.

Type III : Myocardial infraction resulting in death when biomarker values are unavailable.

Type IVa : Myocardial infraction related to percutaneous coronary intervention (PCI)

Type IVb : Myocardial infraction related to stent thrombosis

Type V : Myocardial infraction related to coronary artery bypass grafting (CABG).

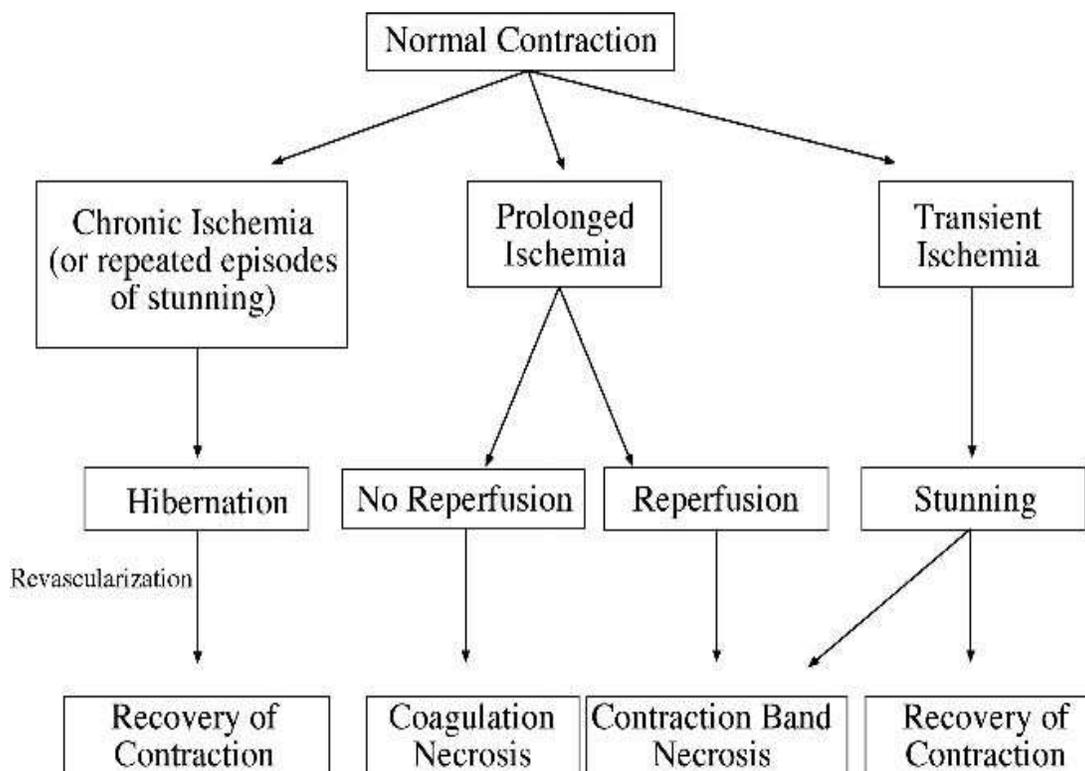


**Figure 1. Myocardial Infraction**

## History and Physical

The imbalance between oxygen supply and the demand leads to myocardial ischemia and can sometimes lead to myocardial infarction. Ischemic symptoms identify by the patients history, electrocardiographic findings and elevated serum biomarkers. In Myocardial ischemia chest pain, upper extremity pain, mandibular or epigastric discomfort that occurs during exertion or at rest. Myocardial ischemia can also present as dyspnea or fatigue, which are known to be ischemic equivalents. The chest pain is usually retrosternal and is sometimes described as the sensation of pressure or heaviness. The pain often radiates to the left shoulder, neck or arms with no obvious precipitating factors and it may be intermittent or persistent. The pain usually last for more than 20 minutes. It is usually not affected by positional changes or active movement of the region. Additional symptoms, such as sweating, nausea, abdominal pain, dyspnea and syncope may also be present. The MI can also present atypically with subtle findings such as palpitations, or more dramatic manifestations such as cardiac arrest. The MI can sometimes present with no symptoms [7].

## Etiology of Reperfusion Injury



**Figure 2. Varying effects of ischemia on contraction.**

There is number of conditions may elicit an ischemic heart disease. For example, atherosclerosis of the coronary arteries- coronary artery disease- reduces the effective coronary artery lumen diameter. Blood flow is significantly decreased, and ischemia occurs when > 75% of the luminal surface area is lost. Necrosis of the myocardium follows when complete loss of blood flow occurs for >20 minutes. Thus, it is imperative that the ischemic myocardium be reperfused before necrosis occurs, for early myocardial reperfusion improves cardiac contractile function and decreases infarct size (Figure) [8]. The latter is defined as death cardiac myocytes that were viable before reperfusion but succumbed to one or more events initiated by reperfusion [9,10,11].

Conditions that reduce coronary blood flow may lead to an ischemic myocardium. Possible scenarios inducing reperfusion injury include coronary thrombus formation, followed by thrombolytic therapy (urokinase, streptokinase, or tissue plasminogen activator). If the obstruction is relieved then reperfusion injury may occur because of coronary blood flow is restored before necrosis. Other instances where ischemia is followed by reperfusion include : 1) coronary artery vasospasm followed by coronary artery dilation; 2) arteriosclerotic coronary angioplasty and stenting; and 3) surgical revascularization via coronary artery bypass grafting. Global myocardial ischemia can result from application of an aortic cross clamp during cardiopulmonary bypass (CPB). Global myocardial ischemia also occurs in transplanted heart (Donor heart) [12]. When number of cardiac muscle cells die, workload for cells is increased. The compensatory response to this increased workload is hypertrophy, where in the myocytes can double in size [13,14] The increased workload imposed upon the myocardium can ultimately lead to depressed cardiac function of previously uninvolved areas of the heart [15]. Myocyte proliferation may replace damaged myocardium, and that mitosis in healthy myocardium implies that myocytes are replaced throughout the life of the human [16].

### **Risk Factors**

There are various risk factors of MI. Among them, some are modifiable (treatable) and others are non-modifiable (can not be changed). The major risk factors of MI are described here;

#### **Physical Activity:**

Multiple cardiac risk factors like MI more in inactive people [17]. Risk of coronary heart disease may reduce upto 20%-30% by physical activity [18,19]. However, studies have shown that different types of physical activities may have different effects on the risk of cardiovascular disease and may interact together. For example, some physical activities such as walking, stair climbing, and cycling provide protection against cardiovascular disease [20-23].

#### **Smoking :**

Premature atherosclerosis and sudden cardiac death because of smoking which is strong risk factor of myocardial infraction. Smoking results in early STEMI especially in otherwise healthier patients [24,25]. There are multiple and complex mechanism for increase the risk of MI by cigarette smoking [26]. Serum LDL-cholesterol and triglyceride concentrations increases and serum HDL-cholesterol reduces by smoking. Furthermore, cigarette smoke promotes free radical damage to LDL, leading to accumulation of oxidized LDL-cholesterol within the arterial wall [27]. Nicotine content in cigarette activates the sympathetic nervous system (SNS), increasing both heart rate and systolic blood pressure. Increased myocardial oxygen demands as per increase the rate-pressure. Increased SNS activity also leads to coronary arterial vasoconstriction [28].

### **Alcohol Consumption :**

Alcohol consumption is associated with an acutely higher risk of myocardial infraction in the subsequent hour among people who do not typically drink alcohol daily. There is consistent evidence that moderate habitual alcohol consumption is associated with a lower risk of cardiovascular events in subsequent months and years and that heavy episodic (binge) drinking is associated with higher cardiovascular risk [29,30]. Some researchers found that alcohol consumption could increase the prostacyclin of blood vessel wall, improve functions of vascular endothelial cells, increase insulin sensitivity and resist thrombosis [31,32]. Moreover, long-term regular alcohol consumption could improve heart rate variability and thus reduce MI onset risks [33,34].

### **Dyslipidemia :**

Dyslipidemia a major risk factor of cardiovascular disease is generally defined as the total cholesterol, LDL, triglycerides, apo B or Lp levels above the 90<sup>th</sup> percentile or HDL and apo A levels below the 10<sup>th</sup> percentile of the general population [35,36]. For myocardial infraction predisposing risk factors such as increased triglyceride levels and small LDL particles. For coronary atherosclerosis major risk factors are high levels of total cholesterol, LDL and low level of HDL [37]. Higher risk of myocardial infraction can reduce by correction of dyslipidemia [38].

### **Diabetes Mellitus :**

In India, type 2 diabetes mellitus is on the verge of becoming pandemic [39]. It is a chronic condition that occurs when the body can not produce enough or effectively use of insulin and are induced by a genetic predisposition coupled with environmental factors. Several risk factors of type 2 diabetes in common with coronary artery disease (CAD), such as age, hypertension, dyslipidemia, obesity, physical inactivity and stress, an increase in the prevalence of diabetes indirectly implicates an increasing rapidly risk of CAD as well [40]. For cardiovascular disease,

diabetes mellitus is a well-established risk factor. Increase the risk of coronary heart disease by two to four times because of diabetes [41]. The life of diabetic patient is reduced by nearly eight years due to increased mortality [42]. More than 80% of all deaths in Coronary artery disease and 75% of all hospitalizations in diabetic subjects. Diabetes increase the risk of myocardial infraction because it increase the rate of atherosclerotic progression and adversely affects the lipid profile and facilitates formation of atherosclerotic plaque. In myocardial infraction diabetes is risk factor which is more often fatal in people with diabetes compared with myocardial infraction in those without diabetes [43].

**Hypertension :**

The risk of a myocardial infraction because of both systolic and diastolic hypertension and the higher the pressure, the greater the risk. Atherosclerosis in coronary blood vessels due to the higher systolic and diastolic pressure, result in heart attack or myocardial infraction. Hypertension and myocardial infraction are closely linked. In old age, hypertension is responsible for at least 70% of heart disease and even worse to heart [44]. Strict compliance of proper medication and adoption of lifestyle modifications may control hypertension and reduce the risk of myocardial infraction significantly [45].

**Obesity/Body Mass Index :**

Incidence of myocardial infraction is directly related to the increased BMI. Recognized risk factor for myocardial infraction is obesity by which infraction is greatly enhanced. Overweight and obesity may affect health, and to prevent MI it is necessary to control one's BMI [46].

**Stress :**

The risk of heart attack and stroke is increase by chronic life stress, social isolation and anxiety [47]. Acute psychological stress also is associated with increased risk for coronary heart disease, and it has been reported that intense grief in the days after death of a significant person may trigger the onset of myocardial infraction [48].

**Family History :**

An independent risk factor for MI is family history of myocardial infraction. In a first-degree relative doubles MI risk the genetic variants are associated with increased risk of MI and family history of MI. If a father develops heart attack before the age of 55 and mother before the age of 65 years, this positive family history becomes very significant for the next generation and more presence of parental and maternal history for premature myocardial infraction may increase the risk to 7 folds in descendents [49].

**Age :**

The rate of mortality is increased in acute myocardial infraction because of advanced age [50,51]. The mechanism by which increasing age contributes so dramatically to mortality is unknown [52]. About 80 % death due to the heart disease occur in people aged 65 or older [24].

### **Gender :**

Men tend to have heart attacks earlier in life than women. Women's rate of heart attack increases after menopause but does not equal men's rate. Even so, heart disease is the leading cause of death for both men and women [24].

### **PATHOPHYSIOLOGY**

More than 20 to 40 minutes for the acute occlusion of one or multiple large epicardial coronary arteries can lead to acute myocardial infraction. 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. From sub-endocardium to sub-epicardium spread necrosis. The subepicardium is believed to have increased collateral circulation, which delays its death. The cardiac function is compromised depending on the territory affected by the infraction. Due to the negligible regeneration capacity of the myocardium, the infractioned area heals by scar formation, and often, the hearts is remodeled characterized by dilation, segmental hypertrophy of remaining viable tissue, and cardiac dysfunction [53].

### **Diagnosis[54]**

A diagnosis of myocardial infraction is created by integrating the history of the presenting illness and physical examination with electrocardiogram findings and cardiac markers. A coronary angiogram allows visualization of narrowing or obstructions on the heart vessels, and therapeutic measures can follow immediately. At autopsy, a pathologist can diagnose a myocardial infraction based on anatomopathological findings. A chest radiograph and routine blood tests may indicate complications or precipitating causes and are often performed upon arrival to an emergency department. New regional wall motion abnormalities on an echocardiogram are also suggestive of a myocardial infraction.

### **Physical Examination**

The general appearance of patients may vary according to the experienced symptoms; the patient may be comfortable, or restless and in severe distress with an increased respiratory rate. A cool and pale skin is common and to vasoconstriction. Some patients have low-grade fever(38-39<sup>0</sup>C).

Blood pressure may be elevated or decreased, and the pulse can become irregular. Various abnormalities can be found on auscultation, such as a third and fourth heart sound, systolic murmurs, paradoxical splitting of the second heart sound, a pericardial friction rub and rales over the lung.

### **Electrocardiogram**

The primary purpose of the electrocardiogram is to detect ischemia or acute coronary injury in broad, symptomatic emergency department populations. A serial ECG may be used to follow rapid changes in time. The standard 12 lead ECG does not directly examine the right ventricle, and is relatively poor at examining the posterior basal and lateral walls of the left ventricle. The use of additional ECG leads like right-sided leads V3R and V4R and posterior leads V7, V8 and V9 may improve sensitivity for right ventricular and posterior myocardial infraction.

The 12 leads ECG is used to classify patients into one of three groups.

1. those with ST segment elevation or new bundle branch block (suspicious for acute injury and a possible candidate for acute reperfusion therapy with thrombolytics or primary PCI)
2. those with ST segment depression or T wave inversion (suspicious for ischemia)
3. those with a so called non-diagnostic or normal ECG.

A normal ECG does not rule out acute myocardial infraction. Mistakes in interpretation are relatively common, and the failure to identify high risk features has a negative effect on the quality of patient care. It should be determined if a person is at high risk for myocardial infraction before conducting imaging tests to make a diagnosis. Imaging tests such as stress radionuclide myocardial perfusion imaging or stress echocardiography can confirm a diagnosis when a person's history, physical exam, ECG and cardiac biomarkers suggest the likelihood of a problem.

### **Cardiac Markers**

Cardiac markers or cardiac enzymes are proteins that leak out of injured myocardial cells through their damaged cell membranes into bloodstream. Until the 1980s, the enzymes SGOT and LDH were used to assess cardiac injury. Now, the markers most widely used in detection of MI are MB subtype of the enzyme creatine kinase and cardiac troponins T and I as they are more specific for myocardial injury. The cardiac troponins T and I which are released within 4-6 hours of an attack of MI and remain elevated for up to 2 weeks, have nearly complete tissue specificity and are now the preferred markers for assessing myocardial damage. Heart type fatty acid binding protein is another marker, used in some home test kits. New markers such as glycogen phosphorylase isoenzyme BB are under investigation. When damage to the heart occurs, levels of cardiac makers rise over time, which is why blood tests for them are taken over a 24 hour period. Because these

enzyme levels are not elevated immediately following a heart attack, patients presenting with chest pain are generally treated with the assumption that a myocardial infraction has occurred and then evaluated for a more precise diagnosis.

### **Angiography**

In difficult cases or in situations where intervention to restore blood flow is appropriate, coronary angiography can be performed. A catheter is inserted into an artery and pushed to the vessels supplying the heart. A radiopaque dye is administered through the catheter and a sequence of x-rays (fluoroscopy) is performed. Obstructed or narrowed arteries can be identified and angioplasty applied as a therapeutic measures. Angioplasty requires extensive skill, especially in emergency settings. It is performed by a physician trained in interventional cardiology.

### **Histopathology**

Histopathological examination of the heart may reveal infraction at autopsy. Under the microscope, myocardial infraction presents as a circumscribed area of ischemic, coagulative necrosis. Within the first 12 hours the infraction is not identifiable on gross examination. Using electron microscopy earlier changes can be discerned, one of the earliest changes under a normal microscope are so called wavy fibers. Subsequently, the myocyte cytoplasm becomes more eosinophilic and the cells lose their transversal striations, with typical changes and eventually loss of the cell nucleus. The interstitium at the margin of the infractioned area is initially infiltrated with neutrophils, then with lymphocytes and macrophages, who phagocytose the myocyte debris. The necrotic area is surrounded and progressively invaded by granulation tissue, which will replace the infraction with a fibrous scar. The interstitial space may be infiltrated with red blood cells.

## **MANAGEMENT AND MEDICAL THERAPY OF MI[6]**

### **❖ Initial Therapy upon diagnosis**

Upon diagnosis, management of MI and unstable angina should simultaneously focus on hemodynamic stabilization, relieving the pain, decreasing myocardial oxygen consumption, increasing myocardial oxygen supply and initiating antithrombotic therapy.

#### **a) Oxygen**

Arterial oxygen saturation below 94% to achieve normoxia then oxygen supplementation should be initiated in patients. Hyperoxia should be avoided since it causes coronary artery vasoconstriction, increases early myocardial injury and infraction size.

#### **b) Nitrates**

In the absence of hypotension (systolic blood pressure <90 mmHg) and signs of right ventricular infraction, patients should be given nitrates, namely nitroglycerin 0.4 mg sublingually every 5

minutes up to three times with the intention of increasing coronary blood flow by decreasing preload. This can also alleviate angina. Before nitrates are given, one has to make sure the patient did not take phosphodiesterase-5 inhibitors (eg. Sildenafil) in last the 24-48 hours, since combined with nitrates they can cause severe hypotension.

c) **Morphine**

Intravenous morphine of 3 to 5 mg may be given if pain persists and repeated every few minutes until the patient is pain-free. Myocardial oxygen consumption reduce by morphine diminishes sympathetic stimulation caused by pain and anxiety.

❖ **Antithrombotic Therapy**

**Antiplatelet Therapy**

Patients should chew non-enteric coated aspirin (150 to 300 mg) which blocks further platelet aggregation as a central pathophysiologic mechanism of MI after plaque disruption. If the patient is unable to take medicine orally also iv preparation of 150 mg can be given. Additional inhibition of platelet aggregation is achieved by P2Y<sub>12</sub> receptor antagonists, normally stimulated by ADP. Clopidogrel and prasugrel are irreversible, whereas ticagrelor is a reversible P2Y<sub>12</sub> inhibitor. Clopidogrel is known to have hypo- and hyper-responders while Prasugrel and ticagrelor have a more rapid and consistent action . Dual antiplatelet therapy (DAPT) with clopidogrel therapy has been shown to reduce ischemic events compared to aspirin alone. Prasugrel has also been shown to have lowest incidences of in-stent thrombosis and has had a great benefit in T2DM patients. Because of a higher bleeding risk, patients over 75 years of age, below 60 kg or history of TIA/stroke should not receive prasugrel, whereas on the other hand, ticagrelor is contraindicated in patients with history of intracranial hemorrhages and caution is advised in patients with bradycardia.

- In STEMI patients with planned PCI, pretreatment with a loading dose of 60 mg of prasugrel or 180 mg of ticagrelor or 600 mg of clopidogrel should be given, bearing in mind the contraindications. The choice of P2Y<sub>12</sub> antagonists should always be guided by local protocols or by consultation with a PCI center. When thrombolysis is planned , newer P2Y<sub>12</sub> antagonists should be avoided as there no data in this setting, and clopidogrel should be used instead.
- If conservative management is preferred, prompt initiation of DAPT is warranted, preferably with ticagrelor in the absence of contraindication in STEMI or NSTEMI-ACS patient.

**Anticoagulation Therapy**

After initiation of DAPT, anticoagulants should also be given. Anticoagulation in adjunct to DAPT was shown to reduce ischemic events. Unfractionated heparin (initial bolus 70-100 U/kg when no glycoprotein(GP) IIb/IIIa inhibitor is planned or 50-60 U/kg when the use of GP IIb/IIIa inhibitors is expected is mainly used in patients requiring immediate PCI. Fondaparinux is considered the safest and as effective as others, and is therefore preferred if there is no immediate PCI planned, whereas on the other hand, in the context of primary PCI, it was associated with potential harm and is therefore not recommended. Glycoprotein IIb/IIIa inhibition is usually used in peri-procedural settings as there is no difference in ischemic events if given pre-procedurally and no increased risk of bleeding.

#### ❖ Reperfusion Strategy

The timing of invasive strategy depends on the risk of complications. In STEMI and very high risk NSTEMI-ACS patients, reperfusion should be initiated as soon as possible in less than 120 min if the time from symptom onset is less than 12 hours. Reperfusion can be achieved by percutaneous coronary intervention (PCI), with a prompt initiation of fibrinolysis, or with a combination of both. When PCI can be performed within a time limit of 60-90 min, then direct transport to the PCI center is indicated. High risk NSTEMI-ACS patients require early PCI in the first 24 hours, since immediate PCI did not result in any major benefit, while longer times, on the other hand, were connected with higher mortality rates.

#### ❖ Beta Blockers

Decreasing myocardial oxygen consumption because of Beta-blockers lower blood pressure, heart rate and myocardial contractility. They should be administered universally in patients without contraindications early after MI because of an observed relative risk reduction in mortality in the first week and in-hospital. Beta-blockers are contraindicated in heart failure, high grade aortic stenosis or other low-output state, bradycardia or heart block.

#### ❖ Angiotensin Converting Enzyme Inhibitors (ACEI)

Early oral introduction of ACEI in the first 24 hours of MI reduces mortality regardless of the reperfusion strategy, especially in patients with anterior infarction, pulmonary congestion or an LV ejection fraction of less than 40%. They should not be given if systolic blood pressure is below 100 mmHg and in presence of contraindications.

#### ❖ Statins

In all ACS patients after the initial stabilization, statin are recommended therapy. Intensive statin therapy is recommended. According to ESC guidelines, the use of statins that lower LDL for more than 50% is recommended and according to ACCF/AHA guidelines, atorvastatin 80 mg should be

used. High intensity statin therapy significantly reduces cardiovascular death and MACE in the first month after ACS. Statin therapy should be titrated to achieve target LDL levels in secondary prevention after two month.

#### ❖ Proton-Pump Inhibitors

Proton pump inhibitor (PPI) should be initiated in all patients receiving dual antiplatelet therapy because of ACS, history of bleeding or regardless of their bleeding tendency . The use of PPIs in combination with clopidogrel may reduce its antiplatelet effect through competitive inhibition of CYP2C19; nevertheless, compliance of DAPT use with concomitant use of PPI is higher.

#### ❖ Myocardial Protection By Conditioning

Many researchers are investing ways of protecting the myocardium after ACS with the purpose of minimizing ischemia-reperfusion injury to reduce infarct size. Depending on the time frame and the site of intervention, three terms are used: ischemic preconditioning (IPC) , Post conditioning (POC) and remote ischemic preconditioning (RIPC). Nowadays, the term conditioning refers to the intervention of increasing the heart's general ability to withstand ischemia.

#### ❖ Ischemic Preconditioning

IPC is defined as a phenomenon where brief periods of ischemia accompanied by reperfusion just before sustained ischemia results in a delayed progression or reduction of infarct size, despite an increase in the total ischemic period. Its positive effects are exerted through a delay reduction in oxygen consumption, delay in ATP consumption, retention of intracellular structures and a delay in cellular necrosis. Typically, it is experimentally achieved by applying several short cycles of ischemia followed by reperfusion and finally sustained ischemia. IPC protection happens in two phases, early (<3h) and late (24-72h). Early phase protection is mainly attributed to the activation of ion channels, phosphorylation of existing enzymes or a rapid turnover or translocation of substances, whereas the late phase is attributable to the changed genetic expression of receptors, membrane channels, enzymes, or immunomodulators. Versatile mechanism/agents have been reported to be more or less successfully influenced/used to mimic protection by IPC. Agonists like adenosine, bradykinin, opioids, acetylcholine, catecholamine and oxygen radicals activate the protein kinase C (PKC) pathway, which seems to be the first convergent step in the cascade leading to the opening of mitochondrial ATP- sensitive potassium ( $K_{ATP}$ ) channel. In humans, IPC has been studied through exercise-induced ischemia, unstable angina, coronary angioplasty and cardiac surgery. Studies investigating unstable angina found smaller infarct sizes, which cannot only be attributed IPC, but can also be a consequence of collateral flow and reperfusion rate. Repeated exercise stress tests in less than 60 minutes showed improved performance by more than

half with oxygen consumption reduction in the second test, suggesting improved metabolic efficiency- IPC. A good experimental human model for IPC is elective coronary angioplasty, where repeated balloon inflations are done, resulting in ECG ST changes in subsequent occlusions and less subsequent angina pain. The role of IPC in the adaptation to ischemia has been proven by usage of specific antagonist of IPC pathways.

## CONCLUSION

Understanding the risk factors which is responsible of development of myocardial infraction and also pathophysiology is associated with myocardial injury. There are many therapeutic intervention such as pharmacologic, non-pharmacologic and combination therapy to improves clinical outcomes in STEMI and NSTEMI patients. MI is diagnosed by physical examination with electrocardiogram findings and cardiac markers. Infractioned person is cure with some physical activity along with medication.

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