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# A Review Article on Myocardial ischemia

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## ABSTRACT

Myocardial infarction (MI) is the term used for a heart attack caused by the accumulation of plaque on the inner walls of the arteries, resulting in reduced blood flow to the heart and damage to the heart muscle due to lack of oxygen. Symptoms of MI include chest pain that moves from the left arm to the neck, shortness of breath, sweating, nausea, vomiting, abnormal heartbeats, anxiety, fatigue, weakness, stress, depression, and other factors. Immediate treatment for MI includes taking aspirin to prevent blood clots and nitro-glycerine for chest pain and oxygen. A heart attack can be prevented by taking early steps to reduce risk by controlling your diet, fat, cholesterol, salt, smoking, nicotine, alcohol, drugs, blood pressure, daily exercise, and weight loss.

Treatment for MI includes aspirin tablets and thrombolytic or clot-dissolving drugs, such as tissue plasminogen activator, streptokinase, or urokinase, within 3 hours of the onset of the heart attack. Pain relievers such as morphine or meperidine may be given to relieve pain. Nitro-glycerine and blood pressure medications such as beta blockers, ACE inhibitors or calcium channel blockers can also be used to lower blood pressure and improve the heart's oxygen demand. Narrowing of the coronary arteries can be monitored by EKG, coronary angiography, and x-rays of the heart and blood vessels..

**KEY WORDS:** MI, CAD, ACE, EKG

## INTRODUCTION

Myocardial ischemia is characterized by an imbalance between myocardial oxygen supply and demand, causing cardiac dysfunction, arrhythmias, myocardial infarction, and sudden death. Various clinical ischemic manifestations are caused by obstruction of coronary blood flow by coronary stenosis, thrombosis, and/or hyper constriction (vasospasm) of epicardial and microvascular coronary arteries. The coronary circulation matches blood flow with myocardial oxygen demand by coordinating the vascular resistances within microvasculature, where the endothelium plays an important role.

The endothelium also regulates the tone of the underlying vascular smooth muscle cells (VSMC) by releasing several endothelium-derived relaxing factors, such as nitric oxide (NO), prostacyclin, and endothelium-derived hyperpolarizing factor (EDHF) . The cells also release several vasoconstricting factors, such as endothelin, superoxide anions (O2 −), and thromboxane, under certain pathological conditions. Endothelial dysfunction is regarded as a clinical syndrome that exhibits systemic manifestation of atherosclerosis and resultant myocardial ischemia, and is associated with significant morbidity and mortality [1-3]

## EPIDEMOLOGY

IHD affects around 126 million individuals (1,655 per 100,000), which is approximately 1.72% of the world's population. Nine million deaths were caused by IHD globally. Men were more commonly affected than women, and incidence typically started in the fourth decade and increased with age. The global prevalence of IHD is rising. We estimated that the current prevalence rate of 1,655 per 100,000 population is expected to exceed 1,845 by the year 2030. Eastern European countries are sustaining the highest prevalence. Age

standardized rates, which remove the effect of population changes over time, have decreased in many regions.[4-5]

**Symptoms**

the most common is chest pressure or pain, typically on the left side of the body (angina pectoris). Other signs and symptoms-which might be experienced more commonly by women, older people, and people with diabetes - include:

- Neck or jaw pain
- Shoulder or arm pain
- A fast heartbeat
- Shortness of breath when you are physically active
- Nausea and vomiting
- Sweating
- Fatigue[6]

**Causes**

Conditions that can cause myocardial ischemia include:

- Coronary artery disease (atherosclerosis).
- Blood clot & Coronary artery spasm. [7]

**Risk factors**

Factors that can increase your risk of developing myocardial ischemia include:

- Tobacco
- Diabetes.
- High blood pressure.
- High blood cholesterol level.
- High blood triglyceride level.
- Obesity.
- Waist circumference.
- Lack of physical activity**.** [8]

**ASSESSMENT:**

Myocardial ischemia is defined as an imbalance between myocardial oxygen demand and supply [9]. Myocardial ischemia is clinically indicated by transient ST-segment electrocardiogram (ECG) changes on exercise or pharmacological stress test and reversible perfusion defects on stress myocardial scintigraphy.

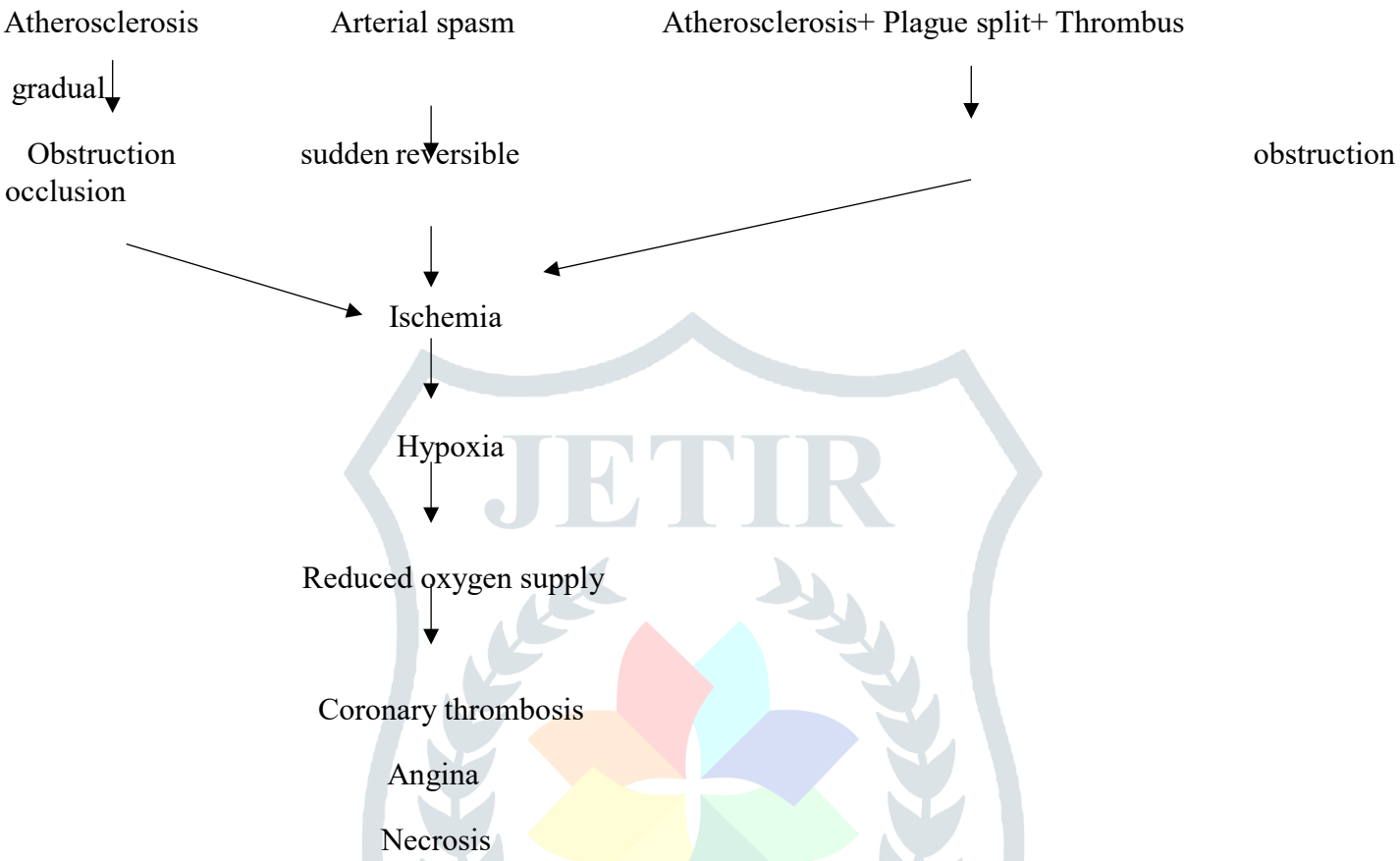
Metabolic changes, including myocardial lactate production, coronary sinus oxygen desaturation, and pH reduction in the coronary sinus, are also important objective proof of myocardial ischemia. Myocardial release of lipid peroxide products in the coronary circulation is a marker of myocardial ischemia with a high sensitivity even for brief and/or mild myocardial ischemia .

Myocardial phosphorus-31 nuclear magnetic resonance (31P NMR) spectroscopy is another sensitive method to identify myocardial ischemia by measuring myocardial high energy phosphates phosphocreatine and adenosine triphosphate . Measurement of coronary blood flow is useful, but only provides information associated with myocardial ischemia. Positron-emission tomography (PET) allows the quantitative calculation of coronary blood flow . [10]

Magnetic resonance imaging (MRI) with intravenous infusion of contrast media can also be used for the quantification of myocardial blood flow . Coronary flow reserve is expressed by the ratio of blood flow

during maximal hyperaemia (e.g. adenosine or papaverine) to that at rest. Coronary flow reserve can be measured invasively by the thermodilution or Doppler technique and is considered abnormal when it is less than 2.0. Trans-thoracic colour Doppler echocardiography enables non-invasive assessment of coronary flow/velocity reserve, especially in the territory of the left anterior descending coronary artery . Since flow resistance is mainly determined at the microvascular level, especially in patients with angiographically normal arteries, the reduction in coronary flow reserve reflects coronary microvascular dysfunction.[11].

**PATHOPHYSIOLOGY [12]**




**MANAGEMENT Pharmacological treatment** [13]

| **Class of drug** | **Action** | **Drug** |
|---|---|---|
| Beta blockers | Heart's workload↓ | Nadolol, Metoprolol, Pindolol, Bisoprolol etc. |
| Diuretics | Rid body of excess fluid and salt | Hydrochlorothiazide, Chlorothiazide, Furosemide, Triamterene, Spironolactone etc |
| ACE inhibitors | Prevent blood vessel constriction | Benazepril, Lisinopril, Captopril, Ramipril, Eosinophil, Maxiprep. |
| Calcium channel blockers. | prevent blood vessel constriction by blocking calcium ions | Verapamil, Diltiazem, Nifedipine |

Beta-blockers, angiotensin-converting– enzyme (ACE) inhibitors, and aldosterone antagonists have been shown to reduce the overall risk of death as well as the risk of major nonfatal cardiovascular events when they are administered to patients with acute myocardial infarction who also have left ventricular systolic dysfunction, clinical evidence of heart failure, or both[14].

Relevant to this discussion is the observation that ACE inhibitors block only 13 percent of the total production of angiotensin II in the human heart because of the existence of ACE independent pathways (e.g., chemise, cathepsin, and kallikrein) that convert angiotensin I to angiotensin II.[15] These observations provided the impetus for the development of angiotensin receptor antagonists that offer more complete protection against angiotensin II by directly blocking the angiotensin type I receptor.[15-16]

## CONCLUSION

Myocardial ischemia occurs when the myocardial demand for substrates exceeds that of supply. Although we often consider myocardial ischemia in the setting of critical CAD, it is clear that ischemia may occur with or without epicardial CAD.

**Conflict of Interest**: Nill

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