INTRODUCTION
In the United States approximately, 700000 coronary artery related deaths and more than 1.1 million Acute Myocardial Infarctions occur each year. The mortality rates with Acute Myocardial Infarction is approximately 30%, with more than half of these deaths occurring before the patient reaches the hospital. The decreasing individual mortality from myocardial infarction is possibly accounted for by earlier detection, use of intensive coronary care unit, advances in drug therapy and reperfusion with thrombolytic agents or balloon angioplasty.

Acute Myocardial Infarction generally occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of a coronary artery previously narrowed by atherosclerosis and which leads to reversible myocardial cell damage and necrosis. Coronary thrombus is formed at the site of vascular injury. This injury is produced by various factors e.g. cigarette smoking, hypertension and hyperlipidemia. In most cases infarction occurs when atherosclerotic plaque fissures, ruptures or ulcerates and when conditions favour thrombogenesis, so that a mural thrombus forms at the site of rupture and leads to coronary artery occlusion. Histologic studies show that the coronary plaques prone to rupture are those with rich lipid core and thin fibrous cap. Such plaques usually have an eccentrically located lipid pool. Plaque rupture usually occurs at the junction between the fibrous cap and the normal vessel wall, probably from stress at this area.
Whether increased shear forces caused by the stenosis, repeated oscillatory stress resulting from contraction of the heart or changes in coronary tone related to circulating catecholamine act single or in concert to potentiate plaque rupture remains conjectural.

After the rupture of plaque, initial platelet monolayer forms and then various agonists e.g. collagens ADP, epinephrine, serotonin etc. promotes platelet activation. After platelet activation there is production and release of thromboxane A2, further platelet activation and potential resistance to thrombolysis.

Activation of platelets by agonists produce a conformational change in the glycoprotein IIb/IIIa receptor. When glycoprotein IIb/IIIa converted to its functional state, this receptor develops high affinity to vWF and fibrinogen. Since vWF and fibrinogen are multivalent molecules they can bind to two different platelets, simultaneously resulting in platelets cross linking or aggregation.

The coagulation cascade is activated on exposure of tissue factor in damaged endothelial cells at the site of ruptured plaque. Factors VII & X an activated, & leading to the conversion of prothrombin to thrombin which then converts fibrinogen to fibrin. Thrombin further leads to auto amplification reaction that results further activation of the coagulation cascade. The athero-sclerosed artery eventually become occluded by a thrombus containing platelet aggregates & fibrin strands.

Infrequently Myocardial Infarction may occur with prolonged or severe coronary spasm in the absence of under lying coronary
artery disease e.g. cocaine use, ergot therapy and severe emotional stress.

Following are rare cause of Myocardial Infarction –

1. Spontaneous coronary artery dissection
2. Serum Sickness
3. Various allergic conditions
4. Profound hypoxemia
5. Sickle cell crisis.
6. Carbon mono-oxide poisoning
7. Acquired hyper-coagulable state

There is an increase in Myocardial Infarction event rates in the early morning hours, probably related to circadian variation in coronary vascular tone, catecholamines and co-agulability.

Totally occlusive thrombus in patients with inadequate distal collateralization most often result in Q-Wave Myocardial Infarction. Transiently occluding thrombus, with spontaneous lysis or distal collateralization may yield lesser degree of necrosis and produce “non-Q” wave Myocardial Infarction.

Patients with intermittent or subtotal occlusive thrombi, adequate collateralization or both may have the syndrome of unstable or prolonged angina in the absence of myonecrosis or if serum cardiac markers are detected as having “non Q” Wave Myocardial Infarction, a minority of patients who present initially without ST segment elevation may develop a "Q" wave myocardial infarction. The presentation that constitute the spectrum ranging from unstable angina through 'non-Q' wave Myocardial Infarction to
'Q' wave Myocardial Infarction are called **Acute coronary syndrome.**

**Acute coronary syndrome**

**SERUM CARDIAC MARKERS** –

1. Creatine – Phospho Kinase MB – appears after 4-6 hrs. and peak level reaches at approximately 20 hrs. after the coronary occlusion and falls to normal within 48-72 hrs.

2. Troponin I & T – These are normally not detected in the blood of healthy individuals but may increase after myocardial infarction to levels over 20 times higher than the cut off value. Trop I may remain elevated for 7 to 10 days after myocardial infarction and Trop T level may remain elevated for upto 10 to 14 days.
3. LDH – It comes in blood more than 24 to 48 hrs after the onset of symptoms and reaches a peak after 2 or 3 days and may elevated for a week or more.

4. AST – It starts to rise about 12 hrs after infarction and reaches a peak on the first or second day, returning to the normal within 3 or 4 days.

MORTALITY:

The mortality rate with Acute Myocardial Infarction is approximately 30% more than half of these death occurring before stricken individual reacher to the hospital. Approximately 1 of every 25 patients who survive the initial hospitalisation, dies in the first year after myocardial infarction. Survival is reduced in over age i.e. > 75yrs., whose mortality rate is 20% at one month and 30% at one year after Acute Myocardial Infarction.

COMPLICATIONS:

Complications of Acute Myocardial Infarction occur in a time dependent manner and can be directly related to the anatomy of the coronary artery blood supply. Early complication may begin within 20min of the onset of Myocardial Infarction. These complications include arrhythmias and heart block (due to injured or ischemic conduction system) and hypotension and congestive heart failure due to ischemic or injured muscle tissue resulting in abnormal filling (diastolic dysfunction) or abnormal emptying (Systolic dysfunction).

Several days later complications of Myocardial Infarction can occur due to “Yellow softening” of myocardial tissue resulting in one
of several "Mechanical complications" of Myocardial Infarction in addition inflammation surrounding the injured myocardial tissue can lead to postinfarction pericarditis.

1. **Ventricular Dysfunction**:

After myocardial infarction the left ventricle undergoes a series of changes in shape, size and thickness in both the infarcted and non-infarcted segment. This process is known as ventricular remodeling. Soon after Myocardial Infarction the left ventricle begins to dilate resulting in disproportionate thinning and elongation of the infarcted zone, later lengthening of the non-infarcted segment occur as well with greater dilatation following infarction of the apex of the left ventricle & causing more marked hemodynamic impairment and frequent heart failure and poorer prognosis. Degree of pump failure correlates hemodynamic assessment of left ventricular failure well with the extent of ischemic necrosis and with mortality. The diagnostic signs are pulmonary rales, S3 and S4 gallop rhythm and pulmonary congestion on X-ray chest. Hemodynamic evidence of abnormal left ventricular function appears when contraction is seriously impaired in 20% to 25% of the left ventricular wall.

2. **Cardiogenic Shock**

Typically patients who develop cardiogenic shock have severe multivessel coronary artery disease. Now-a-days the incidence of cardiogenic shock is about 7%. Only 10% of patients with this condition present with it on admission. While 90% develop during hospitalization. This is characterized by marked hypotension i.e. (<90 mm Hg systolic and decrease in cardiac index (approximately <1.8
L/min/mm²). Cardiogenic shock is generally associated with a mortality rate of 70%³,⁴. Infarction of 40% or more of the left ventricle usually results in cardiogenic shock.

Cardiogenic shock results when there is a marked reduction in forward cardiac output leading to hypotension, decreased organ perfusion and at the same time elevated left ventricular filling pressure leading to congestive heart failure. This can be due to either massive left ventricular complication e.g. Mitral Regurgitation ventricular aneurysm formation, Right ventricular infarction.

3. Right Ventricular Infarction⁵,⁶ :

Approximately 1/3 of patients with inferioposterior area infarction demonstrate a minor degree of right ventricular necrosis. Clinically significant right ventricular infarct presents with increase JVP, Kussmaul's Sign, hepatomegaly with or without hypotension. ST segment elevation of the V1 and V2 and particularly V4R in the first 24 hours.

Right Ventricular infarction occurs almost exclusively in the setting of Right coronary artery occlusion.

2D echo is helpful in determining the degree of right ventricular dysfunction.

4. Mechanical cases of heart failure : it includes

a) Acute mitral regurgitation.

b) Free wall rupture,

c) Left ventricular aneurysm formation.
d) Ventricular septal defect.

a) *Acute Mitral Regurgitation*

Acute mitral regurgitation may occur abruptly from rupture of a left ventricular papillary muscle resulting in a flail mitral leaflet, usually the posterior leaflet. This result in an abrupt decrease in forward cardiac output, leading to congestive heart failure and often to the cardiogenic shock.

This occurs more commonly in the setting of inferior wall Myocardial Infarction since the Right Coronary Artery and Left circumflex artery supply the posteriomedial head of the papillary muscle, which is more prone to rupture than anterolateral head. Ventricular septal defect and mitral regurgitation are often impossible to be differentiated from each other because both presents with pansystolic murmur and tall 'V' wave in pulmonary capillary wedge pressure. Colour Doppler echocardiography may help in differentiating these two conditions. Unlike rupture of ventricular septal defect which occurs with large infarct, papillary muscle rupture occurs with a relatively small infarction.

b) *Left ventricular free wall rupture*

Rupture of the left ventricular free wall is analogous to ventricular septal defect but occurs in the free wall of the left ventricle, usually resulting in abrupt cardiogenic shock due to cardiac tamponade. Rarely a pseudoaneurysm of the left ventricle occurs if there is incomplete rupture of the free wall and this may be undetected clinically until abrupt deterioration occurs.
However, as a group they are probably responsible for about 15% of all deaths from Acute Myocardial Infarction⁸,⁹.

c) *Left ventricular aneurysm formation:*

Left ventricular apical aneurysm formation usually occurs following antero-apical myocardial infarction after Left Anterior Descending occlusion. This weakening of the apical wall results in an out pouching or dyskinesis of the apex of the heart during systole.

The resultant stasis of blood in the dyskinetic segment of the apex may result in thrombus formation and systemic embolization. The reduced left ventricular ejection fraction may lead to congestive heart failure and predispose to ventricular arrhythmias.

It is of 2 type Viz true aneurysm and false aneurysm. Ventricular aneurysm are readily detected by 2 D echo which may also reveal a moral thrombus in an aneurysm. True Left Ventricular aneurysm occurs probably of Acute Myocardial Infarction and are more frequently in patient with transmural myocardial infarction.

d) *Ventricular Septal rupture:*

Acute ventricular septal rupture can occur usually several days following the acute infarction, due to softening of the necrotic tissues of septum. This can occur in both inferoposterior and in anterior Myocardial Infarction. A loud systolic ejection murmur
usually occurs and results in an acute left to right shunt with congestive heart failure and usually cardiogenic shock.

5. ARRHYTHMIAS:

The mechanism responsible for infarction related arrhythmias include autonomic nervous system imbalance, electrolyte disturbance, ischemia and slowed conduction, mortality form arrhythmias is greater during the first few hours.

6. PERICARDITIS:

Pericardial frictions rub and or pericardial pain are encountered in about 10% patient with acute transmural myocardial infarction. Anti co-agulants potentially could cause tamponade, in the presence of acute pericarditis.

Infrequently Dressler’s Syndrome\textsuperscript{10} or post myocardial infarction syndrom\textsuperscript{11} may be present which is characterized by fever and pleuropericardial pain. It may begin from few days to 6 weeks after myocardial infarction.

7. THROMBOEMBOLISM:

Clinically apparent thromboembolism complicate Acute Myocardial Infarction in approximately 10% of cases but embolic lesions are found in 20% of patients in necropsy series, suggesting that thromboembolism is often clinically silent. Thrombus can be detected by echocardiography. Echocardiography reveals left ventricular thrombi in about 1/3 of the patients with anterior
infarction but also in few patients with inferior or posterior infarction.

8. HYPOTENSION:

Hypotension may occur in various setting following Acute Myocardial Infarction. These include –

1. Hypovolemia.
2. Excessive vasodilatation from nitrate therapy.
3. Decreased left ventricular filling, secondary to right ventricular infarction.
4. Marked reduction in cardiac output to extensive infarction or to a mechanical complication.