PLASMA HS-CRP LEVEL AS A PREDICTOR OF LEFT VENTRICULAR FUNCTION & MYOCARDIAL REPERFUSION INJURY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION-THROMBOLYSED

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COIMBATORE

CERTIFICATE

This is to certify that the Dissertation entitled **" PLASMA HS-CRP LEVEL AS A PREDICTOR OF LEFT VENTRICULAR FUNCTION & MYOCARDIAL REPERFUSION INJURY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION-THROMBOLYSED",** submitted by **Dr.VADIVEL KUMARAN.S.,** Post-Graduate in General Medicine , Coimbatore Medical College ,to The Tamilnadu Dr. *M.G.R.* Medical University is a record of a bonafide research work carried out by him under my guidance and supervision from Jan 2006 to Jun 2007.

Professor Dr.M.Ramasamy

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Prof. Dr. K. Umakanthan

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DEAN

DECLARATION

I solemnly declare that the Dissertation titled **" *PLASMA HS-CRP LEVEL AS A PREDICTOR OF LEFT VENTRICULAR FUNCTION & MYOCARDIAL REPERFUSION INJURY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION-THROMBOLYSED*",** was done by me at Coimbatore Medical College & Hospital during the period from Jan 2006 to Jun 2007 under the guidance and supervision of Prof. Dr.M.Ramasamy and Prof.Dr.S.Veerakesari

This dissertation is submitted to The Tamilnadu Dr. *M.G.R.* Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch *I*) in General Medicine.

Place : Coimbatore **Dr.VADIVEL KUMARAN.S.**

Date :

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INTRODUCTION

INTRODUCTION

Myocardial Infarction is a serious complication of Atherosclerotic Coronary Heart Disease. Coronary Heart Disease was first described by William Herberden in 1768. In 1932 unipolar leads were discovered by Wilson and the establishment of coronary care units by Day and Brown has lead to the world wide proliferation of coronary care units. The current knowledge of pathophysiology of acute myocardial infarction started with the autopsy description of Dr. James Herrick from Chicago in 1912 who concluded that acute myocardial infarction results from thrombotic occlusion of coronary artery and prophesized that the hope of salvaging the muscle, lay in restoration of blood flow.

Despite impressive strides in diagnosis and management over the last three decades, Acute Myocardial Infarction (AMI) continues to be a major public health problem even in the industrialized world. In the United States nearly 1.5 million patients annually suffer from AMI (about one patient every 20 seconds).[1] Myocardial Infarction[2] is an acute cardiac disability arising from reduction or arrest of blood supply to the myocardium due to atherosclerotic or non-atherosclerotic lesions of coronary arteries. Virtually all acute infarcts are caused by thrombosis developing in a culprit vessel with ruptured atherosclerotic plaque. Usually coronary artery occlusion is associated with infarction of myocardium, though post-mortem examination of cases of sudden

deaths reveals evidence in only 20% of cases, the remaining 80% do not showing any change.

Accurate diagnosis is mandatory because mistaken diagnosis can be disastrous to the social, economical and family life of the patient. At one end of the spectrum is the danger of missing a potentially lethal illness, on the other hand mistaken diagnosis results in severe cardiac neurosis which is even more difficult to treat than the original disease itself.

The presenting symptoms may vary from severe pain in the chest to minimal symptoms, with the disease remaining unrecognized. In most patients there is a substernal heaviness[3] which radiates to the left shoulder or ulnar surface of forearm and hand. It can also radiate to back, interscapular region, root of neck, jaw and teeth. In others it presents as breathlessness, syncope, giddiness, fatigue, abdominal pain, nausea, vomiting and unexplained hypotension.

Coronary Heart Disease is more age dependant in women than in men. Women are usually 10 years older than men when any coronary manifestation first appears and Myocardial Infarction occurs as much as 20 years later [4]. Coronary Heart Disease is the leading cause of death in women and the lifetime risk of death is 31% in postmenopausal women. The median age of menopause is 51.4 years[5] and age distribution ranges from 40-58 years.

Data from Framingham study says that the incidence of cardiovascular disease is 3 times more in men than women (before menopause) and approximately equal in men and women aged 75-79 years. The incidence in women rises after menopause due to hormonal changes

Overall mortality and morbidity in coronary heart disease depends on age, sex, site and extent of infarction, presence of good collateral circulation and associated co-morbid conditions.

C-reactive protein is a hepatically derived marker of low grade systemic inflammation that largely reflects circulating cytokine formation. It is produced from liver in response to cytokine stimulation (interleukin 1 and 6)

It is reasonable to suggest that high CRP levels are associated with adverse outcome as a result of coronary instability.[6,7] CRP, at least, partially reflects the extent of myocardial necrosis and can be used to predict in-hospital and long-term outcome in patients with AMI.[8-14] Several large-scale prospective studies have shown the inflammatory marker - high sensitivity CRP (hs-CRP) to be a potent predictor of future MI, stroke and peripheral vascular occlusion among apparently healthy men and women, as well as among high-risk smokers[15] and the elderly.[16]

Systemic proinflammatory mediators, particularly hs-CRP and leukocytes were higher in AMI patients. High CRP levels in the acute phase strongly indicate

poor early clinical outcome of the patients with AMI.[17] In AMI, the peak plasma value of CRP can also be used to predict the risk of cardiac rupture[18] as well as short[19, 20] and long-term[21-23] prognosis. Estimation of CRP levels in acute phase may provide valuable information on left ventricular (LV) function and exercise capacity and can help in long-term risk stratification after AMI.

Despite successful early recanalization of an occluded infarct artery by percutaneous coronary intervention (PCI) or thrombolysis, up to one-third of AMI patients still fail to obtain complete myocardial reperfusion due to a process of myocardial reperfusion injury[24, 25]. This phenomenon is characterised by impairment of microcirculatory flow and by ongoing ischemia and tissue necrosis [26, 27]. Although the underlying mechanisms of reperfusion injury are still not fully elucidated, there is accumulating evidence that local inflammatory responses with infiltration of leucocytes in the capillary circulation and release of oxygen-free radicals play a key role in this reperfusion-related tissue injury[28, 29].

Reperfusion injury is characterised by impairment of microcirculatory flow and by ongoing ischemia and tissue necrosis. Evidence has accumulated that progressive capillary plugging by leukocytes causing capillary no-reflow and superoxide radical formation plays a role in this reperfusion injury. Recruitment of neutrophils is regulated through a complex sequence of molecular steps involving the selectins and integrins, which mediate leukocyte rolling and adhesion to the endothelium [6, 12]. There is experimental evidence that

cytokines such as IL-1 and TNF-α are involved in this early stage of neutrophil infiltration by induction of endothelial adhesion molecules[30].

Myocardial reperfusion injury is defined as the presence of persistent ST-segment elevation despite successful coronary intervention ($\geq$ 50% of the initial value).

AIM OF THE STUDY

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1. To analyze high sensitive C-Reactive protein in Myocardial Infarction for

   (a) Prediction of LV function

   (b) Myocardial reperfusion injury

   (c) Risk Prognostication following acute myocardial infarction

2. To study the risk factors, common site of myocardial infarction in admissions to Coimbatore Medical College Hospital, and its complications.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

A. ANATOMY OF CORONARY CIRCULATION (Fig 1)

The epicardial coronary arteries take origin from the right and left coronary sinuses. In 85% of patient the right coronary artery which gives rise to posterior descending artery supplies the entire right ventricle and large a part of the posterior wall of the left ventricle. This is referred to as right dominant circulation.

In 8% of patients the left coronary artery supplies entire left ventricle, interventricular septum and a portion of right ventricle. This is referred to as left dominant circulation.

In 7% of patients referred to as co-dominant circulation, the right coronary artery supplies right ventricle and posterior wall of interventricular septum while left coronary supplies the left ventricle and anterior portion of interventricular septum.

Fig 1. Diagram of **Myocardium** (1) of a branch of the leftcoronary artery (2) of the tip of the anterior wall of the heart (LCA, right coronary artery = RCA).

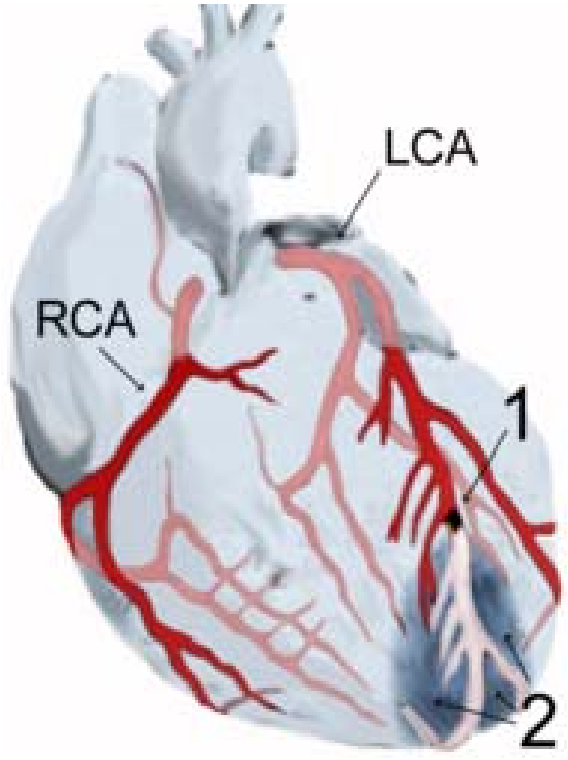
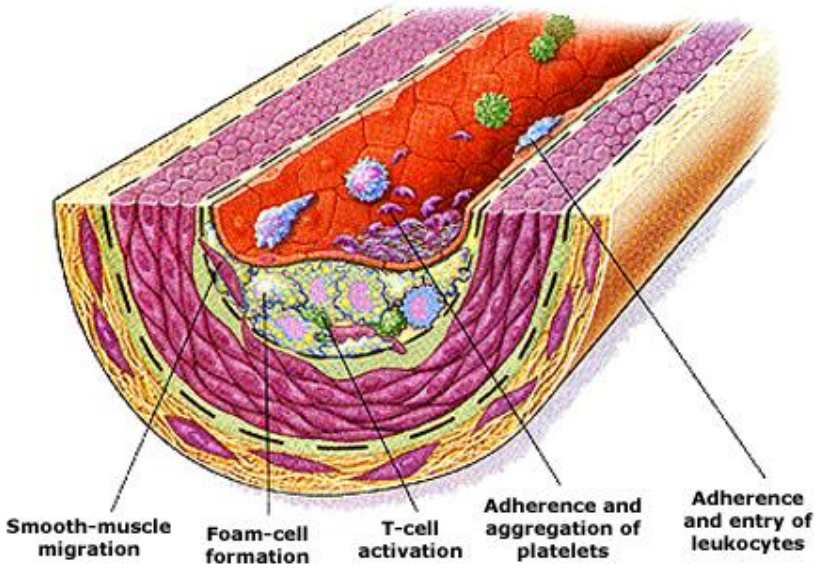


Fig 2. Fatty-streak formation in atherosclerosis



B.AETIOPATHOGENESIS OF MYOCARDIAL INFARCTION

1. ATHEROSCLEROSIS[31, 32]

Atherosclerosis is characterized by formation of atheroma or fibro fatty plaque which consists of a raised focal plaque within the intima, having a core of lipid and a covering of fibrous cap. (Fig 2, 3, 4, 5)

Atherosclerosis[33, 34] is the single most important etiological factor for coronary heart disease. The search for the cause and pathogenesis of atherosclerosis has become an insistent "golden grail". The acceptable hypotheses are

A) LIPID INSUDATION OR INFILTRATION HYPOTHESIS:

This is the modified "imbibition hypothesis"[35] termed by Virchow in 1856 which stated that cellular proliferation in intima was a form of low grade inflammation as a reaction to increased infiltration of plasma protein and lipids from the blood.

B) ENCRUSTATION OR THROMBOGENIC HYPOTHESIS:

This theory ascribed to Rokitansky, postulated that small thrombi composed of platelet, fibrin and leukocytes collected over foci of endothelial injury organized and their gradual growth resulted in plaque formation.

Fig 3. Illustration of the traditional concept of microcirculatory impairment in the setting of no flow in the proximal LAD compared with preserved flow in the left circumflex coronary artery, as typically seen in acute anterior ST-segment elevation myocardial infarction (upper left panel). The sequence starts with the vulnerable atherosclerotic plaque, which, by rupture or fissure, causes thrombus formation and thereby complete coronary artery occlusion. Embolization of particulate matter, potentially aggravated by PCI, in addition to ischaemia, oxidative stress, and inflammation can lead to dysfunction of the myocardial microcirculation with impairment of myocardial perfusion that can persist or be even intensified after restoration of epicardial blood flow.

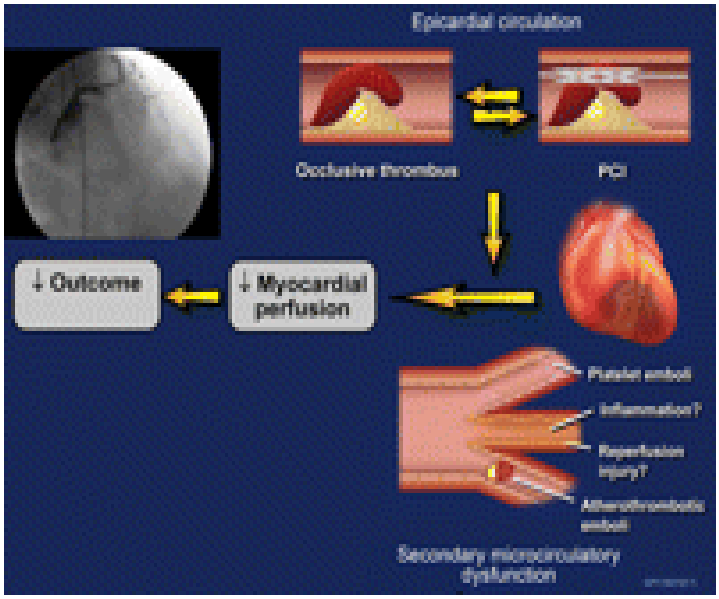
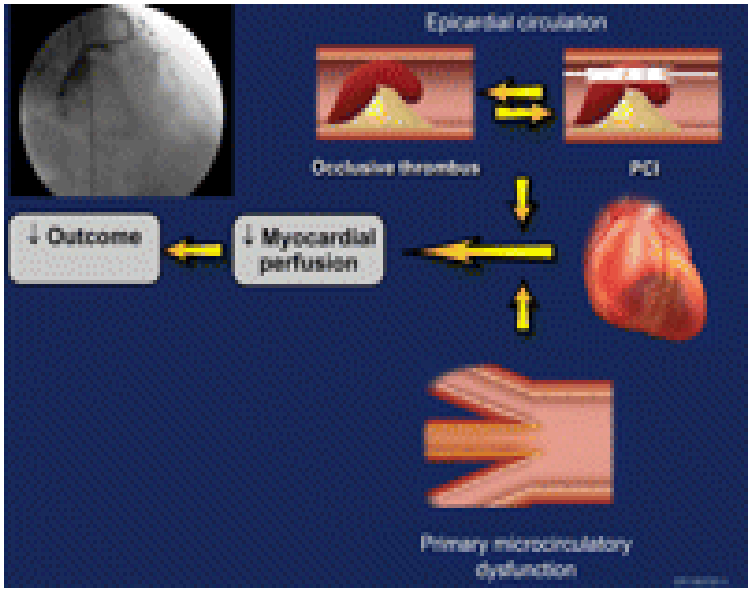


Fig 4. The non-traditional concept centres on the vulnerable patient, taking into account primary dysfunction of the microcirculation of a vulnerable myocardium in addition to the vulnerable plaque, which contributes to the clinical presentation and outcome.



C) REACTION TO INJURY HYPOTHESIS:

This widely accepted theory formulated by Ross and Glomset in 1976 and modified in 1986 states that the lesions of atherosclerosis are initiated as a response to some form of injury to arterial endothelium. Endothelial injury leads to attachment of monocytes and platelets, proliferation of smooth muscle cells in the arterial intima and deposition of intracellular and extracellular lipids.

2. NON-ATHEROSCLEROSIS [36]

Non-atherosclerotic causes of Myocardial Infarction are

1) Congenital anomalies like single coronary artery, atresia of coronary ostium, myocardial bridges, coronary AV fistula etc.
2) Dissection of coronary artery or aorta.
3) Embolic phenomena from prosthetic valves, infective endocarditis, tumors, calcium, paradoxical embolus, etc.
4) Traumatic injury or spasm of coronary artery.
5) Coronary arteritis due to Takayasu Disease, Polyarteritis Nodosa, SLE, Syphilis, Kawasaki Disease
6) Metabolic disorders like Mucopolysaccharidoses, Homocystinuria, Fabry's Disease and Amyloidosis.
7) Substance abuse like cocaine, amphetamine.

8) Myocardial oxygen demand-supply disproportion due to aortic stenosis, systemic hypotension, carbon monoxide poisoning, thyrotoxicosis.
9) Intimal proliferation due to irradiation, cardiac transplantation, fibro muscular hyperplasia.
10) Miscellaneous cause like HOCM, hypercoagulable states, diabetes mellitus.

3. RISK FACTORS FOR MYOCARDIAL INFARCTION [37, 38, 39]

(TABLE 1)

The term risk is widely used to describe those characteristically found in healthy individuals that relate to the subsequent appearance of Ischaemic Heart Disease[37]. The risk of Ischaemic Heart Disease is determined by the aggregation of individual factors.

Table 1: RISK FACTORS FOR CORONARY HEART DISEASE

A. FIXED
1. Age 2. Male sex 3. Family History
B. MODIFIABLE
1. Smoking 2. Hypertension 3. Lipid Disorders 4. Diabetes Mellitus 5.Haemostatic variables 6. Sedentary Life Style 7. Obesity 8. Mental Stress 9. Personality 10. Oral Contraceptive Pills 11. Hyperhomocysteinemia 12. Inflammation

1. AGE:

Age is a definite unmodifiable risk factor. Atherosclerosis develops progressively as age advances. Atherosclerosis is rarely present in early childhood, except in familial hyperlipidemia, but it is often detectable in postmortem specimens of young age between 15-30 years. Atherosclerosis is universal in elderly.

2. SEX:

Men are more affected than premenopausal women. However after menopause the incidence of atheroma rises in women. This suggests that oestrogen probably plays a part in preventing or delaying atherosclerosis. There is also a fall in HDL levels in postmenopausal women which may also contribute.

3. FAMILY HISTORY:

Coronary artery disease runs in families. This may be due to genetic factors or the effects of a shared environment (similar diet, smoking habits etc,). A positive family history is generally accepted to refer to those patients in whom a first degree relative of the patient has developed Ischaemic Heart Disease before the age of 50 years[40].

4. SMOKING:

Tobacco is probably the most important avoidable cause of coronary disease. The incidence of Ischaemic Heart Disease is 3-5 times higher in smokers who smoke 20 cigarettes per day compared to non-smokers. There is a strong, consistent and dose linked relationship between cigarette smoking and Ischaemic heart disease. The incidence of sudden death is also higher in smokers. Smoking decreases HDL cholesterol levels. It accelerates atherosclerosis, plaque instability, increases the risk of thrombosis, myocardial infarction and death.

Continuation of smoking increases incidence of restenosis after Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Bypass Grafting. The relative risk is highest in young people and becomes significantly lower within 6 months of quitting.

5. HYPERTENSION:

The incidence of coronary heart disease increases as blood pressure rises and the excess risk is related to both systolic and diastolic blood pressure. In the Framingham study, the incidence of Coronary Artery Disease in middle aged persons with blood pressure exceeding 160/95mmHg was 5 times more than that in normotensive men. For each 10mmHg increase in Diastolic Blood Pressure there is 37% increase in risk of Coronary Heart Disease[41].

Antihypertensive drugs have shown to reduce coronary mortality particularly by interruption of renin angiotensin system.

6. LIPID DISORDERS:

A diet rich in saturated fat and cholesterol is associated with increased incidence of atherosclerosis. There also appears to be a genetic variation in the ability of dietary cholesterol to influence plasma cholesterol and atherosclerosis.

A wealth of evidence from epidemiological, clinical and experimental studies has established the association between hyperlipidemia and atherosclerosis. Hypercholesterolemia is clearly a risk factor.

Of the lipoprotein, it is the low density lipoprotein (LDL) which is most atherogenic. VLDL is comparatively less atherogenic. HDL offers a protective effect and helps in removing cholesterol from the arterial wall. The ratio of LDL/HDL is a common way to assess atherogenicity of hyperlipidemia. A ratio of more than 4.5 is supposed to be atherogenic. A minor increase of 1mg/dl in HDL-4 Cholesterol produces a 2-4% decrease in the risk of developing Acute Myocardial Infarction.

The LDL cholesterol profiles are categorized as phenotypic pattern A, B and C. Patients with pattern B have mostly small, dense LDL particles. Small, dense LDL particles have been causally linked to an increased risk of coronary artery disease independent of total LDL cholesterol levels. The phenotype B

pattern is often, but not always associated with elevated triglycerides and triglyceride rich lipoproteins, reduced HDL and other features of insulin resistance syndrome. It has been suggested that small dense LDL particles are more atherogenic because of greater retention in the arterial wall and increased susceptibility to oxidation.

There is increasing evidence that hypertriglyceridemia is independently linked with coronary atheroma even after adjustment for HDL levels. In Indians living in India or abroad, raised triglycerides with low HDL has been found to be atherogenic.

7. DIABETES MELLITUS:

Diabetes Mellitus[42] is a coronary heart disease risk equivalent. The abnormal lipid profile with insulin resistance known as Diabetic Dyslipidemia (small dense LDL, low HDL, elevated triglyceride) account for part of elevated cardiovascular risk. Diabetes increases the frequency of atherosclerosis. It is likely that post prandial hyperglycemia may be more important in the development of coronary artery disease than fasting hyperglycemia. Diabetic women are 5-6 times more prone for coronary heart disease[43]compared to men.

8. HAEMOSTATIC FACTORS

High levels of fibrinogen and factor 7 are associated with increased risk of myocardial infarction. Polymorphism of factor 7 gene may increase the risk of Myocardial Infarction[26]. The stability of an arterial thrombus depends on the balance between fibrinolytic factors like plasmin and inhibitors of the fibrinolytic system such as Plasminogen Activator Inhibitor (PAI) 1. However, the levels of tissue plasminogen activator and PAI-1 in plasma have not proven to add information beyond the lipid profile for assessment of cardiovascular risk.

Apolipoprotein (a) has structural homology with plasminogen with which it is able to compete for cell surface binding. By displacing plasminogen, apolipoprotein (a) is able to inhibit cell surface mediated endogenous fibrinolysis by reducing the formation of Plasminogen Activator from Plasminogen. Plasma level of Lp (a) is largely genetically determined. Lp (a) levels do not potently predict risk in the population at large.

9. SEDENTARY LIFE STYLE:

The recent National Institute of Health Consensus Panel on Physical Activity and Cardiovascular Health established a goal of atleast 30 min of moderate intensity physical activity on a daily basis. Exercise increases HDL cholesterol, lowers blood pressure, reduces blood clotting and promote collateral vessel development.

10. OBESITY:

Obesity[44], particularly the male pattern of centripetal or visceral fat accumulation is probably an independent risk factor although it can be associated with hypertension, diabetes mellitus and physical inactivity.

11. MENTAL STRESS AND PERSONALITY:

Stress is associated with increased catecholamine levels and high blood pressure thereby increasing the risk of coronary heart disease.

Type A individuals who are ambitious, aggressive, impatient, competitive, always in a hurry and often frustrated are more prone to Ischaemic heart disease.

12. ORAL CONTRACEPTIVE PILLS:

Oral contraceptives disturb the clotting mechanism through an increased inactivity of factor 7 and 10. It also increases platelet sensitivity to ADP and hence to platelet adhesiveness. Oestrogen retards atherosclerosis but accelerates thrombogenesis resulting in myocardial infarction in the rare susceptible person.

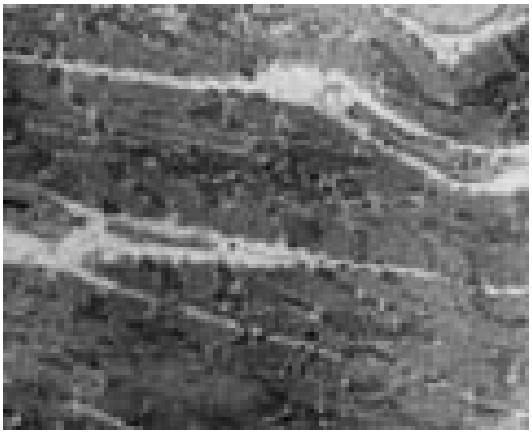
13. HYPERHOMOCYSTEINEMIA:

A large body of literature suggests a relationship between hyperhomocysteinemia and coronary events. Several mutations in the enzymes

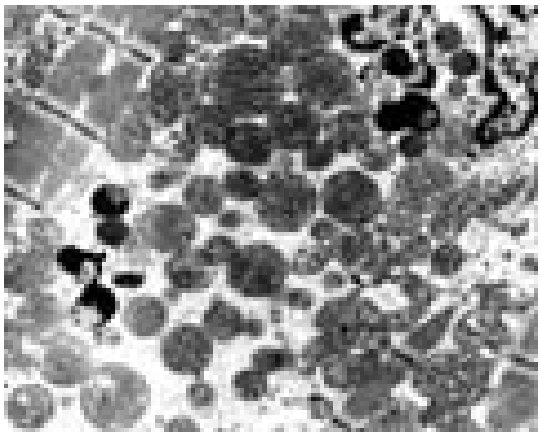
involved in homocysteine accumulation correlate with thrombosis and in some studies, coronary risk. Measurement of homocysteine levels should be reserved for individuals with atherosclerosis at a young age or out of proportion to established risk factors.

14. INFLAMMATION (Fig 5, 6):

An accumulation of clinical evidence shows that markers of inflammation correlate with coronary risk. Inflammatory cells in the plaque may contribute to plaque destabilization by producing matrix degrading metalloproteinases and by inducing smooth muscle cell apoptosis. Inflammatory cells also contribute to plaque thrombogenicity by releasing tissue factor- a procoagulant protein that activates the clotting cascade resulting in thrombin generation which leads to platelet aggregation and fibrin deposition. Inflammation in atherosclerotic plaque may be incited by a number of factors, which include oxidized LDL, cigarette smoking and possibly infectious agents. Variation of plasma levels of CRP can prospectively predict risk of myocardial infarction. CRP levels also correlate with outcome of patients with acute coronary syndrome. Elevated levels of the acute phase

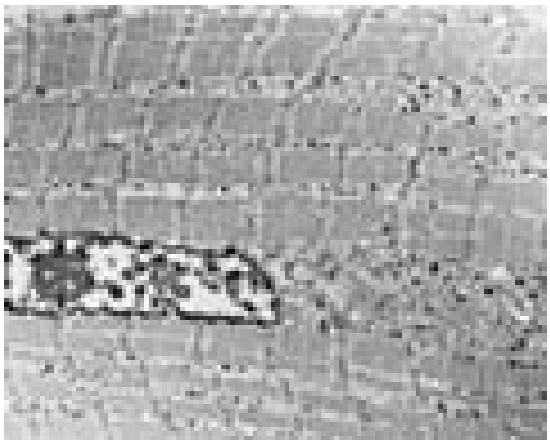


Nonischemic left ventricular myocardium

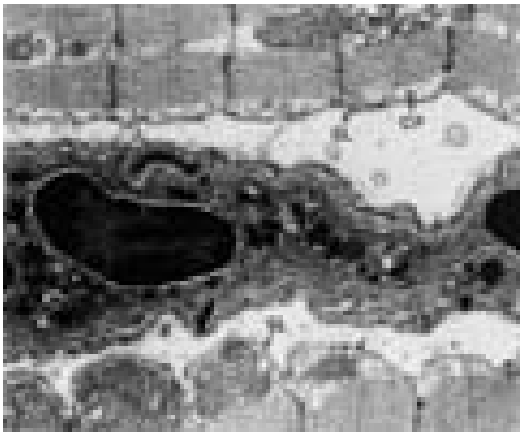


Reversible ischemic injury

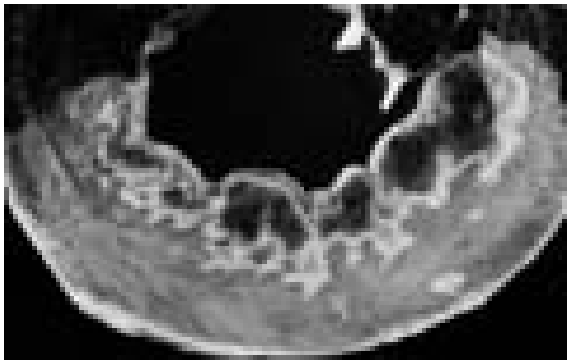
Early irreversible ischemic injury



Acute myocardial infarct after 24 hours of ischemia

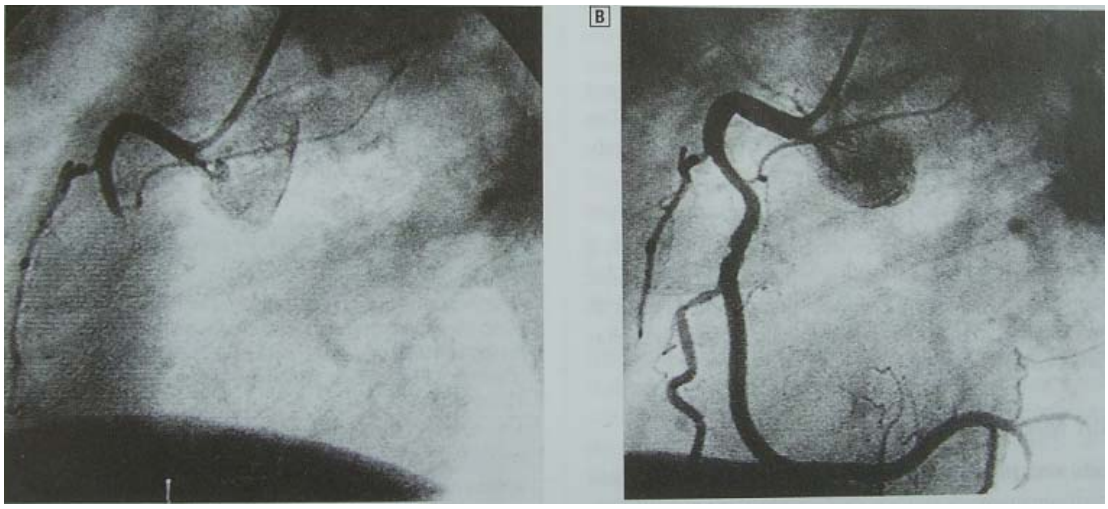


Effect of reperfusion after reversible ischemic injury

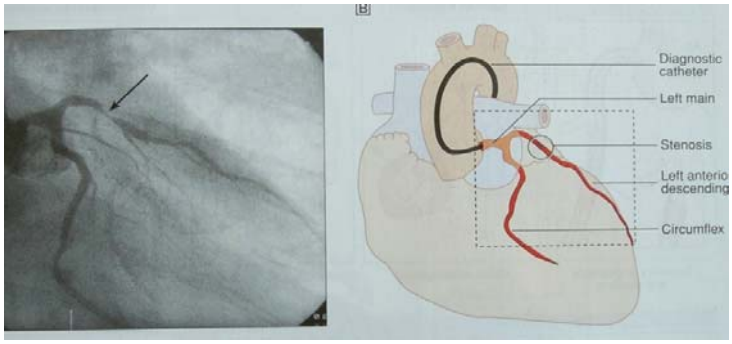


Identification of myocardial infarct with dehydrogenase staining

Fig5.Atherosclerosis- Histopathology



CORONARY ANGIOGRAM SHOWING BLOCK IN PROXIMAL RIGHT CORONARY ARTERY AND ITS REAPPEARANCE AFTER THROMBOLYTIC THERAPY



CORONARY ANGIOGRAM AND SCHEMATIC DIAGRAM SHOWING BLOCK IN LEFT ANTERIOR DESCENDING ARTERY

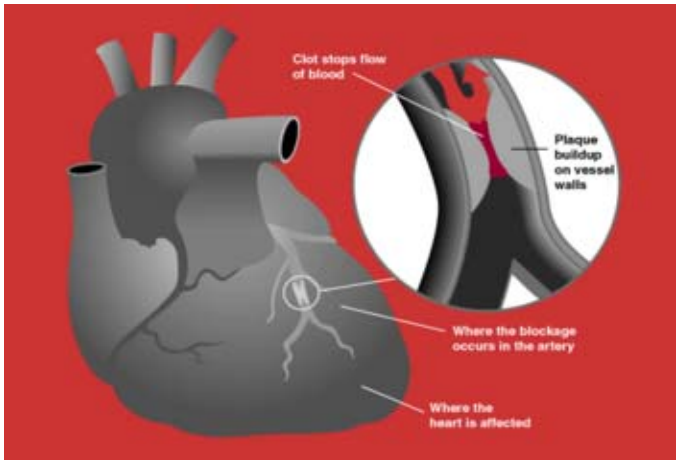


Fig 6. A myocardial infarction occurs when an atherosclerotic plaque slowly builds up in the inner lining of a coronary artery and then suddenly ruptures, totally occluding the artery and preventing blood flow downstream.

reactant CRP may merely reflect ongoing inflammation rather than a direct etiologic role of CRP in the coronary artery disease.

Systemic inflammatory state is evaluated in the acute phase by measuring proinflammatory mediators hs-CRP .C-reactive protein (CRP) has been well recognized as a strong independent predictor of short-term and long-term mortality after non–ST-segment elevation acute coronary syndromes.

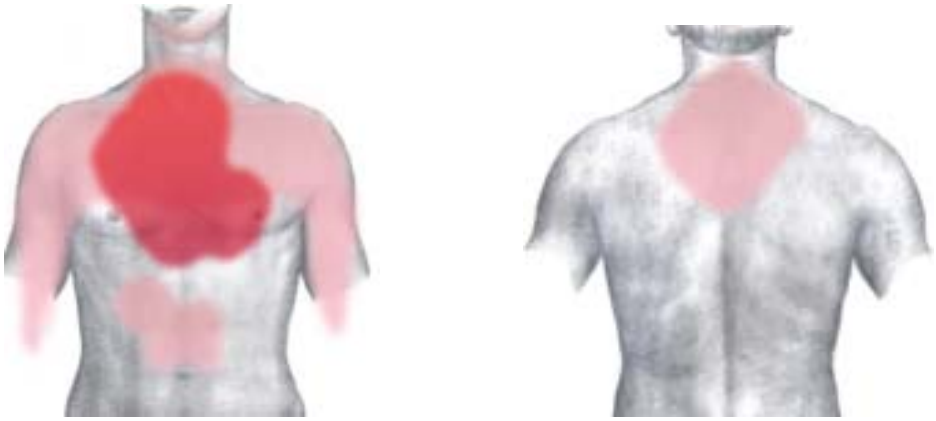
One source of inflammatory stimulus could arise from infectious agents. Interest has resurged in the possibility that infections may cause or contribute to atherosclerosis. Recent evidences support the role of Chlamydia pneumoniae, Cytomegalovirus, Herpes virus, H.pylori or other infectious agents in atherosclerosis and restenosis following coronary intervention.

Chronic bronchitis and chronic gingivitis (most often due to infection with Porphyromonus gingivalis) have also been linked to atherosclerotic vascular disease. These have been linked to Acute Coronary events based on seroepidemiologic data or identification of the organism in the atherosclerotic plaque.

D. CLINICAL FEATURES (Fig 7)

Retrosternal chest discomfort is the cardinal symptom of myocardial infarction, but breathlessness, vomiting, giddiness, syncope are common features. Patient is anxious and experiences the fear of impending death. Syncope is usually due to arrhythmia or profound hypotension. Vomiting and

Fig 7. Rough diagram of pain zones in myocardial infarction (dark red = most typical area, light red = other possible areas, view of the chest).



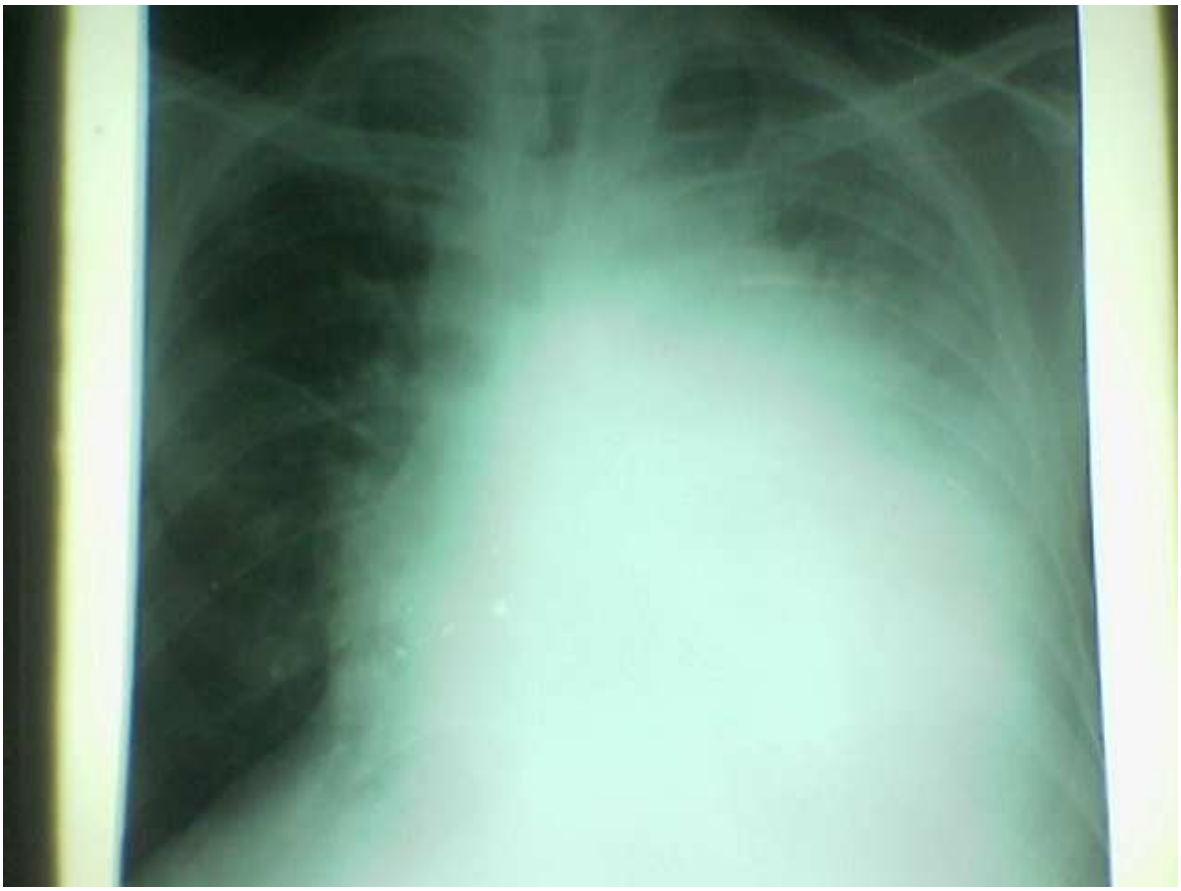


Fig 8: Chest X- Ray showing Pericardial effusion

sinus bradycardia are often due to vagal stimulation and are particularly common in patients with inferior wall myocardial infarction. Many deaths occur within the first hour. Development of cardiac failure reflects the extent of myocardial damage and is the major cause of death in those who survive the first few hours of infarction. On examination signs of sympathetic activity like sweating, tachycardia may be present. Vomiting and bradycardia may also be present. Signs of impaired myocardial function like hypotension, oliguria, cold peripheries, raised JVP, muffled heart sounds, S3, basal crepitations, pansystolic murmurs may also be present. Pericardial rub may be present.

E.INVESTIGATIONS

1. LABORATORY FINDINGS:

Myoglobin levels are the earliest to rise. Creatine Kinase starts to rise at 4 hours, peaks at about 12 hours falls to normal levels within 48-72 hours [45]. The most sensitive markers of myocardial cell damage are the Cardiac Troponins T and I which are released within 4-6 hours and remain elevated for upto 2 weeks. Myoglobin levels peak at 6 hours and returns to normal at 24 hours. Aspartate transaminase starts to rise about 12 hours after infarction and reaches peak on the first or second day returning to normal within 3 or 4 days. Lactate dehydrogenase peaks at 3-4 days remains elevated for upto 10 days.

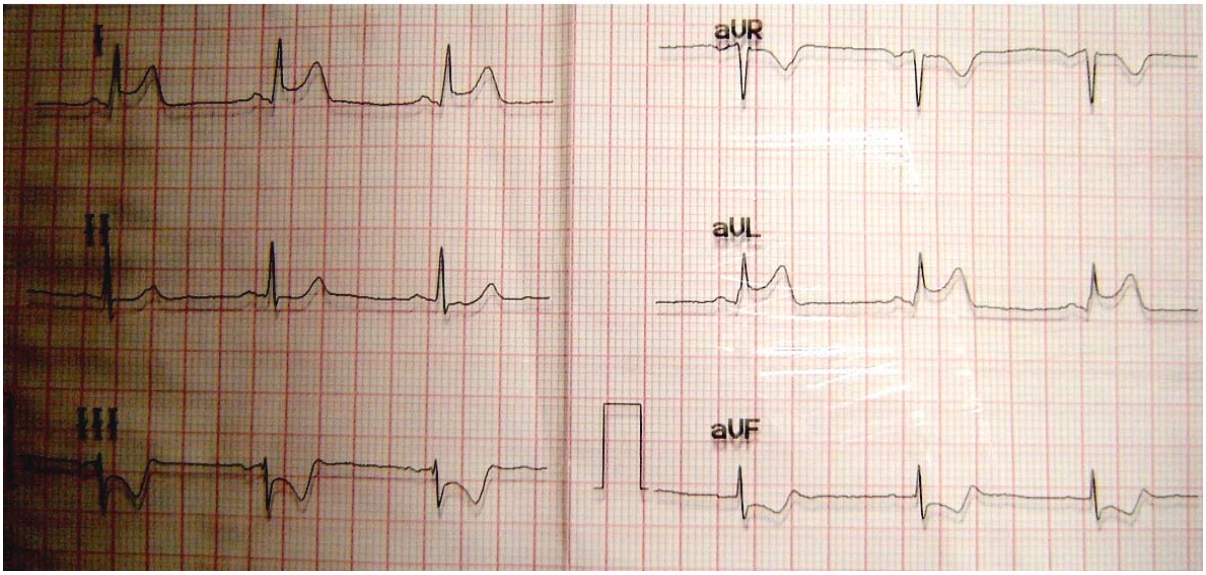
Lipid profile (cholesterol – total, LDL & triglycerides) may be raised. Leukocytosis is usual, reaching a peak on the first day. The ESR becomes raised.

2. CHEST RADIOGRAPHY (Fig 8):

Chest X-ray is important since it may show the consequences of ischaemic heart disease i.e. cardiac enlargement, ventricular aneurysm, signs of heart failure and pericardial effusion. These signs can support the diagnosis of ischaemic heart disease and are important in assessing the degree of cardiac damage.

3. ELECTROCARDIOGRAPHY(FIG 9):

The ECG is usually a sensitive and specific way of confirming the diagnosis; however it may be difficult to interpret if there is bundle branch block or evidence of previous myocardial infarction. Occasionally the initial ECG is normal and the diagnostic changes appear a few hours later. The earliest ECG change is tall and widened T waves followed by ST elevation. Later on there is diminution in the size of R wave and in full thickness infarction a Q wave begins to develop. Subsequently T wave becomes inverted.



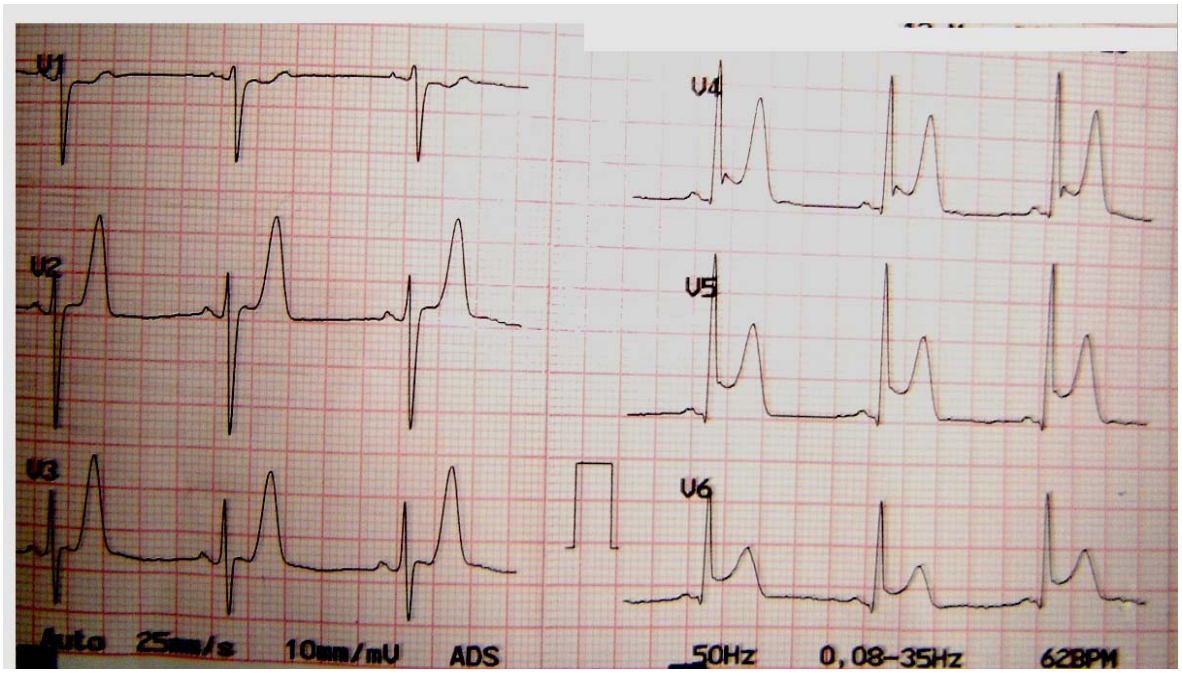


Fig 9. Electrocardiogram showing Acute Lateral wall Myocardial Infarction.

When there has been anteroseptal infarction abnormalities are found in one or more leads from V1 to V4, while Anterolateral Infarction produces abnormalities in V4-V6, aVL and in lead I. Inferior Wall Infarction is best shown in lead II, III and aVF, while at the same time leads I, aVL and the anterior chest leads may show reciprocal changes of ST depression. Infarction of the posterior wall of the left ventricle is not recorded in the standard leads by ST elevation or Q waves, but the reciprocal changes of ST depression and a tall R wave may be seen in leads V1-V3. Right ventricular infarction should be strongly suspected if, in the clinical setting of acute inferior wall myocardial infarction, there is ST elevation of 1mm or more in lead V1, V4R or any of the extra right precordial leads V4R-V6R.

4. ECHOCARDIOGRAPHY (Fig 10):

Two dimensional echocardiography can be done to assess the cardiac chamber size, regional wall motion abnormalities, left ventricular hypertrophy, valve leaflet thickness and mobility, valve calcification, appearance of subvalvular and supravalvular structures, pericardial effusion, intracardiac masses and great vessels.

Doppler echocardiography is done to assess valve regurgitation, valve stenosis, valve area, valve gradients, intracardiac pressures, intracardiac shunts and ventricular diastolic filling.

Transesophageal echocardiography is used to assess aortic disease, infective endocarditis, to find out source of embolism, abnormalities of mitral prostheses etc.

Stress echocardiography is done to find out new regional wall motion abnormalities, declining ejection fraction and increase in end systolic volume which are indicators of myocardial ischemia.

5. RADIONUCLIDE SCANNING:

A radionuclide ventriculogram can be used to assess left ventricular function. Infarct 'avid' scanning is possible because some isotopes (e.g. Technetium) are taken up by freshly infarcted myocardium. This may help to establish the diagnosis in some patients who present after a cardiac arrest when it is sometimes difficult to interpret any ECG and enzyme changes.

F. COMPLICATIONS OF MYOCARDIAL INFARCTION [37]

1. ARRHYTHMIAS:

A. TACHYARRYTHMIAS:

a. Premature ventricular complexes:

The commonest arrhythmia is premature ventricular complexes. This can be suppressed with IV lignocaine 100mg given as bolus. To prevent recurrence lignocaine infusion of 500-1000mg in 500ml of 5% dextrose is administered at a rate of 1-2mg/min. premature ventricular complexes can be

forerunners of life threatening ventricular tachycardia or ventricular fibrillation.

b. Ventricular Tachycardia:

It needs immediate attention and should be treated by IV lignocaine. In case the drug is ineffective, DC shock of 150-200 joules will be effective in majority of the cases. Repeated attacks of ventricular tachycardia can be prevented by IV infusion of lignocaine, amiodarone or mexilitene. Ventricular pacing may also be effective.

c. Ventricular Fibrillation:

The patient is pulseless and will have features of cardiac arrest. Immediate thump on the chest and an external cardiac massage is required. Defibrillation should be one by DC shock of 200-400 joules. Patients with repeated episodes may benefit from intravenous bretylium, amiodarone, mexilitene or lignocaine.

d. Supraventricular Arrhythmias:

Atrial premature beats do not need specific treatment. However supraventricular arrhythmia, atrial flutter and atrial fibrillation require treatment with digoxin or verapamil.

e. Accelerated Ventricular or Junctional Rhythm:

Normally pace maker cells in the AV junctional and ventricular myocardium have a rate of 40-60/min. however in the settings of myocardial infarction, especially in acute inferior wall myocardial infarction, the rate of these pacemakers increases to about 80-120/min. Such a rhythm does not require specific treatment and is normally self limiting. If hemodynamic compromise occurs IV atropine (1.2mg) suppresses it by increasing the sinus rate.

B. BRADYARRHYTHMIAS AND CONDUCTION DISTURBANCES:

a. Sinus Node Dysfunction:

It may present as sinus bradycardia, sinus arrest or sinoatrial block usually due to vagal stimulation or in the settings of inferior wall infarction due to sinus node ischaemia. Symptoms of profound hypotension and shock may occur and occasionally asystole and cardiac arrest. Immediate treatment consists of IV atropine (1.2mg). If atropine is ineffective or this problem is persistent or recurring temporary pace making is necessary.

b. AV Nodal Block:

It usually occurs with inferior wall myocardial infarction as first degree AV block, Wenkebach's block or complete AV block. These blocks are usually transient and respond to IV atropine. If giddiness, hypotension or

other evidence of hemodynamic compromise occurs or if the ventricular rate is less than 50/min temporary pacing may be required.

c. Distal Conduction Disturbances:

The distal conduction system consists of the right bundle and the anterior and posterior fascicles of the left bundle. Since the major blood supply to this part of the conduction system comes from the left coronary artery, conduction defects in the bundle branches are common in anterior wall infarction. Block of one of the fascicles of the left bundle does not have an ominous prognosis. However acute right or left bundle branch block or bifascicular block carries an ominous prognosis. Block in the three fascicles results in complete heart block with an unstable ventricular escape rhythm at a rate of 20-40 beats per minute. Clinical manifestations consists of syncopal attacks, hypotension and may lead to cardiac arrest. Temporary pacemaker insertion is necessary. Temporary pacing is also indicated in patients who develop bifascicular blocks since this can be a precursor of trifascicular block. Patients with bifascicular block who develop trifascicular block may need permanent pacemaker implantation.

2. ISCHAEMIA:

Post infarction angina occurs in upto 50% of patients. This is due to residual stenosis in infarct related vessel despite successful thrombolysis.

3. ACUTE CIRCULATORY FAILURE:

Hemodynamic evidence of left ventricular dysfunction appears when contraction is seriously impaired in 20-25% of the left ventricle. Infarction of more than 40% of left ventricle results in Cardiogenic Shock which carries a bad prognosis.

4. PERICARDITIS:

This may occur at any stage but is particularly common on the second and third day. The patient may recognize a different pain that is positional and worsens on inspiration. Dressler's Syndrome may occur between 2 weeks and 3 months after acute myocardial infarction [46] and has an autoimmune basis often accompanied by pleural and pericardial effusions, fever and raised ESR. Treatment requires the use of steroids.

5. MECHANICAL COMPLICATIONS:

a. Mitral regurgitation:

It is due to ischaemia or rupture of papillary muscle and is recognized by the presence of systolic murmur at the apex. If trivial, it is of no hemodynamic significance. However severe mitral regurgitation can induce

life threatening left ventricular failure and cardiogenic shock and may warrant urgent coronary angiography followed by coronary bypass surgery and mitral valve replacement.

b. Ventricular septal defect:

It is a defect due to rupture of infarcted interventricular septum and is recognized by the presence of pansystolic murmur at the left sternal border. Diagnosis is possible by Echo-Doppler studies. It produces severe left heart failure and needs immediate surgical intervention.

c. Cardiac Rupture:

It is a serious complication which results in cardiogenic shock and almost 100% mortality. Emergency treatment by pericardial tapping may prove life saving. Rare cases have been saved by emergency surgery.

6. OTHER COMPLICATIONS:

a. Left Ventricular Aneurysm:

The infarcted segments are dilated and show paradoxical movement and compromised left ventricular hemodynamics. It is recognized by persistent ST elevation in ECG and dyskinesia seen in echocardiography and radionuclide or contrast ventriculography. It may result in persistent left ventricular failure, arrhythmias and systemic embolism. Treatment consists of aneurysmectomy and associated coronary artery bypass surgery if so indicated.

b. Thromboembolism:

Formation of a thrombus within the left ventricle followed by systemic arterial embolism leading to occlusion of a peripheral artery requires immediate surgical embolectomy in accessible vessels. Pulmonary embolism originates in the leg veins due to prolonged immobilization. These are prevented by anticoagulation. Massive embolism may result in shock and sudden death. Thrombolytic therapy and embolectomy are occasionally required. The condition can be prevented by low molecular weight heparin.

MATERIALS AND METHODS

MATERIAL AND METHODS

During the period from 1st Jan 2007 to 30th Jun 2007 100 patients admitted with acute myocardial infarction in coronary care unit, CMC Hospital, Coimbatore, with a window period of less than 12 hrs were analysed.

In the present study, the WHO criterion 4 was followed to diagnose myocardial infarction. Among the following criteria atleast two of the three had to be present for accepting the case as myocardial infarction (Fig 11).

1) Typical history of retrosternal chest pain.

2) Serial ECG changes [47, 48, 49]: Appearance of pathological Q wave or appearance of ST elevation with T wave inversion in atleast 2 or more leads, new onset left bundle branch block (LBBB)..

3) Raised CPK-MB taken 12-24 hours after the onset of symptoms.

After careful history taking, physical signs were recorded in detail. History of prolonged hypertension, diabetes were taken into consideration. History of oral contraceptive intake, cigarette smoking were noted.

Menstrual history was recorded for females. Serum analysis of High sensitive C-reactive protein, WBC count was taken before thrombolysis. Cholesterol and blood sugar were done. Electrocardiography was taken in all

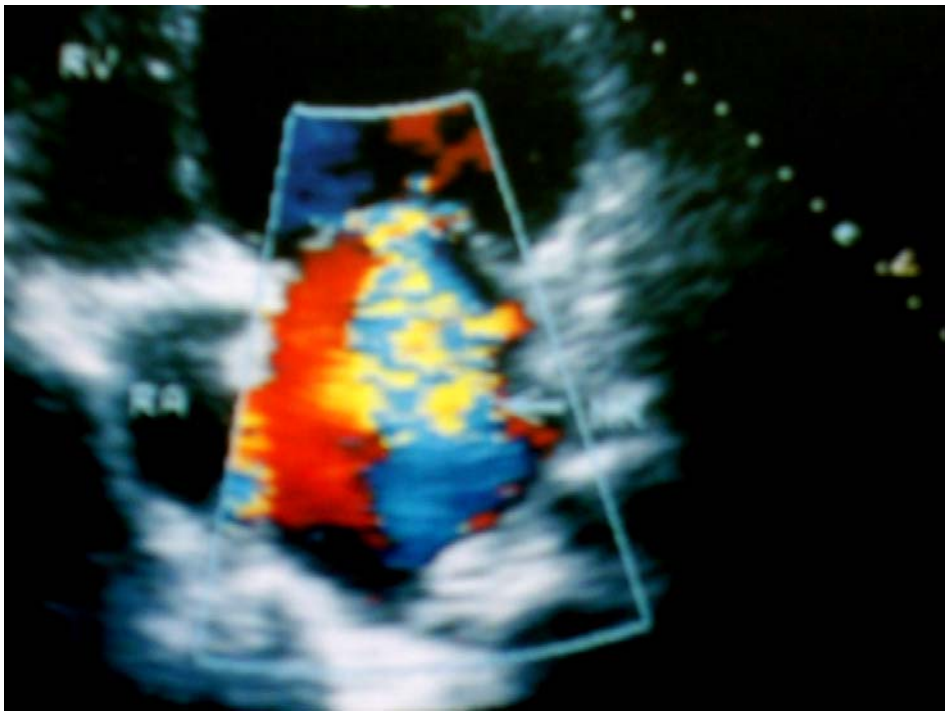


Fig 10. ECHO SHOWING ACUTE MITRAL REGURGITATION

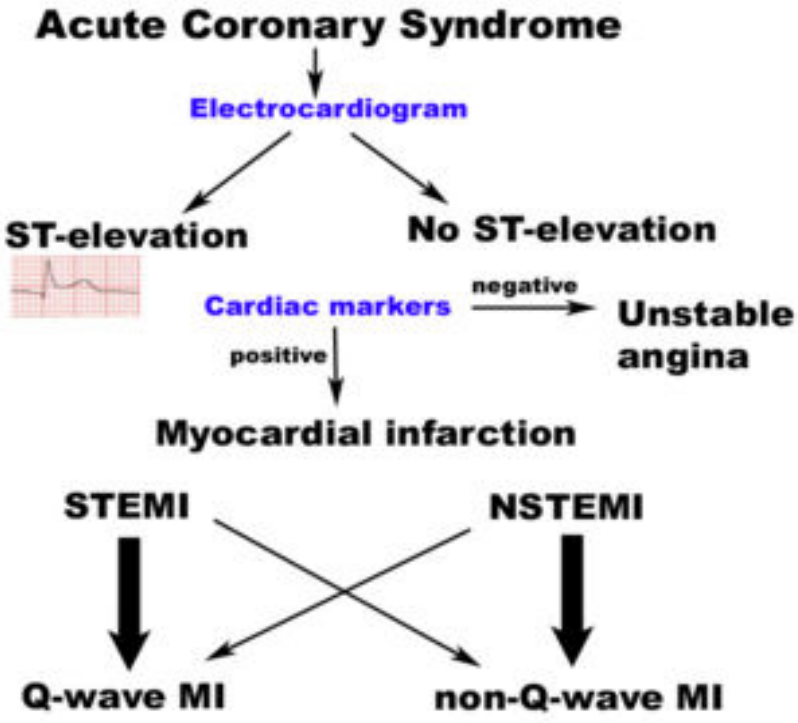


Fig 11. Schema for diagnosis of Myocardial Infarction

cases at the time of admission and Echocardiography was done on day 3.Window Period were also recorded.

Serum cholesterol was considered to be increased if it was greater than 200mgm%. Waist circumference more than 94cms for males and 80cms for females was taken as obese. BMI were also recorded. The type and extent of infarction were judged by the Q wave and ST elevation in serial ECGs.

Myocardial reperfusion injury was defined as the presence of persistent ST-segment elevation despite successful coronary intervention (≥ 50% of the initial value).

Despite successful early recanalization of an occluded artery by thrombolysis, up to one-third of AMI patients still fail to obtain complete myocardial reperfusion due to a process of myocardial reperfusion injury[50, 51]. This phenomenon is characterised by an impairment of microcirculatory flow and by ongoing ischemia and tissue necrosis[52, 53].

Although the underlying mechanisms of reperfusion injury are still not fully elucidated, there is accumulating evidence that local inflammatory responses with infiltration of leucocytes in the capillary circulation and release of oxygen-free radicals play a key role in this reperfusion-related tissue injury[54, 55]. However, the occurrence and the extent of reperfusion injury is variable and is not solely related to the severity and duration of myocardial

ischemia[51, 56]. Whether the pre-existing systemic inflammatory state might be another important determinant of this phenomenon is unknown.

ST-segment elevations of less than 50% (ST < 50%) indicated good myocardial reperfusion without significant ongoing tissue injury. There is a good reproducibility of these measurements in previous reports[57].

Once diagnosis was established routine management was carried out which included bed rest, oxygen therapy, sedation with narcotics, coronary vasodilator, thrombolytic and antiplatelet treatment. Blood pressure and pulse were recorded and continuous ECG monitoring was done for the first 48 hours along with ½ hourly pulse, blood pressure, temperature and respiration recording.

A careful observation was made on all patients for the development of arrhythmias, left ventricular failure, cardiogenic shock and other complications. They were treated accordingly.

A detailed record of the illness was maintained.

Cardiovascular Risk Classification

The AHA and CDC defined three risk groups as follows (TABLE 2):

Risk Level	CRP (mg/L)	CRP (mg/dL)
Low	<1.0	<0.10
Average	1.0 - 3.0	0.10 – 0.30
High	>3.0	>0.30

TABLE 2

Levels of hs-CRP are also elevated among those with acute coronary syndromes at high risk for recurrent events and among post-MI patients at high risk for recurrent instability. These effects are independent of other risk factors and appear to add to the predictive value of lipid screening in terms of risk prediction.

Plasma C-reactive protein was measured at the time of admission by immunonephalometry method. The normal value of the C-reactive protein is taken as 0.08 mg/L. Electrocardiography is done at the time of admission and after 1 hr of thrombolysis. Echocardiographic study is done on day three of admission and ejection fraction is estimated by modified Simpson's method. Patients are classified into three groups based on level of C-reactive protein: those with low C-reactive protein level (<1 mg/L), intermediate (1-3 mg/L) and those with high C-reactive protein level (>3 mg/L).

Two ml of blood is taken before thrombolysis and plasma CRP level was estimated by immunonephalometry method by auto analyzer. Normal value of CRP is < 0.8 mg/dl. Patients should not have other conditions known to modify the plasma CRP level i.e. collagen vascular disease, advanced liver disease, malignancy, septicemia, other inflammatory infectious diseases. i.e. chronic obstructive pulmonary disease/bronchitis, pulmonary tuberculosis and arthritis, as these conditions are known to modify CRP levels.

All patients with AMI are thrombolysed with 1.5 million units of streptokinase. Aspirin, isosorbide dinitrate, beta blocker etc. are used in routine manner. Echocardiography is done in all patients on day three; the LV volume and EF are evaluated by modified Simpson's method. Doppler diastolic indices are also calculated.

Methodology: Latex immunonephalometry

Specimen: Serum or plasma

Volume: 1 mL

Minimum Volume: 0.5 mL

Container: Red-stopper tube

Collection: Separate serum or plasma from cells within 1 hour after collection.

Storage Instructions: Refrigerate (maximum 8 days). May be frozen at -25°C or lower if samples are frozen within 24 hours after collection. Repeated freeze-thaw cycles are to be avoided.

Causes for Rejection: Gross lipemia in tissue injury, infections or inflammation, which may cause elevated CRP levels, should also be considered when interpreting results.

OBSERVATION

OBSERVATIONS

A. HIGH SENSITIVE C-REACTIVE PROTEINS:

Risk stratification following acute myocardial infarction

According to the level of HS-CRP level just before the time of thrombolysis patients were classified into the following groups (TABLE 3),

LOW RISK <1 mg/dl		INTERMEDIATE RISK 1mg/dl- 3mg/dl		HIGH RISK >3 mg/dl	
MALE	FEMALE	MALE	FEMALE	MALE	FEMALE
20	3	19	6	43	9
23		25		52	

TABLE 3

In this series of 100 patients, 23 were in the low risk group, 25 were in intermediate risk group and 52 were in the high risk group **(**Graph 1, 2).

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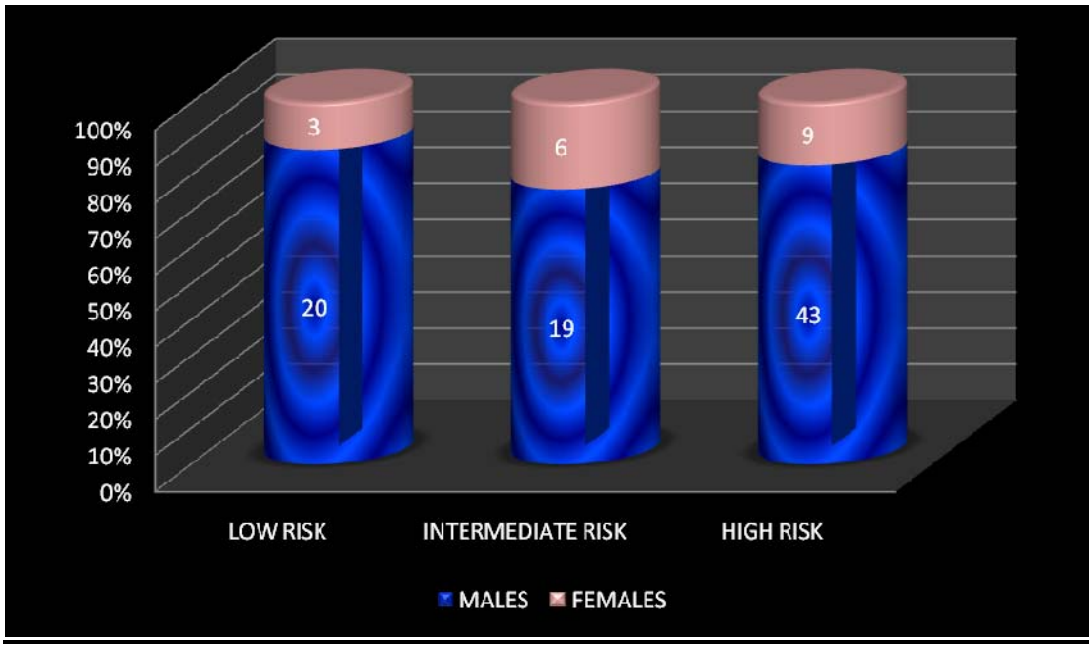
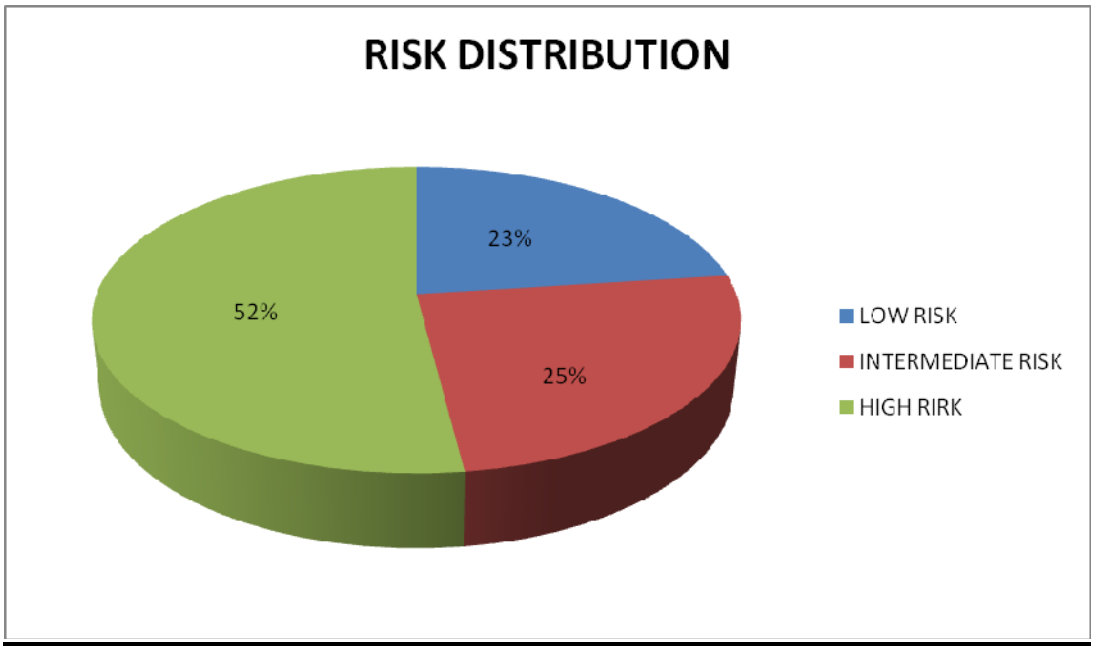
GRAPH 1



GRAPH 2



ANALYSIS OF WINDOW PERIOD (GRAPH 3, TABLE-4, 5, 6)

LOW RISK GROUP:

CHARACTERISTICS OF WINDOW PERIOD	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 1 HR	**4**	**3**	**1**
1 HR- 3 HRS	**4**	**3**	**1**
3 HRS- 6 HRS	**9**	**8**	**1**
> 6 HRS- 12 HRS	**6**	**6**	**0**

TABLE 4

INTERMEDIATE RISK GROUP:

CHARACTERISTICS OF WINDOW PERIOD	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 1 HR	**0**	**0**	**0**
1 HR- 3 HRS	**5**	**3**	**2**
3 HRS- 6 HRS	**18**	**14**	**4**
> 6 HRS- 12 HRS	**2**	**2**	**0**

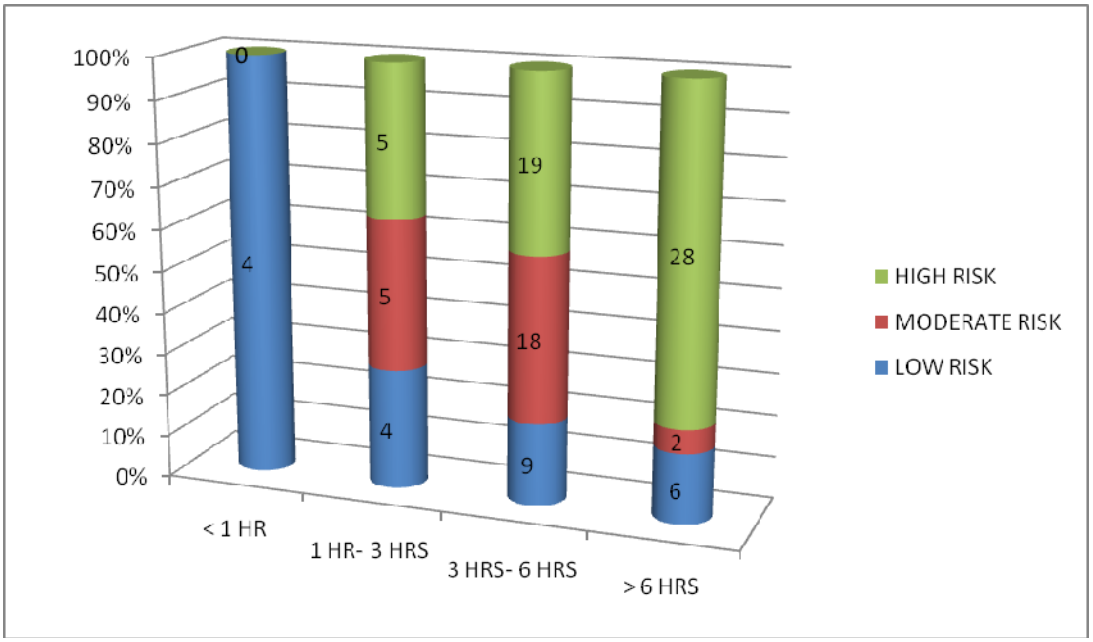
TABLE 5

HIGH RISK GROUP:

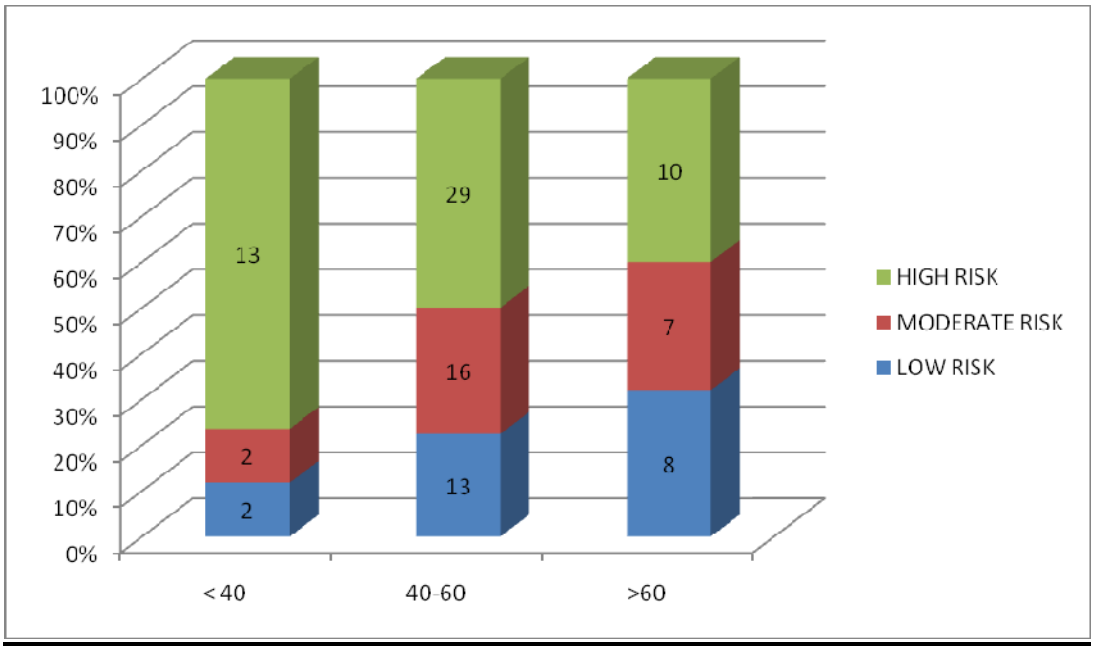
CHARACTERISTICS OF WINDOW PERIOD	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 1 HR	**0**	**0**	**0**
1 HR- 3 HRS	**5**	**4**	**1**
3 HRS- 6 HRS	**19**	**16**	**3**
> 6 HRS- 12 HRS	**28**	**23**	**5**

TABLE 6

GRAPH 3. WINDOW PERIOD DITRIBUTION



GRAPH 4. EJECTION FRACTION DISTRIBUTION



ANALYSIS OF EJECTION FRACTION (GRAPH 4, TABLE-7, 8, 9)

LOW RISK GROUP:

CHARACTERISTICS OF EJECTION FRACTION IN %	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 40	**2**	**2**	**0**
40 – 60	**13**	**12**	**1**
>60	**8**	**6**	**2**

TABLE 7

INTERMEDIATE RISK:

CHARACTERISTICS OF EJECTION FRACTION IN %	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 40	**2**	**1**	**1**
40 – 60	**16**	**13**	**3**
>60	**7**	**4**	**3**

TABLE 8

HIGH RISK:

CHARACTERISTICS OF EJECTION FRACTION IN %	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 40	**13**	**10**	**3**
40 – 60	**29**	**25**	**4**
>60	**10**	**9**	**1**

TABLE 9

<u>ANALYSIS OF REPERFUSION INJURY</u> (GRAPH 5, TABLE-10, 11, 12)

LOW RISK GROUP:

CHARACTERISTICS OF REPERFUSION INJURY BY ST-SEGMENT REGRESSION IN %	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 50	**1**	**1**	**0**
>50	**22**	**20**	**2**

TABLE 10

INTERMEDIATE RISK GROUP:

CHARACTERISTICS OF REPERFUSION INJURY BY ST-SEGMENT REGRESSION IN %	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 50	**2**	**2**	**0**
>50	**23**	**18**	**5**

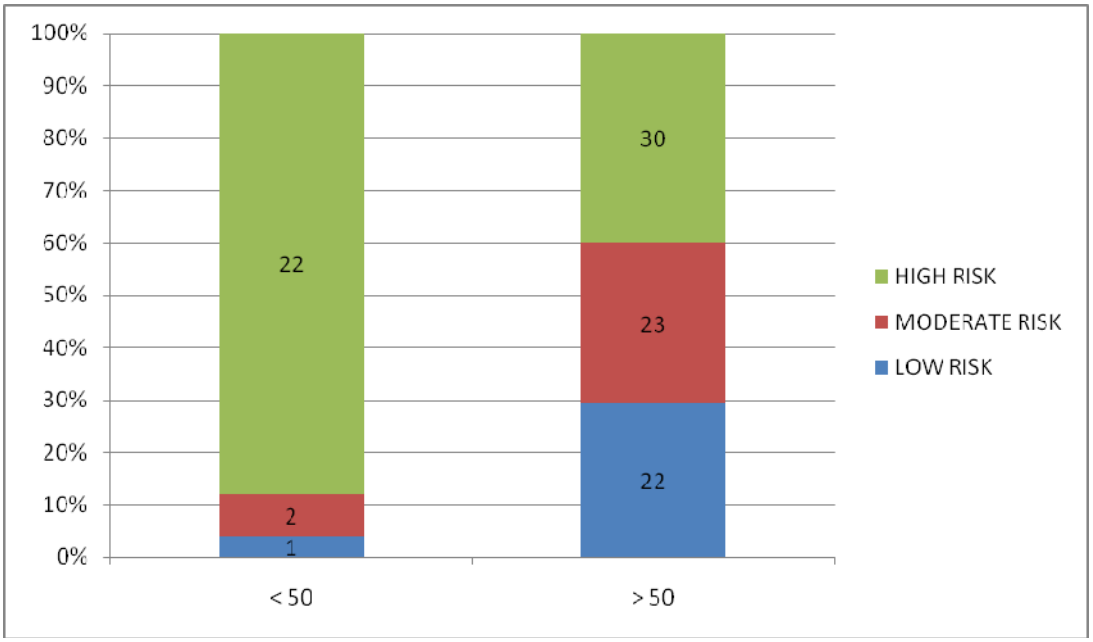
TABLE 11

HIGH RISK:

CHARACTERISTICS OF REPERFUSION INJURY BY ST-SEGMENT REGRESSION IN %	TOTAL NO: OF PATIENTS	MALE	FEMALE
< 50	**22**	**15**	**7**
>50	**30**	**28**	**2**

TABLE 12

GRAPH 5. REPERFUSION INJURY DISTRIBUTION



CHARACTERISTICS	LOW RISK	INTERMEDIATE RISK	HIGH RISK
AGE	46.4 ± 7.86	51 ± 6.56	50.94 ± 8.63
SEX			
MALE	20(82)	19(82)	43(82)
FEMALE	3(18)	6(18)	9(18)
RISK FACTORS (%)			
DIABETES	5(29)	9(29)	15(29)
HYPERTENSION	7(49)	16(49)	26(49)
SMOKING	17(65)	14(65)	34(65)
OC PILLS	1(1)		
HYPERHOMOCYSTEINEMIA	0(3)	1(3)	2(3)
OBESITY	1(22)	7 (22)	14(22)
MEAN HS-CRP	0.75 ± 0.239	2.02 ± 0.083	6.72 ± 1.76
EJECTION FRACTION%	57 ± 11	54.12 ± 10.76	50 ± 10.8

TABLE 13. CHARACTERISTICS

SITE OF INFARCTION:

The most common site of infarction in the present study was Extensive Anterior Wall Myocardial Infarction. Second common site of infarction was in the inferior wall.

Table 14: SITE OF INFARCTION IN BOTH GROUPS

S.NO	SITE OF INFARCTION	NO.OF CASES
1	ANTEROSEPTAL	24
2	ANTERO LATERAL	4
3	EXTENSIVE ANTERIOR	48
4	INFERIOR WALL	21
5	POSTERIOR WALL MI	3
	TOTAL	100

COMPLICATIONS:

Unstable angina-3

Non –ST Elevation Myocardial Infarction (NSTEMI)-1

Death-3(Acute mitral regurgitation-1)

B. RISK FACTORS

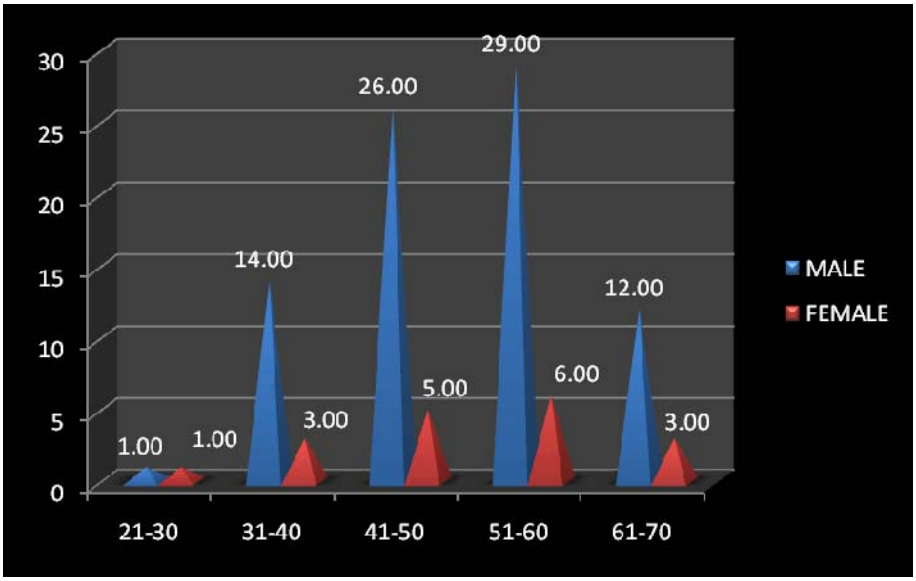
1) AGE & SEX (CHART 6):

Age and sex are definite risk factors. Total number of patients in this series was 100. The overall incidence is more in males compared to females.

Table 15: AGE DISTRIBUTION OF PATIENTS

S.No	AGE GROUP	MALE	FEMALE	No. OF PATIENTS	PERCENTAGE
1	21-30	1	1	2	2
2	31-40	14	3	17	17
3	41-50	26	5	31	31
4	51-60	29	6	35	35
5	61-70	12	3	15	15
	TOTAL	82	18	100	100

CHART 6: AGE AND SEX DISTRIBUTION



2) MENSTRUAL HISTORY:

In the present study 18 women were involved. The incidence of myocardial infarction was higher in the postmenopausal women (Table 16, Chart 7).

TOTAL NO. OF PATIENTS	PRE MENOPAUSAL	PERI MENOPAUSAL	POST MENOPAUSAL
	4	4	10
PERCENTAGE%	22.22	22.22	55.55

TABLE16

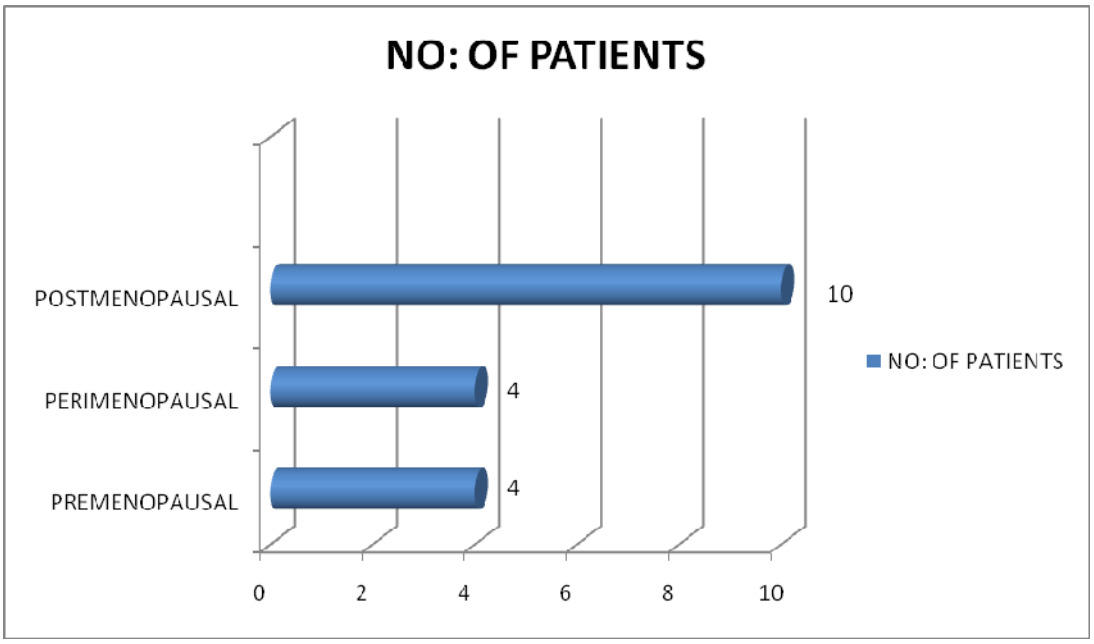


CHART 7

3) ORAL CONTRACEPTIVE PILLS:

In the present study only 1 premenopausal woman was taking oral contraceptive pills for more than 3 months.

4) CIGARETTE SMOKING (TABLE 17, CHART 8):

In the present study 65 (79.2%) males out of 82 males were cigarette smokers.

SMOKERS	65
NONSMOKERS	17

TABLE 17

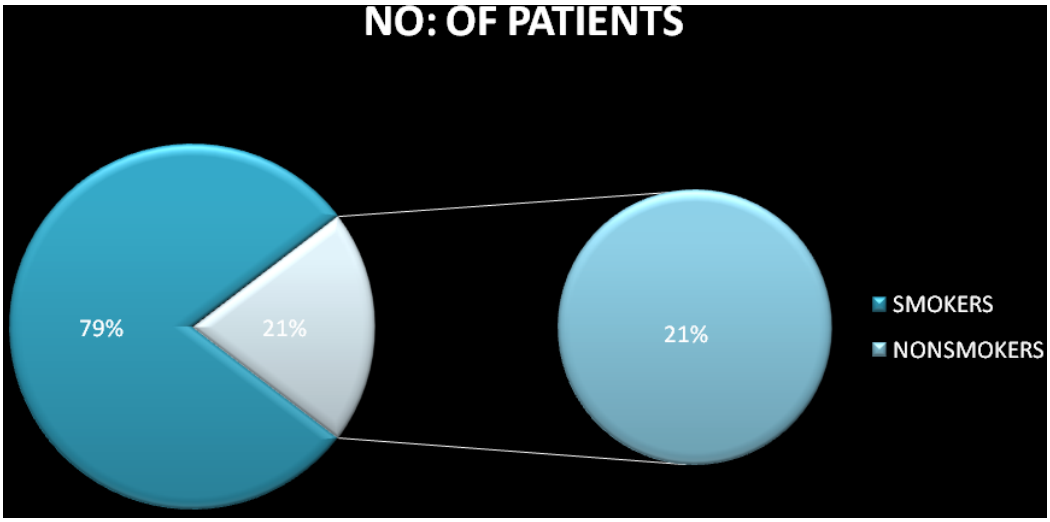


CHART 8

5) ABDOMINAL OBESITY (TABLE 18, CHART- 9, 10):

In this study the waist circumference was recorded for all patients. Waist circumference more than >94cms for males and >80cm for females was taken as abdominal obesity.

In the present study 22 patients (22%) were obese, of which 14.64% were males and 55.55% were female patients.

	NON OBESE	OBESE MALE	OBESE FEMALE
STATURE	78	12	10

TABLE 18

Body mass index

WHO CLASSIFICATION OF OBESITY

TABLE 19

CATEGORY	BMI/QUETELET
UNDER WEIGHT	< 18.5
HEALTHY WEIGHT	18.5- 24.9
OVER WEIGHT	25 – 29.9
MODERATELY OBESE	30 – 34.9
SEVERLY OBESE	35 – 39.9
MORBIDLY OBESE	>40

In this series 33(29 male and 4 female) patients were overweight, 8(6 male and 2 female) were moderately obese, 1 male was severely obese and none were morbidly obese.

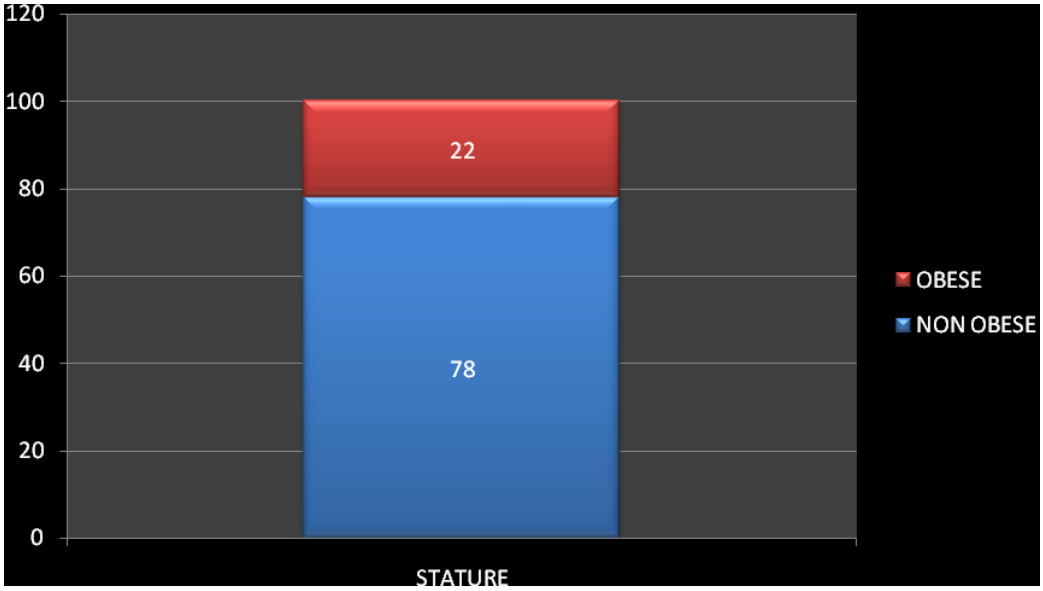


CHART 9

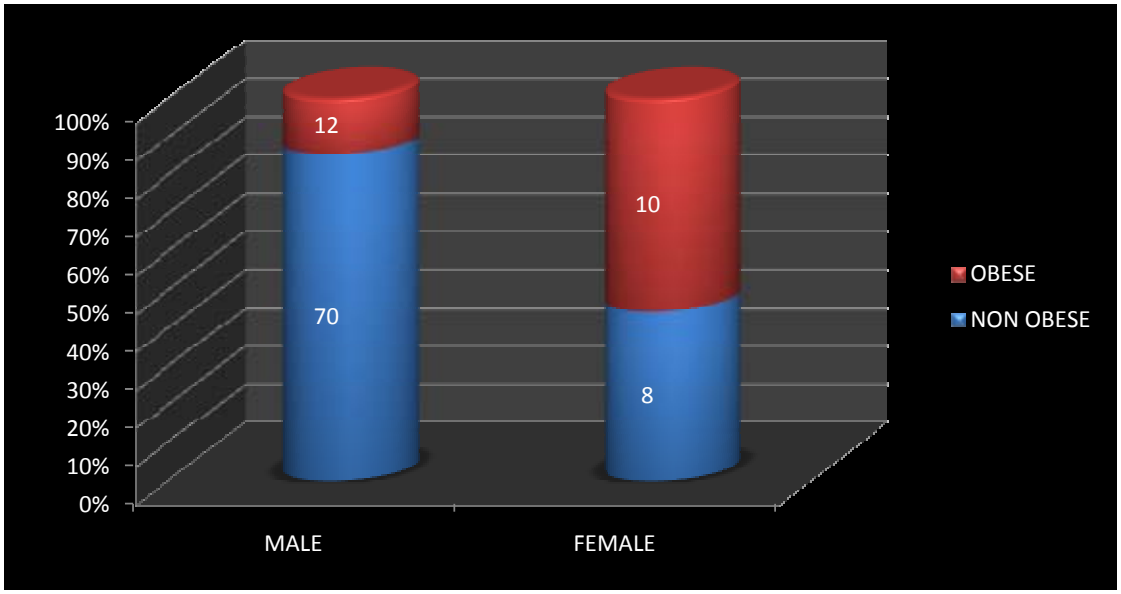


CHART 10

6) DIABETES MELLITUS:

In the present study, information regarding the prevalence of manifest diabetes was obtained from the interviews. The patient was considered diabetic when there was a history of polyuria, polyphagia, and polydypsia, treatment with insulin or oral hypoglycemic agents. Increased blood sugar was also taken into consideration. ADA 2005 guidelines were followed.

In the present series 29 (29%) patients were diabetic. 23(79.3%) were males and 6(20.6%) were females among the diabetics (TABLE 20, CHART 11, 12).

STATUS	PATIENTS
NON DIABETES	71
DIABETIC MALE	23
DIABETIC FEMALE	6

TABLE 20

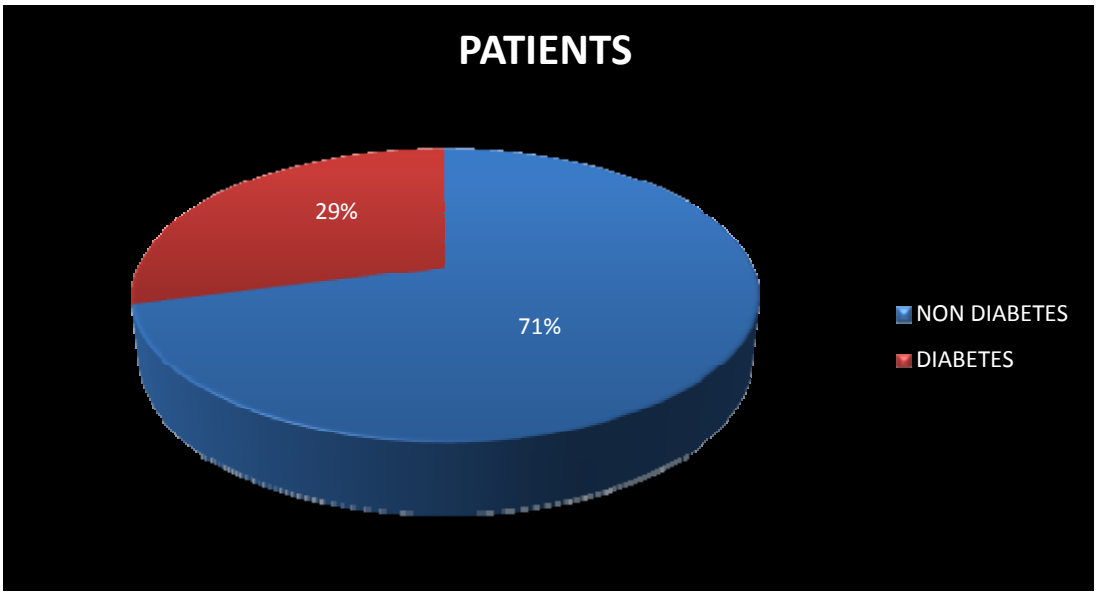


CHART 11

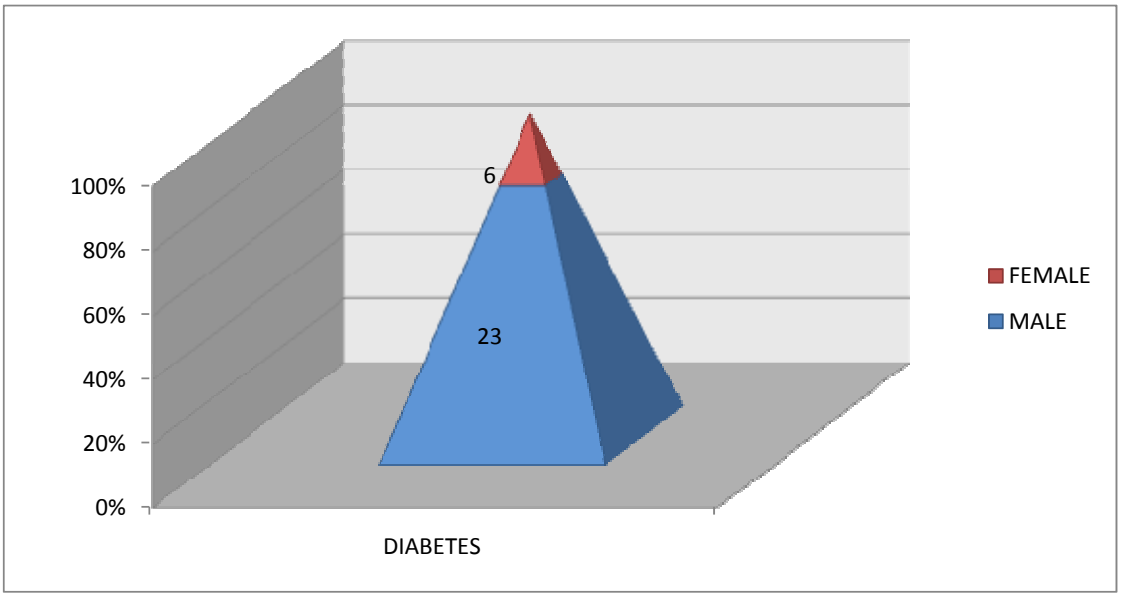


CHART 12

In the present study it is more common in males than in females. So diabetes had an important role both in males and females.

7) HYPERTENSION:

In the present series blood pressure was routinely checked for minimum three consecutive days. History of hypertension and the drugs they were taking were checked. Hypertension was noted in 49 (49%) patients, of which20 (42.5%) were newly detected hypertensive's (TABLE -21, 22, CHART- 13, 14). Hypertension was noted in 39 male and 10 female. Of the newly detected 75% were male and 25% were female.

STATUS	PATIENTS
NORMOTENSIVES	51
HYPERTENSIVE MALE	39
HYPERTENSIVE FEMALE	10

TABLE 21

STATUS	NEW HYPERTENSIVES
MALE	15
FEMALE	5

TABLE 22

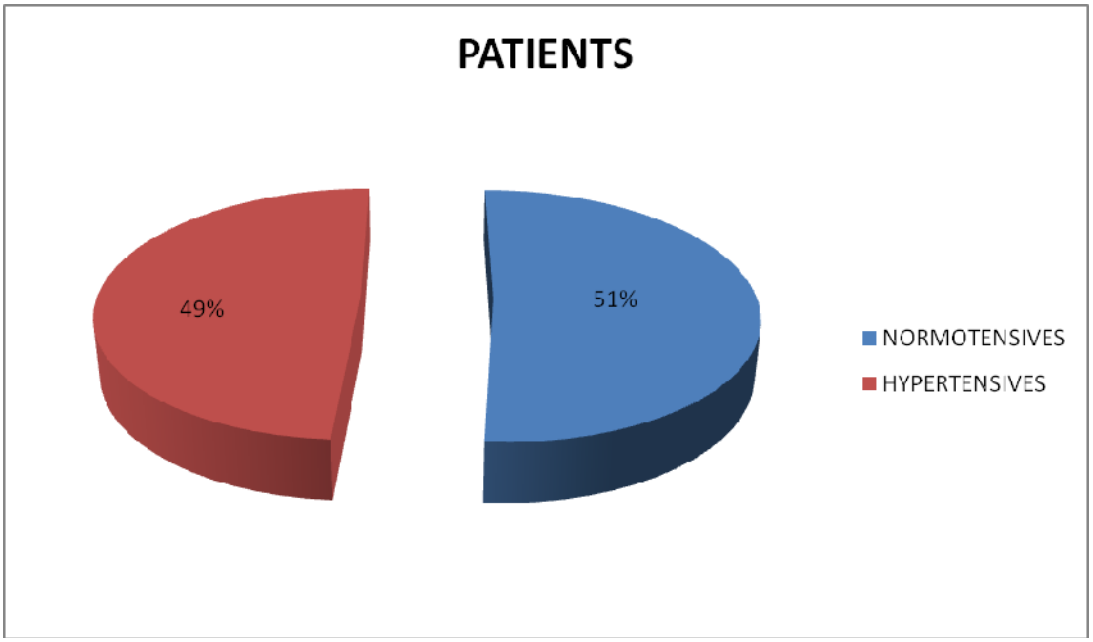


CHART 13

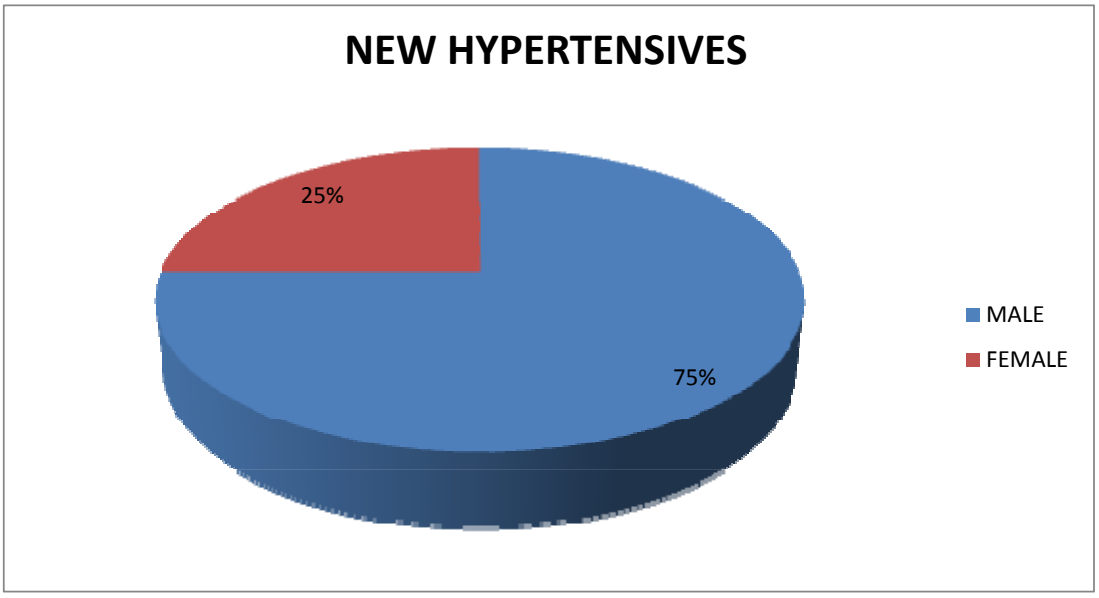


CHART 14

8) SERUM LIPIDS (TABLE-23, 24, CHART -15, 16):

In the present study cholesterol levels >200mg% were considered hypercholesterolemia, hypertriglyceridemia >160mg% and HDL < 40mg% were considered as risk factors. Hypercholesterolemia was seen in 44% patients. Of these 75% were male and 25% were female. Hypertriglyceridemia was seen in 33% patients.

STATUS	DYSLIPIDEMIA IN %
HYPERCHOLESTEROLEMIA	44
HYPERTRIGLYCERIDEMIA	33
LOW HDL	27

TABLE 23

Of these 75.7%were male and 24.24% were female. Low HDL was seen in 27% patients. Of these 22(81.4%) were male and 5(18.5%) were female.

STATUS	TOTAL IN %	MALE	FEMALE
HYPERTRIGLYCERIDEMIA	33	25	8
HYPERCHOLESTEROLEMIA	44	33	11
LOW HDL	27	22	5

TABLE 24

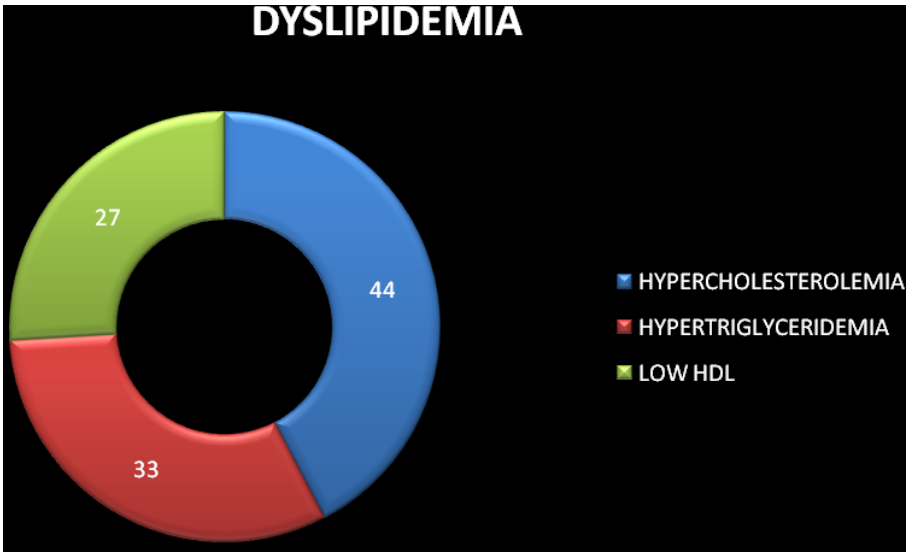


CHART 15

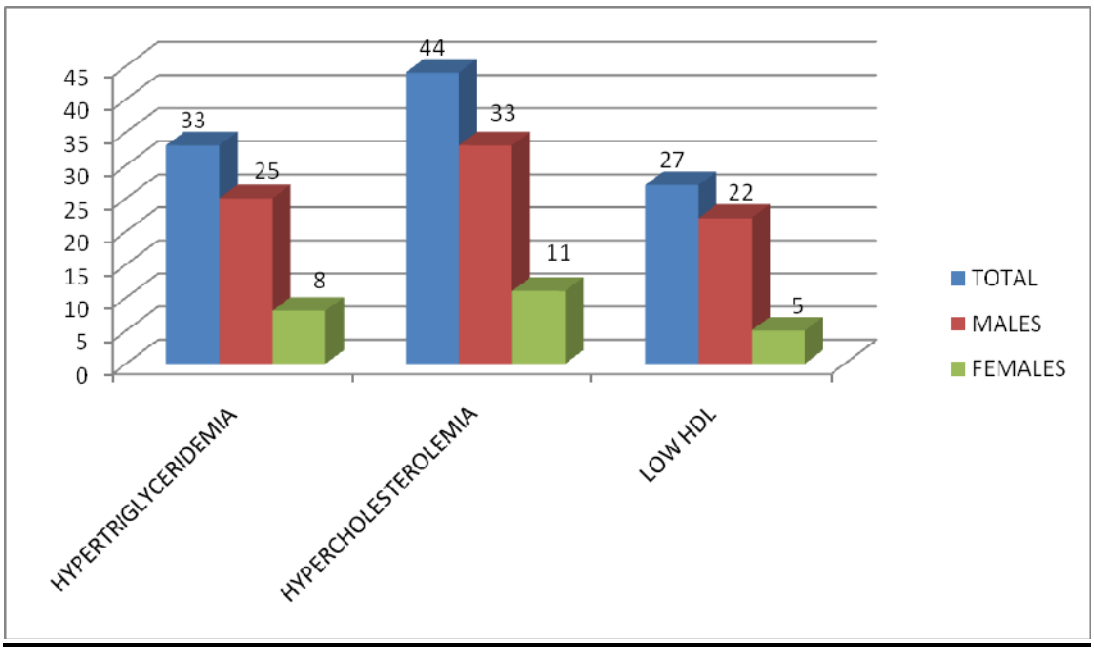


CHART 16

DISCUSSION

DISCUSSION

Myocardial Infarction is more common in men than women. The risk factors, clinical features, course of the illness have extensively been studied. Due to the change in life style and cultural factors the incidence of myocardial infarction in females especially in premenopausal and perimenopausal age groups is on the rise.

In the present series 100 patients with Acute Myocardial Infarction with a window period of less than 12 hours who were admitted to intensive coronary care unit, CMC Hospital, Coimbatore -18, during the period between 1st Jan 2006 to 30th Jun 2007 with typical history and ECG changes showing evidence of Myocardial Infarction were studied.

C-reactive protein is a hepatically derived marker of low grade systemic inflammation that largely reflects circulating cytokine formation. It is produced from liver in response to cytokine stimulation (interleukin 1 and 6). According to AHA/CDC recommendations, High Sensitive -CRP levels are classified as: low High Sensitive -CRP < 1 mg/dl; average 1-3 mg/dl; and high High Sensitive -CRP >3 mg/dl.

Higher High Sensitive - CRP levels in patients with AMI indicate an increased risk of subsequent coronary events because High Sensitive - CRP is associated with inflammation of coronary vessels.

It is reasonable to suggest that high High Sensitive - CRP levels are associated with adverse outcome. Levels of hs-CRP are also elevated among those with acute coronary syndromes at high risk for recurrent events[17,58-60] and among post-MI patients at high risk for recurrent instability.[17,61] These effects are independent of other risk factors and appear to add to the predictive value of lipid screening in terms of risk prediction.[17,58,62] However, there remains much confusion as to when it may be appropriate to measure hs-CRP levels, and what to do about them when they are found to be elevated.

HS-CRP is a non-specific, highly sensitive marker of inflammation. One postulate regarding the unstable character of acute coronary syndrome is inflammation of the atherosclerotic lesion.[6,63] Hence elevated plasma hs-CRP levels in patients with acute coronary syndrome on admission and its persistence after discharge may indicate a state of persistent inflammation and instability with poor short- and long-term prognosis.[8-10,14] This was observed in various studies.[15,64] The association of elevated High Sensitive -CRP and poor prognosis in patients with acute coronary syndrome is independent of other serum markers of myocardial cell injury and necrosis like cardiac Troponins and CK-MB.[14]

Suleiman et al.[65] showed that plasma High Sensitive -CRP level obtained within 12 to 24 hours of symptom onset is an independent marker of 30-day mortality and the development of heart failure in patients with AMI. High Sensitive - CRP levels may be related to inflammatory processes associated

with infarct expansion and post-infarction ventricular remodeling. Anzai et al.[8] showed that cardiac rupture, LV aneurysm formation, and one-year cardiac mortality were associated with an elevation of serum High Sensitive -CRP early after AMI. Berton et al.[10] showed that first-day hs-CRP is a strong independent predictor of both heart failure progression and depressed LV ejection fraction in AMI. In contrast to above studies, Kimura et al.[66] showed that elevated High Sensitive -CRP immediately after onset of AMI is associated with less myocardial damage and better LV function in reperfused anterior AMI. They have suggested that two mechanisms may account for the myocardial protective effect associated with the elevation of High Sensitive -CRP. First, silent myocardial ischemia, which is frequently associated with unstable angina, and in which High Sensitive -CRP levels may increase greatly, and may exert an ischemic preconditioning effect on the myocardium. Second, inflammation induces expression of angiogenic growth factors associated with reduced infarct size, and endogenous production of nitrous oxide protects the myocardium from ischemic reperfusion injury.

In the present study of 100 Patients Of Acute Myocardial Infarction male (82%) had increased incidence than that of female (18%). Of the female, the post-menopausal age group (55.55%) had higher incidence than that of premenopausal (22.22%) and perimenopausal (22.22%) age groups.

Maximum number was in the 51-60 year age group (35%). 41-50 year age group comes next (31%). The incidence was less below 30 years and above

60 years. The youngest patient was 26 year old female and the oldest patient was 70 years female. Among men youngest was 28 years and oldest was 69 years.

Oral contraceptive pills contributed to acute myocardial infarction in 1 patient (1%).

In the present study 65 (79.2%) males out of 82 males were cigarette smokers. There were no smokers in the female group.

In the present study 22 patients (22%) were obese, of which 12(14.64%) were male and 10(55.55%) were female patients. So obesity is a major risk factor in female than in male, probably due to sedentary life style.

In the present study, information regarding the prevalence of diabetes was obtained from the interviews. The patient was considered diabetic when there was a history of polyuria, polyphagia and polydypsia, treatment with insulin or oral hypoglycemic agents. Increased blood sugar was also taken into consideration. ADA 2005 guidelines were followed.

In the present series, 29 (29%) patients were diabetic. 23(79.3%) were male and 6(20.6%) were female among the diabetics. So diabetes has an important role both in male and female.

In the present series blood pressure was routinely checked for minimum three consecutive days and history of hypertension and the drugs they were

taking were checked. Hypertension was noted in 49 (49%) patients, of which 20(42.5%) were newly detected hypertensives. Hypertension was noted in 39 male and 10 female. Of the newly detected 15(75%) were male and 5(25%) were female. So hypertension is an important risk factor in male than in female.

In the present study cholesterol levels >200mg% was considered, hypertriglyceridemia >160mg% and HDL < 40mg% were considered as risk factors and they were analysed in the study group. Hypercholesterolemia was seen in 44(44%) patients. Of these 75% were male and 25% female. 33(33%) were hypertriglyceridemia patients. Of these 25(75.7%) were male and 8(24.24%) were female. Low HDL was seen in 27(27%) patients. Of these 22(81.4%) were male and 5(18.5%) were female. Hypertriglyceridemia was seen in 33(33%).

1) MULTIPLICITY OF RISK FACTORS:

Multiplicity of risk factors leading to Myocardial Infarction are common in men than women.

In the present study, risk stratification was done by the levels of HS-CRP level just before the time of thrombolysis. Of these 23% belonged to the low risk group (<1 mg/dl), 25% belonged to the moderate risk group (1-3 mg/dl) and 52% belonged to the high risk group (>3 mg/dl).

No. of Risk Factors	TOTAL NO: OF PATIENTS	MEN IN Percentage	WOMEN IN Percentage
NIL	NIL	NIL	NIL
1	16	12	4
2	38	30	8
3	27	23	4
4 or More	19	17	2
TOTAL	100	82	18

TABLE 24: RISK FACTORS IN BOTH GROUPS

In the low risk group 86.9% were male and 13% were female .In moderate risk group 76% were male and 24% were female and in high risk group 82% were male and 18% were female.

In the present study, in the low and moderate risk group thrombolysis was done at a lower window period i.e., it was < 6 hrs.In the high risk group thrombolysis was done between 6hrs-12 hrs.

PARTICULARS		S Pandian et al 67		PRESENT		
		LOW RISK	HIGH RISK	LOW RISK	MODERATE RISK	HIGH RISK
MEAN AGE		**50.2±7**	**50.1±8**	46.4±**7.86**	**51±6.56**	**50.94±8.63**
SEX	MALE	36(90)	54(90)	20(82)	19(82)	43(82)
	FEMALE	4(10)	6(10)	3(18)	6(18)	9(18)
MEAN CRP LEVEL		**1.26±0.91**	**6.52±3.97**	0.75±**0.239**	2.02±**0.083**	**6.72±1.76**
EJECTION FRACTION		**56.9±7.7**	**46.7±11.9**	57±**11**	54.12±**10.76**	50±**10.8**
DIABETES		20(50)	30(50)	5(29)	9(29)	15(29)
HYPERTENTION		14(35)	23(37)	7(49)	16(49)	26(49)
SMOKING		18(45)	30(50)	17(65)	14(65)	34(65)

In the present study, the ejection fraction was higher in the low and moderate risk group than that of the high risk group.

In the present study, the reperfusion injury was found to be higher in the high risk group than that of the low and moderate risk group.

SUMMARY

SUMMARY

1) In the estimation of High sensitive- C –reactive protein, the high risk group predominated at 52% with a mean of 6.72±1.76, intermediate risk - 25% with a mean of 2.02±0.083 and low risk - 23% with a mean of 0.75±0.239.
2) In all the groups male patients predominated.
3) In the low and intermediate group the window period was predominantly in the range of 3- 6 hrs, in high risk group it was > 6 hrs.
4) The mean ejection fraction in the low risk was 57±11, intermediate risk was 54.12±10.76 and in high risk group it was 50±10.8.
5) The reperfusion injury was only 4% in the low risk group, 8% in the intermediate risk and it was higher in the high risk group at 42%.
6) The most common site of acute myocardial infarction was extensive anterior wall myocardial infarction followed by inferior wall myocardial infarction.
7) Among women, 55.55% of post menopausal women had acute myocardial infarction.
8) Oral contraceptive pill was a risk factor in 1 patient (5.5%).
9) Smoking was one of the major risk factor in 79.2% of male patients.
10) Obesity was found to be a risk factor in 22% of patients.55.55% among female were obese.
11) Diabetes was found in 29% of patients of which 79.3% were male.
12) Hypertension was seen in 49% patients. Of these 79.59% were male.

CONCLUSION

CONCLUSION

In this Present study it is found that Ejection Fraction is lower in the high CRP group. Reperfusion injury is lower in the low and intermediate risk group according to the High Sensitive C –Reactive Protein assay. As echocardiogram was done only on day three after Acute myocardial infarction, a lot of stunned myocardium may be present, which is the limitation of this study.

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APPENDIX

PROFORMA

RECORD

NAME: **AGE:** **SEX: M/F**

I.P.NO: **DOA:** **DOD:**

DIAGNOSIS:

WINDOW PERIOD:

C/O: ***CHEST PAIN-TYPE, RADIATION***

SWEATING

PALPITATION

SYNCOPE

DYSPNOEA

PND, ORTHOPNOEA

GIDDINESS

NAUSEA & VOMITING

HOPI:

RISK FACTORS: DM/HT/SMOKING/COPD

DIET: ***VEG/NONVEG/MIXED***

FAMILY HISTORY:

IHD/OBESITY/DIABETES/HYPERTENSION/HYPERLIPIDEMIA

TREATMENT HISTORY: ***DRUG INTAKE***

MARITAL HISTORY

GENERAL EXAMINATION:

- ***OBESITY***
- ***DYSPNOEA***
- ***CYANOSIS***
- ***CLUBBING***
- ***PITTING PEDAL EDEMA***
- ***XANTHALESMA***
- ***TENDON XANTHOMAS***
- ***ARCUS SENILIS***
- ***PULSE***
- ***BLOOD PRESSURE***
- ***WAIST CIRCUMFERENCE***

BP:

HEIGHT: **WEIGHT:** **BMI:**

WAIST:HIP RATIO:

ANKLE-BRACHIAL INDEX:

CARDIOVASCULAR SYSTEM

APICAL IMPULSE ***S1*** ***S2*** ***S3***

PERICARDIAL RUB ***PERIPHERAL VASCULAR SYSTEM***

RESPIRATORY SYSYEM

ABDOMEN

CENTRAL NERVOUS SYSTEM

INVESTIGATION

LIPID PROFILE:-

TOTAL CHOLESTEROL: mg%

TGL: mg% HDL: mg% LDL: mg%

WBC COUNT:

HS-CRP: mg/L URIC ACID: mg%

ELECTROCARDIOGRAM:

BEFORE THROMBOLYSIS:

AFTER THROMBOLYSIS:

ECHOCARDIOGRAM:

CHEST X-RAY

URINE ROUTINE

BLOOD SUGAR

BLOOD UREA

SERUM CREATININE

RISK:

FOLLOW UP SHEET

COIMBATORE MEDICAL COLLEGE

&

HOSPITAL

DEPARTMENT OF MEDICINE

NAME:

AGE: **SEX:M/F** **I.P.NO:**

DOA:

DOD:

DIAGNOSIS:

WINDOW PERIOD:

HS-CRP: **EF:**

RISK:

COMPLICATIONS-

SYMPTOMS	SIGNS	DIAGNOSIS	THERAPY

MASTER CHART

MASTER CHART.xls