A STUDY OF

MYOCARDIAL INFARCTION IN WOMEN

M.D. DEGREE EXAMINATION
BRANCH I - GENERAL MEDICINE

COIMBATORE MEDICAL COLLEGE
COIMBATORE

DISSERTATION SUBMITTED TO
THE TAMIL NADU DR. M.G.R
MEDICAL UNIVERSITY

SEPTEMBER 2006

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY OF MYOCARDIAL INFARCTION IN WOMEN"** is a bonafide work done by **Dr.C.J.SELVAKUMAR**. It is a regular, systematic study done under my guidance and supervision during the period Jan 2005 to Dec 2005 and submitted for the ensuing **M.D. Branch I - General Medicine Examinations, September 2006** of The Tamil Nadu DR.M.G.R. Medical University, Chennai.

Place:

Date:

DEAN
COIMBATORE MEDICAL COLLEGE
Coimbatore

Prof. Dr. G.YASODHARA, M.D.
Prof. & HOD of Medicine,
COIMBATORE MEDICAL COLLEGE
Coimbatore

DECLARATION

I solemnly declare that the dissertation titled, **"A STUDY OF MYOCARDIAL INFARCTION IN WOMEN"** was done by me at Coimbatore Medical College and Hospital during the period Jan 2005 to Dec 2005 under the guidance and supervision of **PROF. DR. G. YASODHARA, M.D.**

The dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of **M.D. Degree Branch I - General Medicine.**

PLACE:

DATE:

DR.C.J. SELVAKUMAR

ACKNOWLEDGEMENT

I sincerely thank **Dr.T.P.KALANITI, M.D.,** Dean C.M.C Hospital, Coimbatore for having permitted me to undertake the study in this prestigious institution.

It is great pleasure to express my sincere thanks to **Prof. Dr.G.YASODHARA, M.D.,** Head of the Department of Medicine, C.M.C Hospital, Coimbatore for her able stewardship in the preparation of this work.

I gratefully acknowledge my indebtedness to **Prof. Dr.V.E.DHANDAPANI, M.D., D.M.,** Professor of Cardiology, C.M.C Hospital, Coimbatore for his valuable guidance in the preparation of this dissertation work.

I whole heartedly thank my unit Assistant Professors, **Dr. R.ANANDAN, M.D., Dr.T.GEETHA, M.D.,** and **Dr. S.ARUN, M.D.,** for their professional assistance in shaping out this dissertation work.

I express my sincere gratitude to all the patients for their co-operation during the study.

S.No.	CONTENTS	PAGE No.

INTRODUCTION

Myocardial Infarction is a serious complication of Atherosclerotic Coronary Heart Disease. Coronary Heart Disease was first described by William Heberden 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 prophesised that the hope of salvaging the muscle lay in restoration of blood flow.

Myocardial Infarction[9] 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[14] 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 [55]. 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[10] and age distribution ranges from 40-58 years.

Data from Framingham study says that the incidence of cardiovascular disease is 3 times lower in women than men before menopause and approximately equal in men and women aged 75-79 years. The incidence in women rises after menopause due to hormonal changes, resulting in increase in LDL Cholesterol, enhancement of oxidation of LDL Cholesterol and fall in HDL levels.

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.

AIM OF THE STUDY

1.To analyze various risk factors in women with Myocardial Infarction.

2.To study the incidence, presenting features, site of infarction, and complications of the disease.

3.To study mortality in women with Acute Myocardial Infarction.

REVIEW OF LITERATURE

1. ANATOMY OF CORONARY CIRCULATION

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 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.

B. AETIOPATHOGENESIS OF MYOCARDIAL INFARCTION

1. ATHEROSCLEROSIS[19, 11]

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 fibrous cap.

Atherosclerosis[2, 15] is the single most aetiological 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”[7] 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.

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 [3]

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.

C. RISK FACTORS FOR MYOCARDIAL INFARCTION [1, 16, 17]

The term risk is widely used to describe those characteristically found in healthy individuals that relate to the subsequent appearance of Ischaemic Heart Disease[1]. The risk of Ischaemic Heart Disease is determined by the aggregation of individual factors.

Table1: 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 in whom a first degree relative of the patient has developed Ischaemic Heart Disease before the age of 50 years[8].

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 and may be the first manifestation of the disease. Smoking decreases HDL cholesterol levels. It accelerates atherosclerosis, plaque instability, increases the risk of thrombosis, myocardial infarction and death.

Women cigarette smokers are also at greater risk of developing coronary artery disease than non-smokers. In those who smoke and also use oral contraceptives the risk increases many fold. 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[26]. 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[22] 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[27] 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[8]. 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[5], particularly the male pattern 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 individual 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:

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

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

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 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 fall to normal levels within 48-72 hours [18]. 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:

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:

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.

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:

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 [1]

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 40-6-/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 do 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 20-4- 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 [12] 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 anerysmectomy 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.

G. CORONARY HEART DISEASE IN WOMEN:

Women with heart disease may present differently than men, have unique underlying pathophysiologies and have distinctive risk benefit profiles with commonly accepted therapies. Heart disease is far more age dependant in women than in men. Women with cardiovascular disease are older and have more co-morbidity. This fact, in turn, makes diagnostic and treatment procedures more

problematic in women. In addition, may effective pharmacological strategies are underutilized and there is a lack of gender specific data on numerous therapies. Clinicians have long known of the apparent protection of younger, premenopausal women from ischaemic heart disease and this protection has been attributed to oestrogen. Further nuclear oestrogen receptors are present in cardiac myocyte of males and females, a potential explanation for gender differences in gene regulation. However these physiological differences have failed to translate into a useful pharmacopoeia.

Two genes were relevant in women – Plasminogen activator inhibitor 1 (PAI-1) and Stromelysin-1. Such genetic differences are manifest in the physiology of atherosclerosis, including plaque components (more cellular and fibrous tissue in women), endothelial function (oestrogen induced coronary vasodilation) and hemostasis (higher fibrinogen and factor 7 levels in women). Women are twice more likely to have plaque erosion than men.

Women are older and have a greater burden of risk factors and concomitant disease, they are more frail and less likely to recover fully from cardiovascular events. Women's greater longevity and our society's gender roles combined to make women caregivers to men but often leave the same women without social, emotional or

financial support at the time of their own illness. Women, as well as their caregivers may underestimate the importance of atherosclerotic disease and fail to implement the preventive strategies fully or even recognize and act on symptoms appropriately. These factors have a significant negative impact on optimal care delivery for women.

Coming to the risk factors diabetes is associated with a greater incremental risk in women completely eliminating the female advantage. The American Heart Association awards double weight to diabetes in women when calculating coronary heart disease risk. The population risk attributable to hypertension is higher in women than men because of the increased incidence with age and longevity of women. Cigarettes have an antiestrogenic effect and induce an unfavorable lipid profile, leading women to lose their “natural” protection against atherosclerotic vascular disease. The average lipid profile in women is affected by hormonal status and changes throughout life. Young women have lower LDL and higher HDL than men of same age. As women ages LDL increases, HDL decreases and the risk of coronary heart disease climbs. Triglycerides, Lp (a), Apolipoprotein B-100 and Apolipoprotein (a) is also associated with higher cardiac risk in women. Higher oestrogen, oxytocin levels, lower iron levels have been suggested to

afford protection against coronary heart disease in premenopausal females. Obesity is associated with increased CRP particularly in women. Physical inactivity is more prevalent among women than men.

Women have a different clinical presentation and hospital course following acute coronary syndrome and acute myocardial infarction and respond differently to medical and procedural therapies. Women are likely to be older and are more likely to have a history of systemic hypertension, diabetes mellitus, unstable angina, hyperlipidemia and congestive cardiac failure. Women are also more likely to experience jaw, neck and shoulder pain, abdominal pain, nausea, vomiting, palpitation, fatigue and dyspnoea in addition to chest pain and are less likely to report diaphoresis than men. Although chest pain is the single most common presenting symptom of myocardial infarction in women, women seem more susceptible to silent infarction particularly elderly women. Perhaps in part because of these more atypical symptoms women seek medical attention more slowly and even after hospital arrival may experience greater delays in receiving care. Physicians' uncertainty about the true clinical diagnosis is more common for women than men with acute myocardial infarction and misdiagnosis is more likely.

Women with infarction have higher risk initial presentations, with greater prevalence of tachycardia, rales, heart block of higher Killips class. Women have higher rates of in-hospital complications from infarction including bleeding, stroke, shock, myocardial rupture and recurrent chest pain than do men although most of these differences appear attributable to age and co-morbidities. There is a two fold greater mortality in women younger than 50 years compared with similarly aged men. Mortality 1-3 years after hospital discharge is also increased in younger women, although it is similar in postmenopausal women and men. Women are more likely than men to experience vascular and renal complications form diagnostic angiography.

Eligible women now seem to receive thrombolytic therapy at rates equal to those of men, but women more frequently have contraindications to thrombolysis. Primary angioplasty is more effective in women. Percutaneous revascularization has the similar efficacy as in men but the procedural complications remain higher. The difference in outcome has been variously attributed to women's older age, smaller body size, greater severity of angina, more fragile vessels and greater burden of co-morbidity. In-hospital mortality for coronary artery bypass graft is 1.4 to 4.4 times higher particularly in

women less than 60 years. Women are less likely to receive internal mammary grafts and undergo complete revascularization and are more likely to experience the complications of heart failure, perioperative infarction and hemorrhage. Neurological complications of coronary artery bypass graft, including stroke, transient ischaemic attack and coma are more frequent in women. Rehospitalization rates for women are two times those for men in the first two months after coronary artery bypass grafting. The causes for higher operative mortality and morbidity appear to be multiple including technical factors such as smaller body size and coronary diameter, advanced age and co-morbid factors and clinical factors such as the urgency of the procedure. After coronary artery bypass graft women have a lower likelihood of being free of angina than do men and experience greater physical disability and less return to work..

Improvement in prevention of coronary artery disease in women requires earlier awareness and identification of the risk. Social networks and support influence coronary heart disease outcome both independently and through the likelihood of compliance of therapeutic strategies (e.g. cardiac rehabilitation) and their impact may be greater in women.

MATERIAL AND METHODS

During the period from 1st Jan 2005 to 31st Dec 2005 sixty five female patients were admitted with acute myocardial infarction in coronary care unit, CMC Hospital, Coimbatore. These cases were taken up for study and they were divided into two groups.

GROUP A: Includes Premenopausal women.

GROUP B: Includes Postmenopausal women.

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

1. Typical history of retrosternal chest pain.
2. Serial ECG changes [23, 24, 25]: Appearance of pathological Q wave or appearance of ST elevation with T wave inversion in atleast 2 or more leads.
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 was taken into consideration. History of oral contraceptive intake, tobacco chewing,

cigarette smoking were noted. Family history of IHD, hypertension, diabetes, obesity, and sudden death were also noted.

Menstrual history, obstetric history, year and month of menopause, were recorded. Serum analysis of cholesterol and blood sugar along with enzyme studies were done within 24 hours of admission. Chest X-ray and Echocardiography were taken in all cases.

Serum cholesterol was considered to be increased if it was greater than 200mgm%. Waist circumference more than 88cm was taken as obese. The type and extent of infarction were judged by the Q wave and ST elevation in serial ECGs.

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.

OBSERVATIONS

A. RISK FACTORS

1. MENSTRUAL HISTORY:

In the present study 14 (21.5%) women were menstruating (Group A). 51 (78.5%) women had attained menopause (Group B). The incidence of myocardial infarction was higher in the postmenopausal women.

2. AGE:

Age is a definite risk factor. Total number of patients in this series was 65. Maximum number was in the 55-65 year age group (35.4%). 46-55 year age group comes next (23%). The incidence was less below 35 years and above 75 years. The youngest patient was 35 year and the oldest patient was 79 years.

Table2: AGE DISTRIBUTION OF PATIENTS

S.No	AGE GROUP	No. OF PATIENTS	PERCENTAGE
1	26-35	2	3
2	36-45	12	18.4
3	46-55	15	23
4	56-65	23	35.4
5	66-75	10	15.4
6	ABOVE 75	3	4.6
	TOTAL	65	100

3. FAMILY HISTORY:

Coronary artery disease was often found in several members of the same family. In this study, family history of diabetes was noted in 24 (36.9%) patients, hypertension in 28 (43%) patients, obesity in 14 (21.5%) patients, and myocardial infarction in 15 (23%) patients.

4. ORAL CONTRACEPTIVE PILLS:

In the present study 5 (7.7%) were taking oral contraceptive pills for more than 3 months. All of them were premenopausal women.

5. CIGARETTE SMOKING:

In the present study only 3 (4.6%) were cigar smokers and all were postmenopausal. There was no smoker in the premenopausal group.

6. TOBACCO CHEWING:

In the present study 15 (23%) patients were taking tobacco for a long period (more than 3 months).

Only 1 (7.1%) of the 14 premenopausal women took tobacco. 14 (27.4%) of the 51 postmenopausal women took tobacco.

7. ABDOMINAL OBESITY:

In this study the waist circumference was recorded for all patients. Waist circumference more than 88cm (35in) was taken as abdominal obesity.

In the present study 20 (30.8%) patients were obese. Out of the 14 premenopausal women 9 (64.3%) were obese. 11 (21.5%) out of the 51 postmenopausal women were obese. So obesity is a major risk factor in the premenopausal group.

8. 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 27 (41.5%) patients were Diabetic. 6 (42.9%) out of 14 premenopausal women were Diabetic. 21 (41.2%) out of 51 postmenopausal women were diabetic. So diabetes had an important role both in the premenopausal and postmenopausal groups.

9. HYPERTENSION:

In the present series blood pressure was routinely checked for minimum three consecutive days. Hypertension was noted in 26 (40%) patients. Hypertension was noted in 6 (42.9%) out of 14 premenopausal women. 20 (39.2%) out of 51 postmenopausal women had hypertension. So hypertension is an important risk factor in both groups.

10. SERUM CHOLESTEROL:

In the present study cholesterol levels were raised in 25 (38.5%) patients. Hypercholesterolemia was seen in 7 (50%) out of 14 premenopausal patients, while it was present in 18 (35.3%) patients out of 51 postmenopausal female patients. Hypercholesterolemia was present in both groups particularly more in the premenopausal women.

11. MULTIPLICITY OF RISK FACTORS:

Multiplicity of risk factors leading to angina pectoris and myocardial infarction are common in women. In the present study multiple risk factors were seen in all the members in the premenopausal group. 14 (27.5%) out of 51 postmenopausal women had no risk factors.

Table3: RISK FACTORS IN BOTH GROUPS

No. of Risk Factors	Premenopausal women		Postmenopausal women	
	No. of Patients	%	No. of Patients	%
NIL	NIL	NIL	14	27.5
1	NIL	NIL	1	2
2	5	35.7	10	19.6
3	3	21.4	10	19.6
4 or More	6	42.9	16	31.4
	14		51	

B.SYMPTOMATOLOGY

1. PRESENTING FEATURES

Table4: PRESENTING FEATURES IN BOTH GROUPS

S.NO	PRESENTING FEATURES	NO.OF PATIENTS
1	RETROSTERNAL PAIN	60
2	RADIATION	30
3	SWEATING	40
4	NAUSEA &VOMITING	19
5	DYSPNOEA	36
6	BASAL CREPITATIONS	22
7	GALLOP RYTHM	15
8	ELEVATED JVP	07
9	GIDDINESS	20

It has been stated that myocardial infarction is often atypical in women (Jouve.A.et al). But in the present study retrosternal pain was present in most cases. Characteristic features of myocardial infarction like sweating, radiation of pain, nausea, vomiting, and giddiness were present in a large number of cases. There was no difference in symptomatology between premenopausal and post menopausal women.

C. INVESTIGATIONS

1. ENZYME CHANGES:

CPK-MB levels were done for all the 65 cases .For diagnosis of Acute Myocardial Infarction CPK-MB values should be more than 10IU/L.

Table5: ENZYME STUDY WITHIN 24 HOURS OF ADMISSION

ENZYME	NO.OF CASES	ABNORMAL RISE	PERCENTAGE
CPK-MB	65	49	75.4

2. X-RAY FINDINGS:

Chest x-ray was taken in all the 65 cases. The following findings were noted.

Table6: CHEST X-RAY FINDINGS

S.NO	ABNORMALITY	No. OF CASES
1	Pulmonary venous congestion	22
2	Cardiomegaly	20
3	Calcified aortic knuckle	5

3. ECHOCARDIOGRAPHY:

2D and Doppler Echocardiography was done for all 65 cases. All cases showed regional wall motion abnormality. Left ventricular dysfunction was noted in 22 cases out of which 4 were severe. 2 cases showed Mitral Regurgitation. Left ventricular hypertrophy was noted in 20 cases.

4. SITE OF INFARCTION:

The most common site of infarction in the present study was in the anteroseptal region. Second common site of infarction was in the inferior wall.

Table7: SITE OF INFARCTION IN BOTH GROUPS

S.NO	SITE OF INFARCTION	NO.OF CASES	PERCENTAGE
1	ANTERO SEPTAL	31	47.7
2	ANTERO LATERAL	6	9.2
3	EXTENSIVE ANTERIOR	7	10.8
4	INFERIOR WALL WITHOUT RVMI	11	16.9
5	INFERIOR WALL WITH RVMI	7	10.8
6	INFERO LATERAL	3	4.6
7	TRUE POSTERIOR	NIL	NIL
	TOTAL	65	100

D. COMPLICATIONS:

- Arrhythmia- 19 cases

  a) Ventricular premature beats- 7

  b) Ventricular tachycardia- 6

  c) Ventricular fibrillation- 2

  d) Atrial ectopics- 4

- Cardiogenic shock- 5 cases
- Acute left ventricular failure- 4 cases
- Cardiac arrest- 1 case
- First degree heart block-5 cases
- Papillary muscle dysfunction- 2 cases

E. MORTALITY:

Robinson. K. and colleagues noticed that women with acute myocardial infarction showed a higher rate of complications and mortality than men

In the present study also it was found that mortality was seen in 5 (7.69%) out of 65 patients. 1 (7.14%) out of 14 premenopausal women and 4 (7.84%) out of 51 postmenopausal women died. Mortality was higher in 56 – 65 year age group.

Table8: MORTALITY IN BOTH GROUPS

S.NO	AGE GROUP IN YEARS	NO. OF PATIENTS	%
1	26 - 35	NIL	NIL
2	36 – 45	1	7.14 #
3	46 – 55	1	1.96 $
4	56 – 65	2	3.92 $
5	66 – 75	1	1.96 $
6	ABOVE 75	NIL	NIL

- Percentage calculated for 14 patients in premenopausal group

$- Percentage calculated for 51 patients in postmenopausal group

Table9: CAUSE OF DEATH IN BOTH GROUPS

S.No	CAUSE OF DEATH	NO OF PREMENOPAUSAL WOMEN	NO OF POSTMENOPAUSAL WOMEN
1	LVF	1	1
2	VENTRICULAR FIBRILLATION	NIL	1
3	CARDIOGENIC SHOCK	NIL	2

DISCUSSION

Myocardial Infarction in women is much less when compared to men. So the risk factors, clinical features, course of the illness have not been studied extensively but due to the change in life style and cultural factors the incidence of myocardial infarction in females is on the rise.

In the present series 65 women with Myocardial Infarction who were admitted in intensive coronary care unit, CMC Hospital, Coimbatore -18, during the period between 1st Jan 2005 to 31st Dec 2005 with typical history, enzyme and ECG changes showing evidence of Myocardial Infarction were included.

The patients were divided into two groups as Premenopausal (Group A) and Postmenopausal (Group B). There were 14 Premenopausal and 51 Postmenopausal women in this study.

1) AGE:

In the present study 35.4% were between 55 – 65 years. The incidence above 75 years was 4.6% and below 45 years was 21.4%.

Khyati et al (IHJ 2004) [28] who studied the risk factors in women with coronary artery disease noted that the mean age was 53.8 ± 6.9 years.

Tumulu B Narayan et al (IHJ 2004) [29] in his study noted that the mean age was 55 ± 14 years.

Jaume Marrugat et al (JAMA 1998) [42] in their study noticed that the mean age in women was 68.6 years.

Age is a definite risk factor and the incidence increases after the age of 50 years.

2. FAMILY HISTORY:

In the present study positive family history of Hypertension was noted in 43%, Obesity in 21.5%, Diabetes in 36.9%, and Myocardial Infarction in 23% patients.

Burke.G.C.et al (Circulation 1991) [30] noticed that positive family history of Hypertension was present in 56%, Obesity in 44%, Diabetes in 17% and Myocardial Infarction in 13% women.

So family history is a definite risk factor in both Western and Our studies. Family history of hypertension scores over others in the development of coronary artery disease.

3. ORAL CONTRACEPTIVE PILLS:

In the present study 7.69% of the women were taking oral contraceptive pills for more than 3 months. The details of the dose could not be elicited. All of them were premenopausal (GroupA).

Nicholas Dunn et al (BMJ 1999) [31] in his study noticed that there was no significant increased risk of myocardial infarction in users of oral contraceptives.

Bea C.Tanis et al (NEJM 2001) [32] noticed that the risk of myocardial infarction among users of any type of oral contraceptives was twice that of non users.

Jordanka et al (Int.J.Epi 1989) [34] in their study noticed that there is 2.48 times increased risk of myocardial infarction among the users of Oral Contraceptives.

Oomman et al (JAPI 2003) [33] noticed that out of 660 females in his study 71% patients used oral contraceptives.

In this study the incidence is very low due to the low use of oral contraceptive pills among Indian women.

4. SMOKING:

In the present study only 4.6% were cigar smokers. All belonged to the postmenopausal group .

Croft.P.et al (BMJ 1989) [35] observed that among smoking women the risk of Myocardial Infarction is 1.7 times more in light smokers and 4.3 times more in heavy smokers, compared to nonsmokers.

N.R.Dunn et al (Heart 1999) [36] noticed a 2.47 times increased risk of Myocardial Infarction in smokers of 1-5 cigarettes/day to 74.6 times increased risk in smokers who smoke 40 cigarettes/day. It is estimated that if all women aged 16-44 yrs were able to stop smoking 400 cases of Myocardial Infarction per annum would be prevented.

Leroy Nickles et al (Ac. Em. Med 2004) [37] who conducted the study in young women less than 50 yrs noticed that 77% were smokers.

In the Indian study by Prabakaran.D.et al (IHJ 1991) [41] mentioned that among women with smoking habit the risk of myocardial infarction was only 4.3%.

Smoking in Indian women is less because of cultural reasons. So the incidence is low compared with Western study.

5. TOBACCO CHEWING:

In this present study 23% were tobacco chewers. The incidence was 7.1% among premenopausal and 27.4% among the postmenopausal group.

Bolinder et al (Euro. Heart Journal 1992) [38] noted increased risk of death from coronary heart disease due to use of smokeless tobacco.

Tobacco chewing is common among South Indians. It is a definite risk factor for coronary artery disease.

6. ABDOMINAL OBESITY:

In the present study 30.8% women had abdominal obesity. 64.3% of the premenopausal group and 21.5% of the postmenopausal group had abdominal obesity.

Fraser B.E. et al (Circulation 1992) [39] pointed out that obesity is more closely associated with myocardial infarction in 43% of female patients.

Kathryn M. Rexrode et al (JAMA1998)[40] noted that a waist circumference of 96.5cm or more was associated with a relative risk of 3.06. He concluded that waist circumference was independently associated with risk of coronary heart disease in women.

Dave.T.H. et al (IHJ1991) [41] noted that the relationship between Myocardial Infarction and Obesity is 58.3% in Indian women. Oomman et al (JAPI 2003) [33] in his study done on 660 women noted that 41% was obese.

Comparing the Western and Indian studies with the present study it shows that abdominal obesity is a definite risk factor. In the present study abdominal obesity is particularly a definite risk factor in Premenopausal women.

7. DIABETES MELLITUS:

In this series 41.5% were Diabetic. The incidence was 42.9% among the premenopausal (GroupA) and 41.2% among the postmenopausal women.

Jaume Marrugat et al (JAMA 1998) [42] noticed in their study that 52.9% women with myocardial infarction had diabetes.

Leroy Nickles et al (Ac.Em.Med 2004) [37] in their study conducted in young women less than 50 yrs noticed that 26.8% had Diabetes.

Oomman et al (JAPI 2003) [33] noted diabetes mellitus in 52% of the cases and it was a predominant risk factor in postmenopausal women.

A Chennai based study by Khyati et al (IHJ 2004) [28] conducted on 50 women with coronary heart disease revealed that 29 women were diabetic.

Shankar Krishnaswami [21] in the community based study sponsored by ICMR using 1995 urban and 3686 rural individuals around Vellore area noticed that diabetes was the risk factor in 7.3% urban females and 1.3% rural females.

Bobby John et al (IHJ 2004) [44] conducted a study on 324 women with coronary heart disease and found that 140 (43.2%) patients had diabetes mellitus.

Comparing the result of Western studies, Indian study and the present study diabetes mellitus is a definite risk factor in both the groups.

8. HYPERTENSION:

In the present study hypertension was noted in 40% women. The incidence was 42.9% among premenopausal women and 39.2% among postmenopausal women.

Jaume Marrugat et al (JAMA 1998) [42] noted in their study that 63.9% of women who had myocardial infarction were hypertensive.

Croft.P.et al (BMJ 1989) [35] noted that among women with hypertension, the risk of myocardial infarction is 2.4 times more than those without risk factors.

In a study conducted by Oommam et al (JAPI 2003) [33] in 660 females 60% had hypertension which was a predominant risk factor in postmenopausal women.

Alkesh Jain et al (IHJ 2004) [43] who conducted a risk factor profile study in Indian females noted that 38% women had hypertension.

Bobby John et al (IHJ 2004) [44] who conducted a study on 324 women with coronary heart disease noticed that 201 (62%) women had hypertension.

Comparing the result of Western and Indian studies with the present study, hypertension is a definite risk factor in women with coronary artery disease.

9. HYPERCHOLESTEROLEMIA:

In this present series 38.5% patients had serum cholesterol values more than 200mgm%. The incidence among Premenopausal women was 50% and it was 35.3% among the Postmenopausal women.

Assmann.G.et al (AHJ 1988) [45] conducted a study among 1333 women and observed that 43.27% had hypercholesterolemia. They were followed up and out of which 74% developed Myocardial Infarction later.

Miller.M.et al (Circulation 1992) [46] conducted a study among 24 women for a period of 13 years and noticed that 45% of them who developed Myocardial Infarction had hypercholesterolemia.

Bobby John et al (IHJ 2004) [44] conducted a study on coronary heart disease in Indian women over a 2 year period in 324 women and noted that 130 women (40.1%) had total cholesterol more than 200mgm%.

Khyati et al (IHJ 2004) [28] noted in their study that 24 women out of 50 women with coronary heart disease were hypercholesterolemic.

10. MULTIPLE RISK FACTORS:

In this study, the analysis of multiple risk factors showed that 2 or more were present in 100% of Premenopausal and 70.6% of Postmenopausal women. 3 or more risk factors were present in 64.3% of Premenopausal and 51% of Postmenopausal women. 4 or

more risk factors were noted in 42.9% Premenopausal and 31.4% of Postmenopausal women.

Croft.P.et al (BMJ 1989) [35] noted that relative risk of smoking in Myocardial Infarction was 1.7 times for light smokers and 4.3 times for heavy smokers and in combined smoking and oral contraceptive use the relative risk is 20.8 times and smoking with toxemia of pregnancy the relative risk of Myocardial Infarction was 41 times.

Chasan Taber et al (NEJM 2001) [47] noted that heavy smokers who used oral contraceptive pills had risks that were atleast 30 times as high as those of women with neither risk factor.

N.R.Dunn et al (Heart 1999) [36] noticed that there was an additive risk of Myocardial Infarction in smokers with other clinical risk factors such as Hypertension and Diabetes.

Leroy Nickles et al (Ac.Em.Med 2004) [37] noticed that there was atleast one risk factor in all Acute Myocardial Infarction cases of the Premenopausal group and 87% had 2 or more risk factors.

Tumulu B. Narayan et al (IHJ 2004) [29] noted in their study on 318 women that 81 women had Diabetes, 174 women had Hypertension, 99 women had Dyslipidemia, and 51 women had a

previous history of Myocardial Infarction. They noted that clustering of risk factors occur commonly in women.

11. CLINICAL FEATURES:

In our present study majority (92.3%) of the women presented with typical retrosternal chest pain. 30 patients had a typical radiation of chest pain. 40 patients had sweating.

Jean.C.McSweeney et al (Circulation 2003) [48] in their study noted that only 29.7% of women had typical chest discomfort. The most frequent acute symptoms were shortness of breath (57.9%), weakness (54.8%) and fatigue (42.9%). Chest pain was absent in 43% women.

Robinson.K et al (J.Am.Coll.Cardio 1988) [49] did not agree with this view and found that myocardial infarction was typical in women also.

Bobby John et al (IHJ 2004) [44] in their study on 324 women noted typical chest pain in 139 (42.9%) women, atypical chest pain in 154 (47.5%) women, and no chest pain in 31 (9.6%) women.

12. INVESTIGATIONS:

Creatine phosphokinase-MB was raised in 75.4% of the

patients which was done within 12 hours of admission. The rise was significant.

Anteroseptal myocardial infarction present in 47.7% cases was the most common site. Inferior wall myocardial infarction present in 27.7% cases was the second common site.

X-ray chest was taken for all cases. 20 cases had cardiomegaly. 22 cases had pulmonary venous congestion and 5 had aortic knuckle calcification.

13. COMPLICATIONS:

In our study 55.4% women had complications. In the present series 19 cases had arrhythmias which were the most common complication. Most common arrhythmia was ventricular premature contractions. Next was ventricular tachycardia. Other complications were cardiogenic shock, heart block and left ventricular failure.

Robinson.K.et al (J.Am.Coll.Cardio 1988) [49] noted higher rate of complication in women with acute myocardial infarction.

Weaver et al (JAMA 1996) [50] found in his study done on 10315 women that the nonfatal complications were more in women compared to men. The complications were shock (9%), congestive

cardiac failure (22%), serious bleeding (15%), and reinfarction (5.1%).

14. MORTALITY:

Total mortality in the present study was 7.14% in Premenopausal and 7.84% in postmenopausal women.

Jaume Marrugat et al (JAMA 1998) [42] noticed that the 28 day mortality rate was significantly higher among women (18.5%) compared to men (8.3%). Even the 6 month mortality rate was higher (25.8%) in women compared to men in whom it was 10.8%.

Viola Vaccarino et al (NEJM 1999) [51] noticed that among patients less than 50 years of age the mortality rate for the women was more than twice that for the men. The difference in the rates decreased with increasing age and was no longer significant after the age of 74. Logistic regression analysis showed that the chance of death was 11.1% greater for women than for men with 5year decrease in age.

K.H.Mak et al (HEART 2004) [52] noticed in their study done on 3497 women that the 28 day case fatality rate was greater in women (51.5%) with a larger sex difference evident among younger Malay patients. Case fatality rate was higher among women with

adjusted hazard ratios of 1.64 and 1.50 for 28 day and mean 4year follow up periods.

Cheng I Cheng et al (CHEST 2004) [53] in their study noticed that 30 day mortality in women was 14.6% which was higher when compared to men in whom it was 7.4%.

Mady Moriel et al (Arch. Int. Med 2005) [54] in their study done on elderly women noted that the mortality rates were 12% at 7 days which was higher than men.

CONCLUSION

1. The incidence of Myocardial Infarction is less common in women particularly in Premenopausal women and this may be due to protection offered by female sex hormones to Atherosclerosis.
2. Risk factors like Diabetes Mellitus, Hypertension, Obesity, Hypercholesterolemia, and Positive family history played an important role in the causation of Acute Myocardial Infarction in women particularly in Premenopausal women.
3. The lower incidence of Acute Myocardial Infarction in Indian women particularly in Premenopausal group can be attributed to the rarity of smoking and less frequent use of Oral Contraceptive Pills.
4. Majority of women presented with typical Retrosternal chest pain and sweating as in the case of men. There was no major difference in the presenting features between the Premenopausal (group A) and Postmenopausal (group B) women.
5. The most common site of infarction was in the Anteroseptal region which was present in 47.7% women.

6. Complications were higher (55.4%) in women. The most common complication of Myocardial Infarction in women was arrhythmias which was present in 19 cases. Ventricular arrhythmias were the commonest among them which was present in 15 cases.
7. Mortality rates were higher in women. Total mortality in the present study was 7.14% in Premenopausal and 7.84% in Postmenopausal women.

SUMMARY

Coronary artery disease has been frequently viewed as a disease only of men. Carefully designed clinical trials in women with coronary artery disease have begun only since 1980. Now coronary artery disease has begun to be recognized as a leading cause of death in women. Risk factor of the disease and medical interventions have been studied thoroughly only in men. In women, use of oral contraceptive pills, hormone replacement therapy, menopause, cultural and social roles, family pressure seem to act synergistically with the known coronary artery disease risk factors and also contribute to the sex differences of the disease.

Age at presentation, less specific or subtle clinical manifestations, hence not reaching the tertiary care centre at the appropriate time, greater difficulties and bias in the clinical diagnosis, higher risks involved in angiography, angioplasty and coronary artery bypass graft techniques, higher incidence of cardiac morbidity and mortality are the major sex differences studied and analyzed.

Once coronary artery disease develops women have no survival advantage over men, whatever be the age group.

Ventricular septal rupture, free wall rupture, congestive cardiac failure, recurrent myocardial infarction, bleeding with thrombolytic therapy is some of the common problems faced in women.

Late reference, smaller body size, fragile coronary blood vessels are some of the reasons attributed to high mortality and morbidity.

In the present study conducted at CMC hospital from 1st Jan to 31st Dec 2005 it was found out that the Myocardial Infarction was higher in Postmenopausal women than Premenopausal women. The multiplicity of risk factors was higher in the Premenopausal women.

Typical risk factors for male Myocardial Infarction like Hypertension, Diabetes Mellitus, Obesity, Hypercholesterolemia, Positive family history also prevailed in the females. Complication and mortality were on the higher side in the case of females.

Hence, coronary artery disease needs to be addressed as a special and specific issue in women. There is great scope for further studies regarding therapeutic interventions in women.

BIBLIOGRAPHY

1. N.A.Boon, K.A.A.Fox: Cardiovascular Disease in ***Davidson's Principles and Practices of Medicine*** 19th Ed, Chap 12, Pgs 422-423,440-441.
2. Peter Libby: Prevention and Treatment of Atherosclerosis in ***Harrison's Principles of Internal Medicine*** 16th Ed, Vol 2, Chap 225, Pgs 1430-1433.
3. Bruce F Waller: Nonatherosclerotic Coronary Heart Disease in ***Hurst's The Heart*** 11th Ed, Vol 1, Part 6, Chap 47, Pg 1174.
4. Elliott M.Antman et al: ST Elevation Myocardial Infarction: Pathology, Pathophysiology and Clinical Features in ***Braunwald's Heart Disease – A Textbook of Cardiovascular Medicine.*** Douglas P.Zipes et al (Eds), 7th Ed, Part VI, Chap 46, Pgs 1141-1164.
5. Jeffrey S Flier, Eleftheria Maratos-Flier: Obesity in ***Harrison's Principles of Internal Medicine*** 16th Ed, Vol 1, Chap 64, Pgs 422-429.
6. K.Sethi: Ischaemic Heart Disease in ***API Text Book of Medicine*** 7th Ed, Part 10, Chap 16, Pgs 432-434.

7. Cotran et al: Blood Vessels in ***Robbins Pathologic Basis of Disease*** 5th Ed, Chap 12, Pgs 565-567.
8. A.J.Camm: Cardiovascular Medicine in ***Kumar and Clark's Clinical Medicine*** 5th Ed, Chap 13, Pgs 767-768.
9. A.R.Ness and G.Davey Smith: The Epidemiology of Ischaemic Heart Disease in ***Oxford Textbook of Medicine***, David A.Warrell et al, 4th Ed, Vol 2, Chap 15.4.1.2, Pgs 912-919.
10. Rogerio A Lobo: Menopause in ***Cecil Textbook of Medicine*** 22nd Ed, Vol 2, Chap 256, Pgs 1533-1534.
11. Robert S.Schwartz et al: Pathogenesis of Atherosclerosis in ***Mayo Clinic Cardiology Review***, Joseph G.Murphy (Ed), 2nd Ed, Chap 9, Pgs 113-126.
12. Keith A.A.Fox: Management of Acute Coronary Syndrome in ***Oxford Textbook of Medicine***, David A.Warrell et al (eds), 4th Ed, Vol 2, Chap 15.4.2.3, Pgs 939-941.
13. Bogan Cercek and Prediman K.Shah: Complication of Acute Myocardial Infarction in ***Cardiology, Micheal H.Crawford*** (Ed) 2nd Ed, Section 2, Chap 16, Pgs 371-385.
14. Curtis M.Rimmerman: Acute Myocardial Infarction in the ***Cleveland Clinic Intensive Review of Internal Medicine, James K.Stoller*** et al (Eds) Chap 67, Pgs 732-737.

15. Russel Ross: The Biology of Atherosclerosis in ***Comprehensive Cardiovascular Medicine, Eric J.Topol*** (Ed), Vol 1, Section 1, Chap 1, Pgs 14-23.
16. Srikanth Ramachandruni et al: Management of Angina Pectoris in ***Conn's Current Therapy 2005***, Robert E.Rakel, Section 5, Pgs 329-330.
17. Chuichi Kawai and Yasuyuki Nakamura: Coronary Artery Disease-***Natural History in Cardiovascular Medicine, James T.Willerson*** et al (Eds), 2nd Ed, Chap 33, Pgs 691-705.
18. Allan S.Jaffe et al: Diagnosis of Acute Coronary Syndromes including Acute Myocardial Infarction in ***Cardiology, Micheal H.Crawford*** (Ed) 2nd Ed, Section 2, Chap 12, Pgs 320.
19. Peter Libby et al: The Vascular Biology of Atherosclerosis in ***Braunwald's Heart Disease – A Textbook of Cardiovascular Medicine***. Douglas P.Zipes et al (Eds), 7th Ed, Part V, Chap 35, Pgs 921-935.
20. David Tschopp and Sorin J.Brener: Complication of Acute Myocardial Infarction in ***The Cleveland Clinic Review***, Jan 19, 2005.

21. Shankar Krishnaswami: Conventional Risk Factors for Coronary Artery Disease in Indian Patients in ***Coronary Artery Disease in Indians- A Global Perspective***, Pgs 73-82.

22. Eric J.Topol et al: ***Diabetes in Acute Coronary Syndrome***, 3rd Ed, Chap 9, Pgs 549-570.

23. ***Chou: Electrocardiography*** in Clinical Practice 4th Ed, Chap 9, Pgs 121-141.

24. Galen S.Wagner: ***Marriott's Practical Electrocardiography***, 10th Ed, Chap 7-10, Pgs 140-198.

25. ***Colin Schamroth: An Introduction to ECG*** 7th Ed, Section 2, Chap 11, Pgs 131-156.

26. Lori Mosca et al: Cardiovascular Disease in Women. ***Circulation*** 1997 96:2468-2482.

27. DECODE Study Group. European Diabetes Epidemiology Group. Glucose Tolerance and Mortality. Comparison of WHO and ADA Criteria. ***Lancet*** 1999; 354:617-621.

28. Khyati et al: Women with Coronary Artery Disease – Risk Factors and Angiographic Profile. ***Indian Heart Journal*** Sep-Oct 2004; 56 :(5).

29. Tumulu B.Narayan et al: Risk Factor and Angiographic Profile in Women with Coronary Artery Disease. ***Indian Heart Journal*** Sep-Oct 2004; 56 :(5) Article No.59.

30. Burker G.L. et al: Relation of Risk Factor Levels in Young Adulthood to Parental History of Disease. ***Circulation*** 1991; 84(3):1176-1187.

31. Nicholas Dunn et al: Oral Contraceptives and Myocardial Infarction results of the MICA Case - Control Study. ***BMJ*** 1999; 318:1579-1584.

32. Bea C.Tanis et al: Oral Contraceptives and the risk of Myocardial Infarction. ***NEJM*** 2001; 345:25:1787-1793.

33. Oomman. A et al: Profile of Female Patients undergoing Coronary Angiogram at Tertiary Centre. ***JAPI*** 2003; 51:16-19.

34. Jordanka Ananijevic – Pandey et al: Myocardial Infarction in Young Women with reference to Oral Contraceptive use. ***International Journal of Epidemiology*** 1989; 18:3:585-588.

35. Croft .P et al: Risk Factors for Acute Myocardial Infarction in Women. Evidence from Royal College of General Practitioners. ***BMJ*** 1989; 298(6667):165.

36. Dunn N.R et al: Risk of Myocardial Infarction in Young Female Smokers. ***Heart*** 1999; 82:581-583.

37. Leroy Nickles et al: Risk Factors for Acute Myocardial Infarction in Young Women. ***Acad Emerg Med*** 2004; 11:5:508.

38. Bolinder et al: Increased Risk of Death from Coronary Heart Disease due to the use of Smokeless Tobacco. ***European Heart Journal*** 1992; 13(supp):393.

39. Fraser B.E et al: Effect of traditional risk factor on rates of incident Coronary Events in low risk population. ***Circulation*** 1992; 86(2):406-413.

40. Kathryn M.Rexrode et al: Abdominal Adiposity and Coronary Heart Disease in Women. ***JAMA*** 1998; 280:1843-1843.

41. Dave T.H, Prabakaran.D.et D et al: Profile of Coronary Heart Disease in Indian Women; Correlation of Clinical, Non-Invasive and Coronary Angiographic Findings. ***Indian Heart Journal*** Jan-Feb1991; 43(1):25-29.

42. Jaume Marrugat et al: Mortality differences between Men and Women following first Myocardial Infarction. ***JAMA*** 1998; 280:16:1405-1409.

43. Alkesh Jain et al: Risk Factor Profile in Indian Females with Angiographically proven Coronary Heart Disease. ***Indian Heart Journal*** 0ct 2004; 56(5): Article No.101.

44. Bobby John et al: Profile of Coronary Artery Disease in Indian Women. ***Indian Heart Journal*** Sep-Oct 2004; 56(5): Article No.82.

45. Assmann G et al: The Prospective Cardiovascular Monster Study. Prevalence of Hyperlipidemia in person with Hypertension and/or Diabetes Mellitus and relationship to Coronary Artery Disease. ***Am Heart Journal*** 1988; 116:1713-1724.

46. Miller M et al: Long Term Predictors of subsequent Cardiovascular Events with Coronary Heart Disease and desirable level of Plasma Cholesterol. ***Circulation*** 1992; 86(4):1165-1170.

47. Chasan-Taber L et al: Oral Contraceptives and Myocardial Infarction-The Search for the Smoking Gun. ***NEJM*** 2001; 345:25:1841-1842.

48. Jean C.Mc Sweeney et al: Women's Early Warning Symptoms of Acute Myocardial Infarction. ***Circulation*** 2003; 108:2619.

49. Robinson K et al: Risk Factors and In-Hospital Mortality of first episode of Myocardial Infarction or Acute Coronary Insufficiency in Women. ***J.Am.Coll.Cardio*** 1988; 5:923-926.

50. Weaver W.D et al: Comparison of characteristics and outcomes among Women and Men with Acute Myocardial Infarction treated with Thrombolytic Therapy. GUSTO I Investigators. ***JAMA*** 1996; 275:10.

51. Viola Vaccarino et al: Sex Based Differences in Early Mortality after Myocardial Infarction. ***NEJM*** 1999; 341:4:217-225.

52. Mak K.H et al: Ethnic Variations in Female Vulnerability after an Acute Coronary Event. ***Heart*** 2004; 90:621-626.

53. Cheng I.Cheng et al: Comparison of baseline characteristics, clinical features, angiographic results and early outcomes of Men Vs Women with Acute Myocardial Infarction undergoing PCI. ***Chest*** 2004; 126:47-53.

54. Mady Moriel et al: Management and Outcomes of Elderly Women and Men with Acute Coronary Syndrome in 2000 and 2002. ***Archives of Internal Medicine*** 2005; 165:13:1521-1526.

55. Nanette K.Wenger et al: Coronary Heart Disease – an older women's major health risk. ***BMJ*** 1997; 315:1085-1090.

MASTER CHART

S.NO	NAME	AGE In Yrs	1P.No.	Men. Status	F.H.	OCP Use	Smoking	Tobacco Chewing	Abdl Obesity	D.M	H	Hyper-Cholesterolemia	CPK-MB	CXR	Infarct Site	Complication	Mortality
1	ALAGAMMAL	53	30407	PostM	H						P	145	↑	C.Mly	AS		
2	AMANIAMMAL	64	31890	PostM	DM			YES		P		166	↑		AS		
3	AMBUJAM	54	35890	PostM	MI							256	↑	PVC	EA	VE	
4	ANTHONIAMMAL	68	36765	PostM	DM/H					P	P	154	↑	C.Mly	AS	AE	
5	ARTHI	43	46075	PreM	H						P	156	↑	PVC/C.Mly	IL		
6	ARUKANI	62	48976	PostM	DM/H			YES		P	P	249	↑	PVC/C.Mly	EA	VT	
7	BANNARI	63	32167	PostM	DM/MI					P		276		PVC	EA	IstHB	
8	CHENTHAMARAI	55	34217	PostM	H						P	157	↑	PVC/C.Mly	AS		
9	CHINNAKANNU	65	35786	PostM								142			AL	AE	
10	CHINNAMMAL	68	42876	PostM	O			YES			P	145	↑	C.Mly	AS		
11	CHINTHAMANI	55	45678	PostM	MI/O				P	P		252	↑	PVC	AS		
12	CHITRAKALA	72	47098	PostM	DM/H			YES		P	P	167	↑	CAK/PVC	EA	VF	DIED
13	DAVAMANI	54	47365	PostM								163	↑		AS	PMD	
14	DEVI	44	43876	PreM	DM/MI				P	P		292	↑		AS	VF	
15	FATHIMA	53	34569	PostM	DM/H			YES	P	P	P	289	↑	C.Mly	AS	VT	
16	FARIDA	65	38762	PostM	O		YES			P		251	↑		IL		
17	FIROZ	62	40594	PostM	H						P	176	↑	PVC/C.Mly	AL		

FH-Family History, OCP-Oral Contraceptive Pill ,H-Hypertension, DM-Diabetes Mellitus, MI-Myocardial Infarction, O-Obesity, CPK-MB-Creatine Phosphokinase, P-present AS-Anteroseptal, AL-Anterolateral, EA-Extensive anterior, IL-Inferolateral, RV-Right Ventricular,Inf-Inferior Wall, VE- Ventricular Ectopics ,VT-Ventricular Tachycardia, VF- Ventricular Fibrillation, AE-Atrial Ectopics, IstHB-Ist Degree Heart Block , CS-Cardiogenic shock, CA-Cardiac Arrest, PMD-Papillary Muscle Dysfunction, C.Mly-Cardiomegaly , PVC-Pulmonary Venous Congestion, CAK-Calcified Aortic Knuckle, ↑-Raised.

MASTER CHART

S.NO	NAME	AGE In Yrs	1P.No.	Men. Status	F.H.	OCP Use	Smoking	Tobacco Chewing	Abdl Obesity	D.M	H	Hyper-Cholesterolemia	CPK-MB	CXR	Infarct Site	Complication	Mortality
18	GEETHA	35	45632	PreM	DM/HT/ MI	YES				P		258	↑	C.Mly	AS	VE	
19	GOUTHAMI	63	34516	PostM								156	↑	PVC	AS	LVF	
20	HEMA	56	45678	PostM								178	↑		AS		
21	ILAVARASI	64	31231	PostM								158	↑		AS	VE	
22	INDUMATHI	51	39654	PostM								156	↑	PVC	Inf+RV	Ist HB	
23	INDIRA	35	46703	PreM	DM/HT MI/O	YES			P		P	290			AS	VT	
24	INDRAKUMARI	77	47628	PostM	H			YES		P	P	276	↑	PVC/C.Mly	Inf+RV		
25	JAMUNA	61	47893	PostM								157	↑			AS	
26	JEBAKUMARI	54	43508	PostM	O					P		176	↑		EA	CA	
27	JOHIRA	55	42369	PostM	DM/H					P	P	168	↑	C.Mly	AS		
28	JOTHI	44	36781	PreM	H	YES			P			152		PVC	AS	VE	
29	KAMALAMMAL	76	33456	PostM	H						P	170	↑	C.Mly	Inf		
30	KARUPAYEE	62	44444	PostM	H					P		175			AS		
31	KARUPAMMAL	69	36480	PostM	DM/H/O			YES	P	P	P	275	↑	C.Mly	AL	IstHB	
32	KONDAMMAL	54	37654	PostM	H						P	177		C.Mly/PVC	AS	LVF	DIED
33	KUPPAMMAL	65	39463	PostM	H/MI/O		YES	YES			P	250	↑		Inf	IstHB	

FH-Family History, OCP-Oral Contraceptive Pill ,H-Hypertension, DM-Diabetes Mellitus, MI-Myocardial Infarction, O-Obesity, CPK-MB-Creatine Phosphokinase, P-present AS-Anteroseptal, AL-Anterolateral, EA-Extensive anterior, IL-Inferolateral, RV-Right Ventricular,Inf-Inferior Wall, VE- Ventricular Ectopics ,VT-Ventricular Tachycardia, VF- Ventricular Fibrillation, AE-Atrial Ectopics, IstHB-Ist Degree Heart Block , CS-Cardiogenic shock, CA-Cardiac Arrest, PMD-Papillary Muscle Dysfunction, C.Mly-Cardiomegaly , PVC-Pulmonary Venous Congestion, CAK-Calcified Aortic Knuckle, ↑-Raised.

MASTER CHART

S.NO	NAME	AGE In Yrs	1P.No.	Men. Status	F.H.	OCP Use	Smo king	Tobacco Chewing	Abdl Obesity	D.M	H	Hyper-Choles terolemia	CPK-MB	CXR	Infarct Site	Comp lication	Mortality
34	LALITHA	58	45790	PostM	H				P		P	267		PVC	Inf+RV	CS	DIED
35	LATHA	44	43213	PreM	H				P		P	288	↑		Inf		
36	LAVANYA	58	33333	PostM	MI							156	↑	PVC	AS		
37	MANJU	42	34590	PreM	DM/O	YES						148	↑		AL	VE	
38	MANJULA	79	47832	PostM	DM			YES		P		150	↑	CAK	AL	VT	
39	MARAGATHAM	69	45702	PostM	MI/O		YES		P			262			Inf+RV	VT	
40	MEGHALA	52	43567	PostM	DM					P		276	↑	PVC	Inf		
41	MOHINI	45	44466	PreM	DM				P	P	P	241	↑	C.Mly	AL	VT	
42	NAGAMMAL	63	45666	PostM	H/MI			YES			P	263	↑	C.Mly	AS		
43	NALINI	43	48102	PreM	DM					P		150	↑		Inf		
44	NANJAMMAL	60	34789	PostM								168	↑	PVC	AS	LVF	
45	NOORJAHAN	57	37845	PostM								167	↑		Inf		
46	PADMAKUMARI	54	35098	PostM	MI/O				P			291			AS		
47	PANKAJAM	62	45877	PostM	DM					P		185		PVC	AS	PMD	
48	PARVATHAMAL	50	47555	PostM	DM/H				P	P	P	282	↑		AS		
49	PARVATHI	59	34566	PostM	DM/O				P	P		174	↑		AS	AE	

FH-Family History, OCP-Oral Contraceptive Pill ,H-Hypertension, DM-Diabetes Mellitus, MI-Myocardial Infarction, O-Obesity, CPK-MB-Creatine Phosphokinase, P-present AS-Anteroseptal, AL-Anterolateral, EA-Extensive anterior, IL-Inferolateral, RV-Right Ventricular,Inf-Inferior Wall, VE- Ventricular Ectopics ,VT-Ventricular Tachycardia, VF- Ventricular Fibrillation, AE-Atrial Ectopics, IstHB-Ist Degree Heart Block ,CS-Cardiogenic shock, CA-Cardiac Arrest, PMD-Papillary Muscle Dysfunction, C.Mly-Cardiomegaly , PVC-Pulmonary Venous Congestion, CAK-Calcified Aortic Knuckle, ↑-Raised

MASTER CHART

S.NO	NAME	AGE In Yrs	1P.No.	Men. Status	F.H.	OCP Use	Smoking	Tobacco Chewing	Abdl Obesity	D.M	H	Hyper-Cholesterolemia	CPK-MB	CXR	Infarct Site	Complication	Mortality
50	POORANI	44	47836	PreM	H				P		P	152		C.Mly	EA		
51	RANGAMMAL	60	45690	PostM	H/O			YES	P		P	266	↑	PVC	Inf+RV	CS	DIED
52	RANGANAYAGI	54	32478	PostM	DM			YES		P		156	↑		Inf	IstHB	
53	RASATHI	60	44556	PostM								158		CAK	Inf		
54	RATHI	44	45666	PreM	MI				P			276	↑		Inf	VE	
55	REKHA	64	47880	PostM	DM/H/O				P	P		270	↑		AS		
56	SARANYA	44	43562	PreM	H/MI	YES		YES	P	P	P	290	↑	C.MlyPVC	Inf+RV	LVF	DIED
57	SENIAMMAL	63	37628	PostM								186	↑		AS	VE	
58	SELVANAYAGI	53	37650	PostM	H/MI			YES	P		P	289	↑		AS		
59	SENTHAMARAI	68	46653	PostM	DM			YES		P		168		C.Mly	AS	AE	
60	SENTHOORAM	75	47134	PostM								159	↑	CAK	Inf		
61	TAMILSELVI	72	39345	PostM								148	↑	PVC	IL		
62	THAMARAI	68	36741	PostM						P		172	↑		EA	CS	
63	URVASI	43	43756	PreM	DM/O							165		PVC	Inf+RV	CS	
64	VANITHA	70	48356	PostM	H/MI						P	164	↑	CAK/C.Mly	AS		
65	VASAVI	43	46834	PreM	DM				P			175	↑		Inf	CS	

FH-Family History, OCP-Oral Contraceptive Pill ,H-Hypertension, DM-Diabetes Mellitus, MI-Myocardial Infarction, O-Obesity, CPK-MB-Creatine Phosphokinase, P- present AS-Anteroseptal, AL-Anterolateral, EA-Extensive anterior, IL-Inferolateral, RV-Right Ventricular,Inf-Inferior Wall, VE- Ventricular Ectopics ,VT-Ventricular Tachycardia, VF- Ventricular Fibrillation, AE-Atrial Ectopics, IstHB-Ist Degree Heart Block , CS-Cardiogenic shock, CA-Cardiac Arrest, PMD-Papillary Muscle Dysfunction, C.Mly-Cardiomegaly , PVC-Pulmonary Venous Congestion, CAK-Calcified Aortic Knuckle, ↑-Raised.

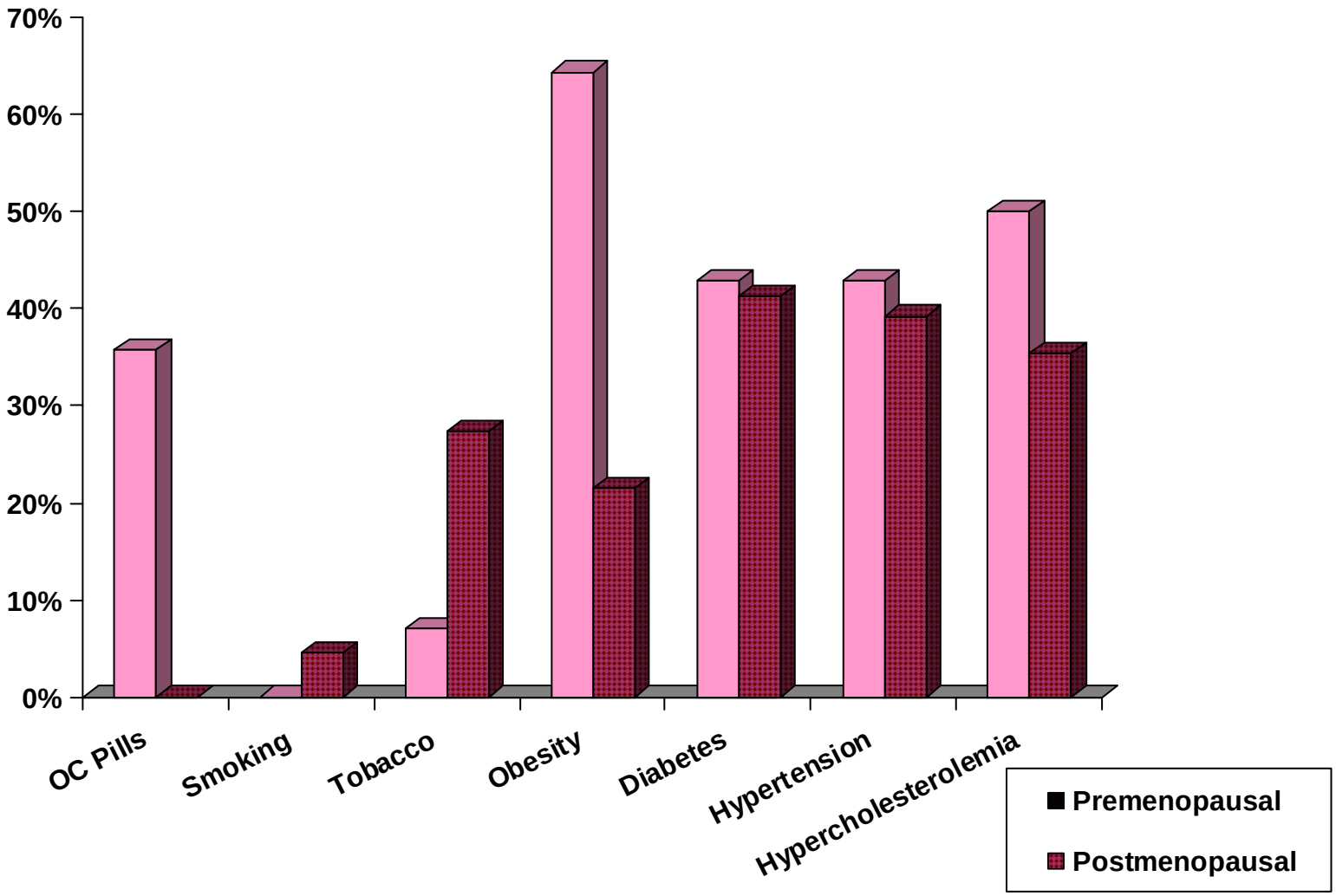


FIG 6: COMPARISON OF INDIVIDUAL RISK FACTORS BETWEEN PREMENOPAUSAL AND POSTMENOPAUSAL WOMEN

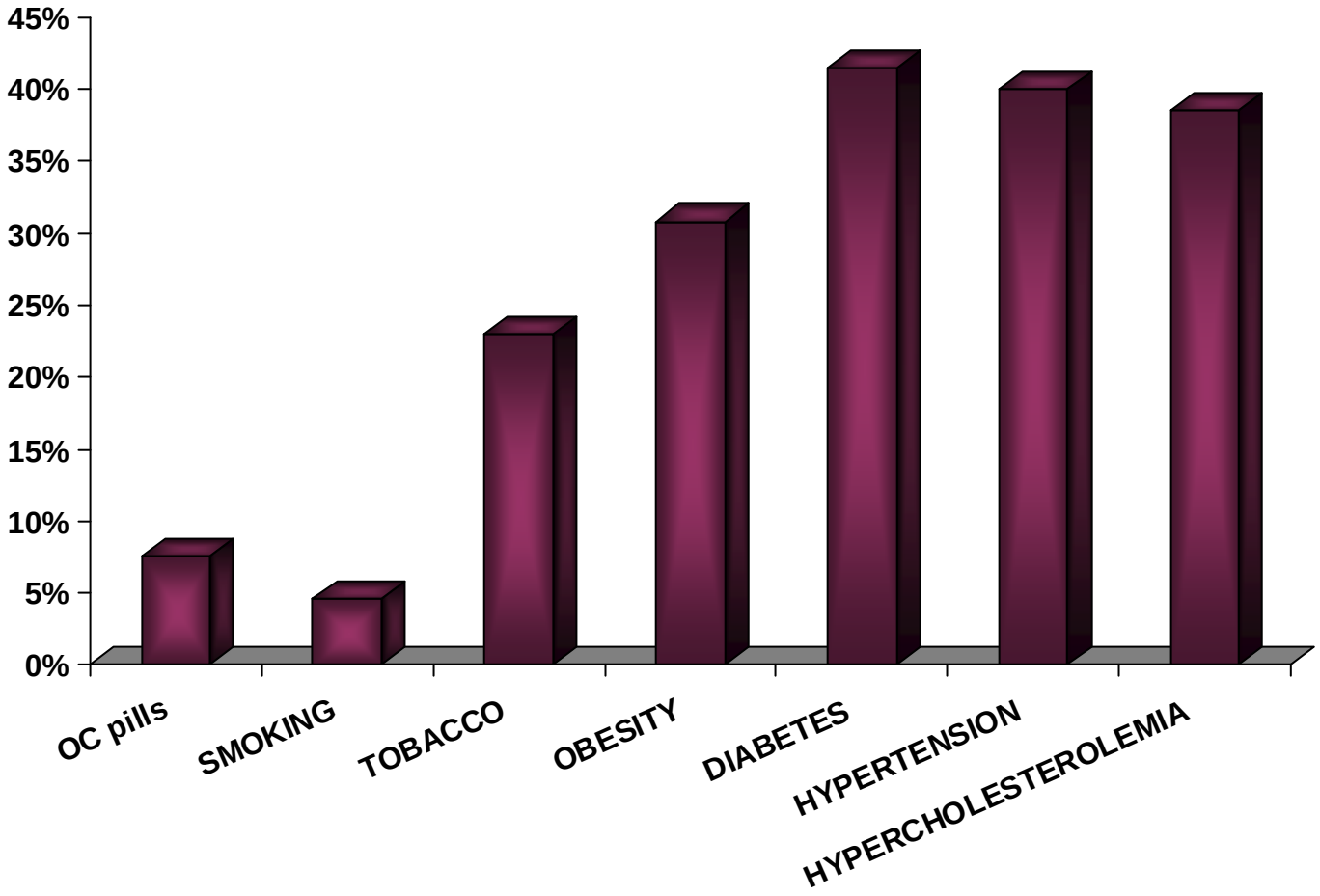


FIG 4: COMPARISON OF RISK FAFCTORS OF MYOCARDIAL INFARCTION IN WOMEN

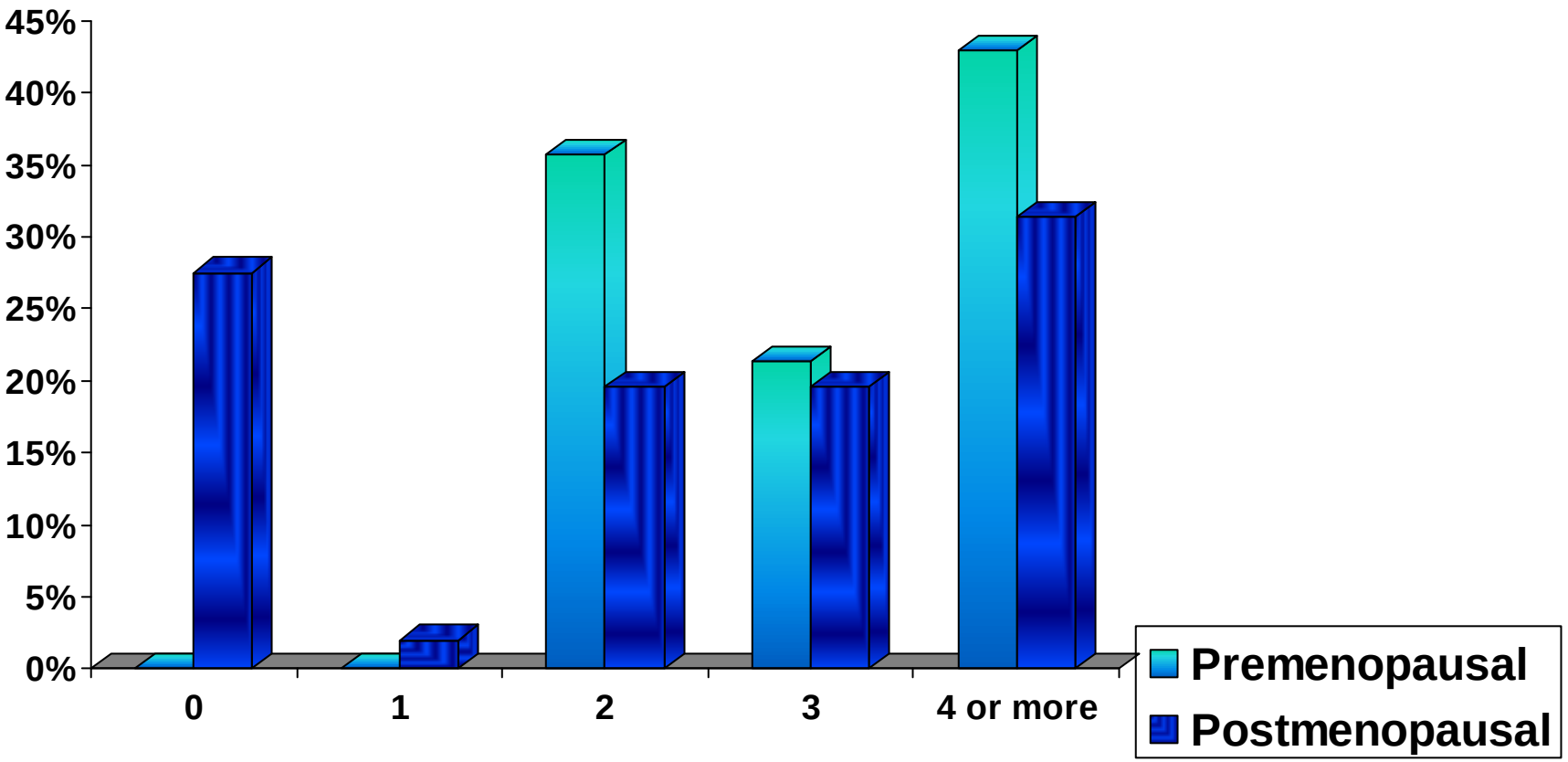


FIG 5: COMPARISON OF MULTIPLE RISK FACTORS IN GROUP A & B

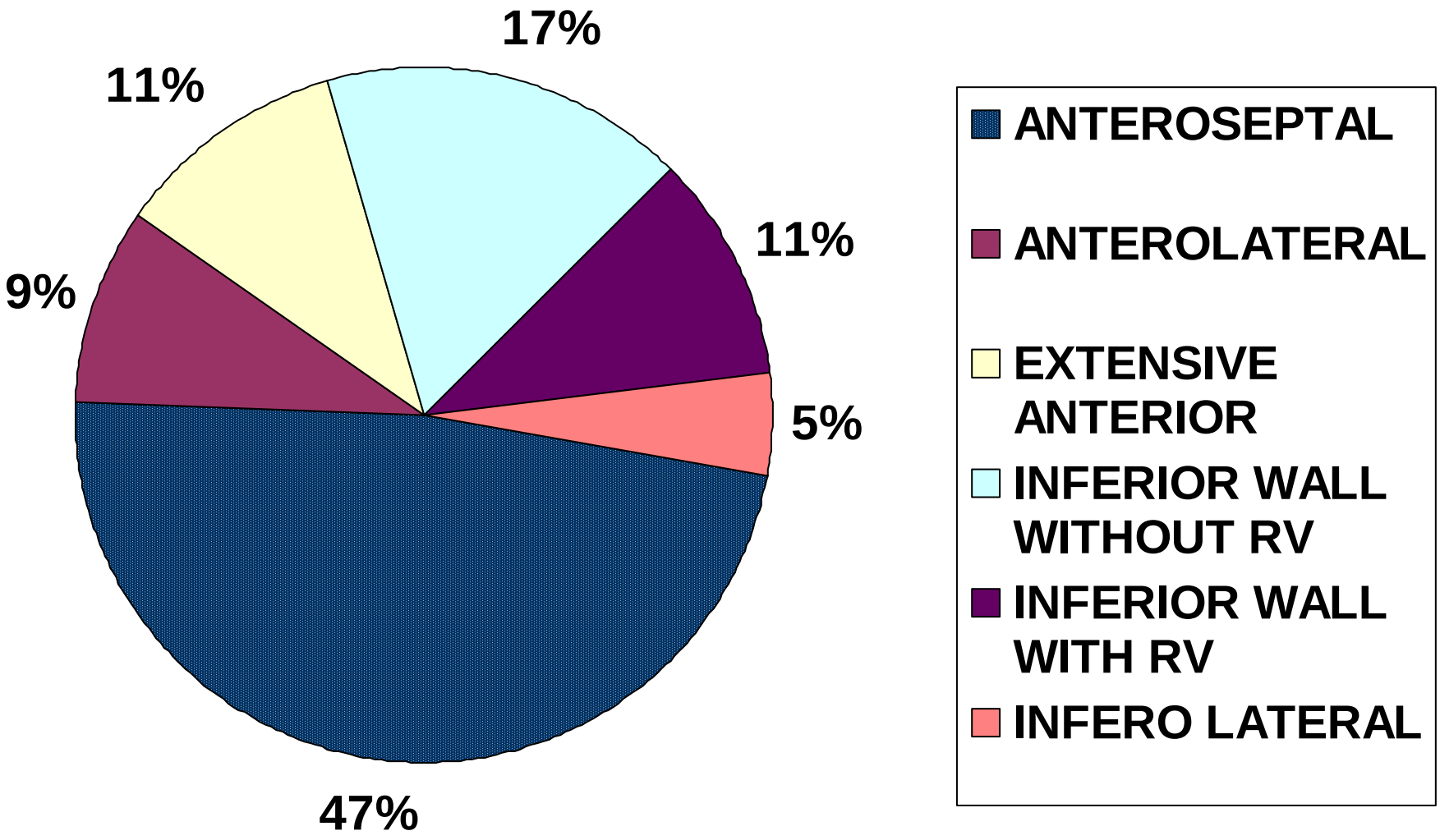

17%
11%
11%
9%
5%
47%
ANTEROSEPTAL
ANTEROLATERAL
EXTENSIVE ANTERIOR
INFERIOR WALL WITHOUT RV
INFERIOR WALL WITH RV
INFERO LATERAL

FIG 7: SITE OF INFARCTION

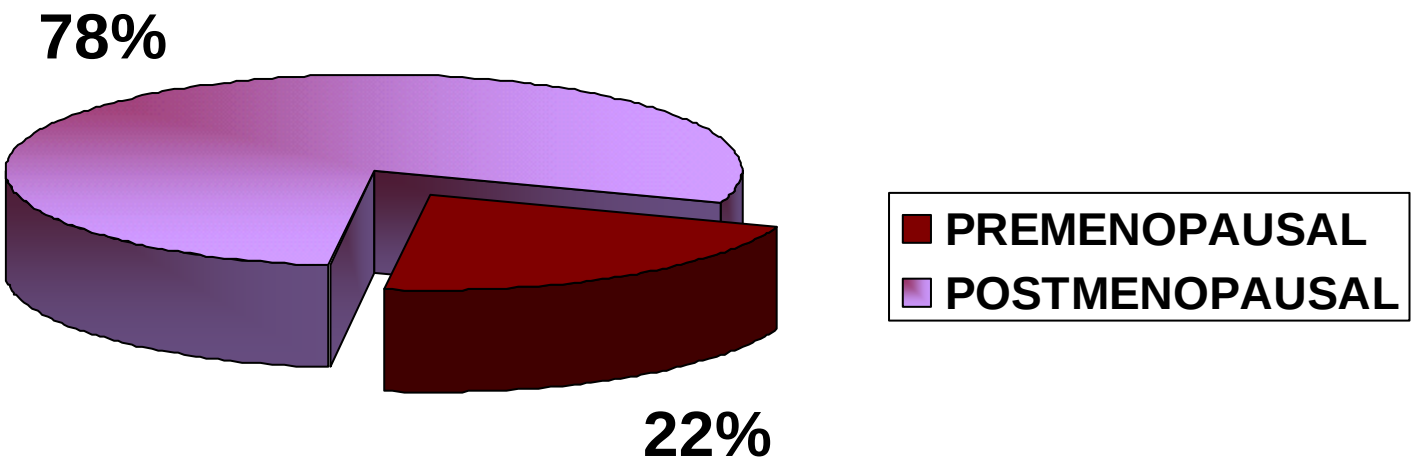
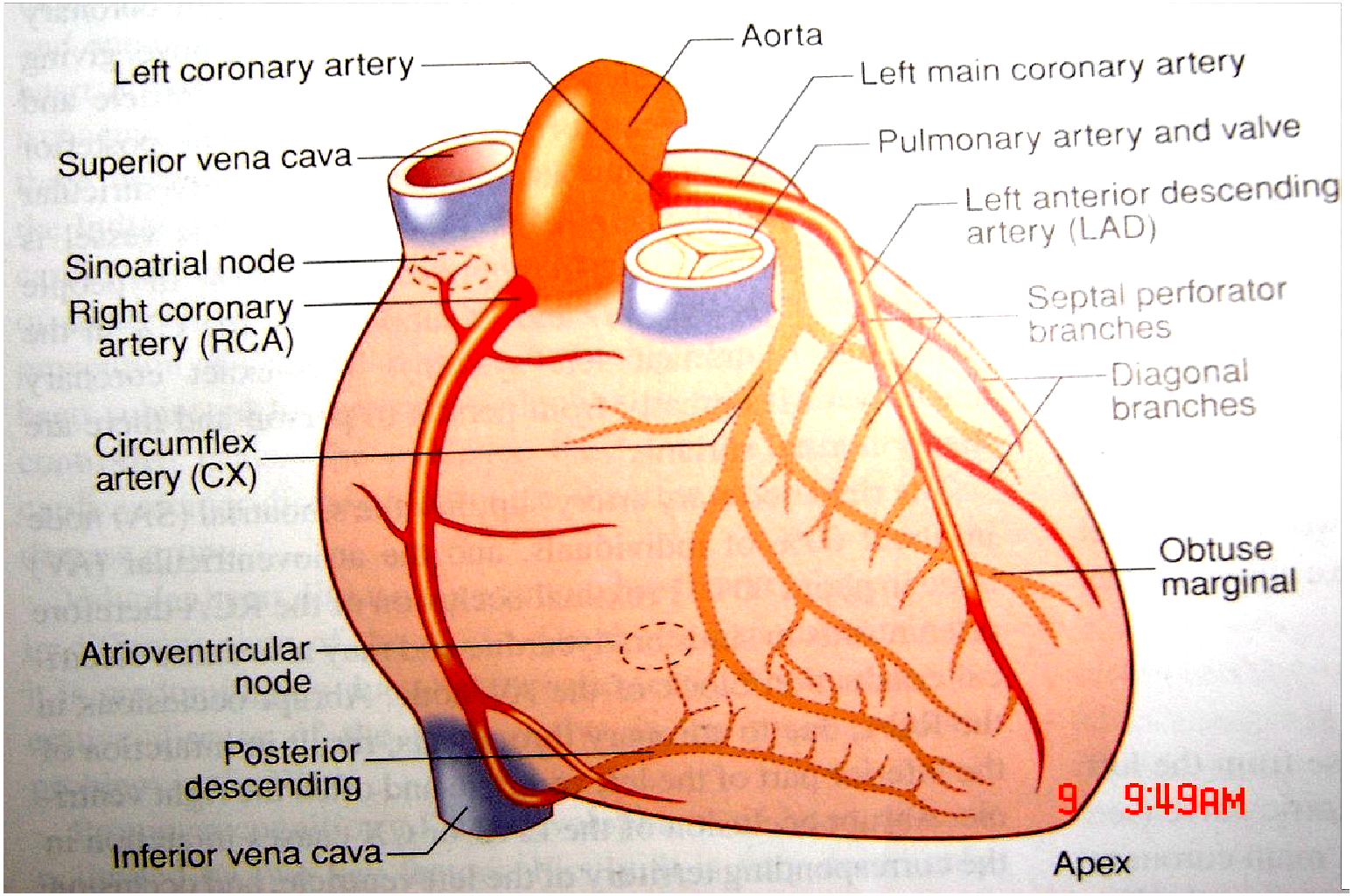
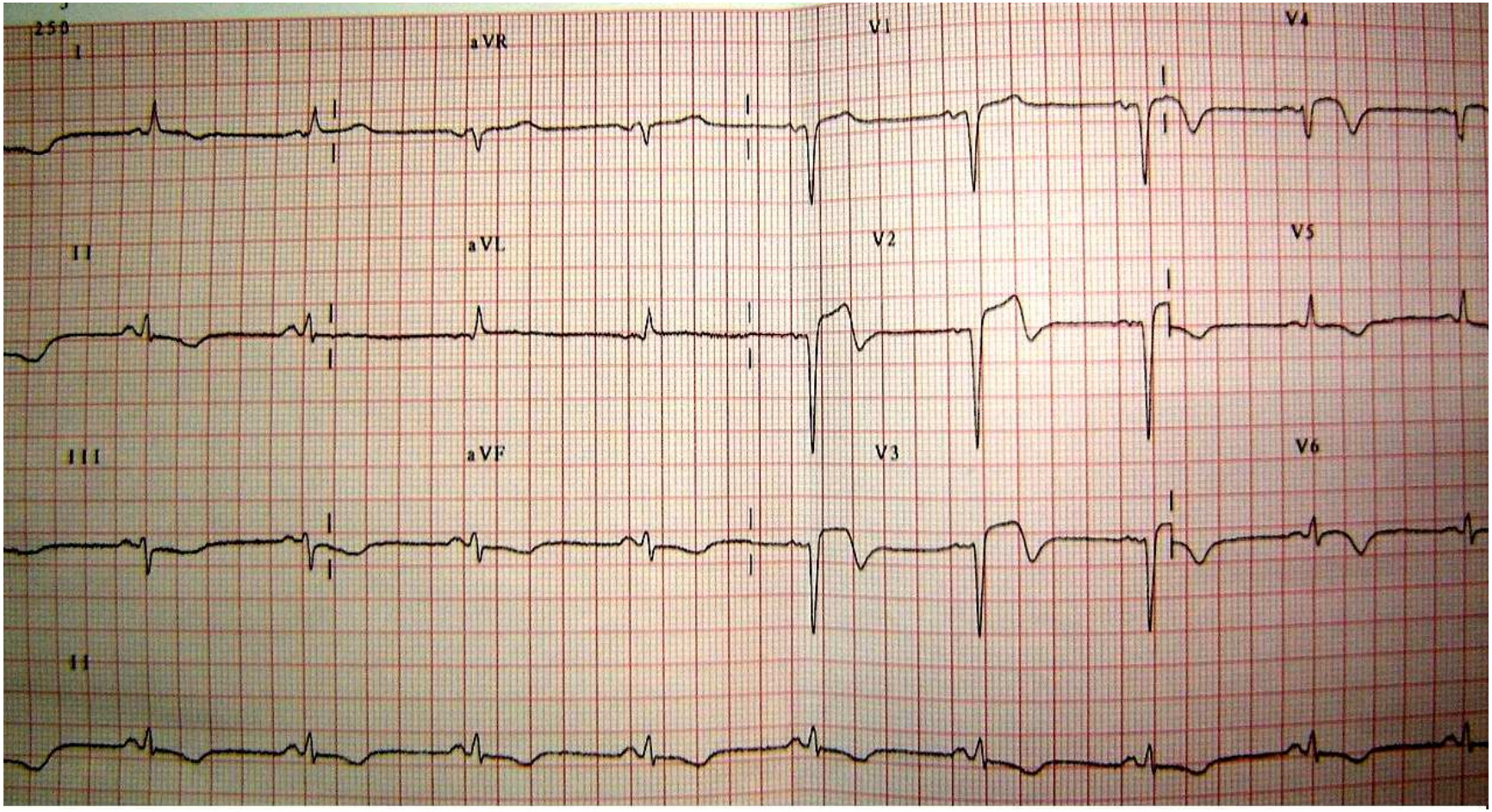


FIG 1: PERCENTAGE OF WOMEN IN GROUP A & B



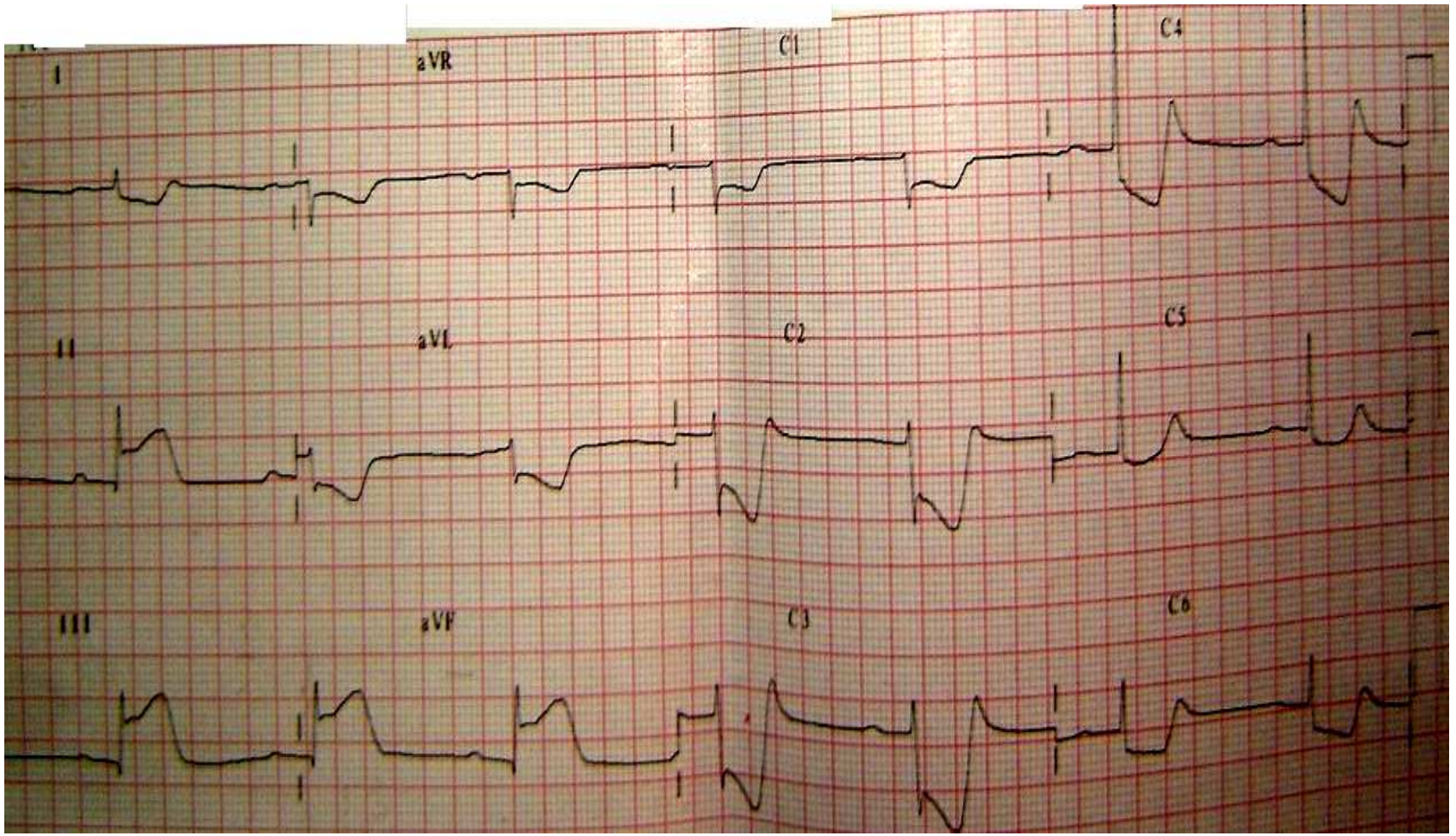
CORONARY CIRCULATION

ALAGAMMAL 53yrs IP.No.30407



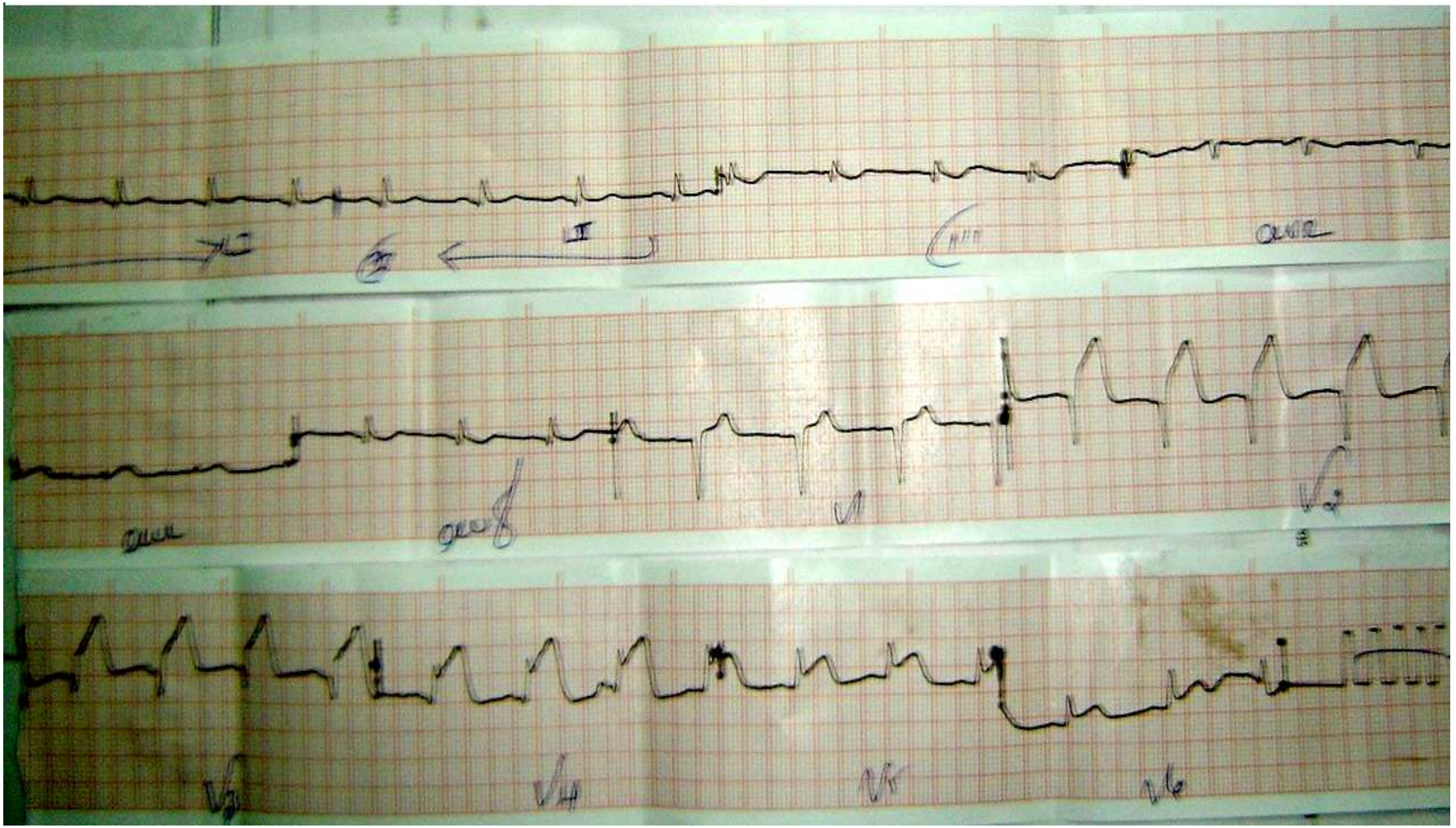
ECG SHOWING ACUTE ANTEROSEPTAL MYOCARDIAL INFARCTION

LATHA 44Yrs IP.No.43213



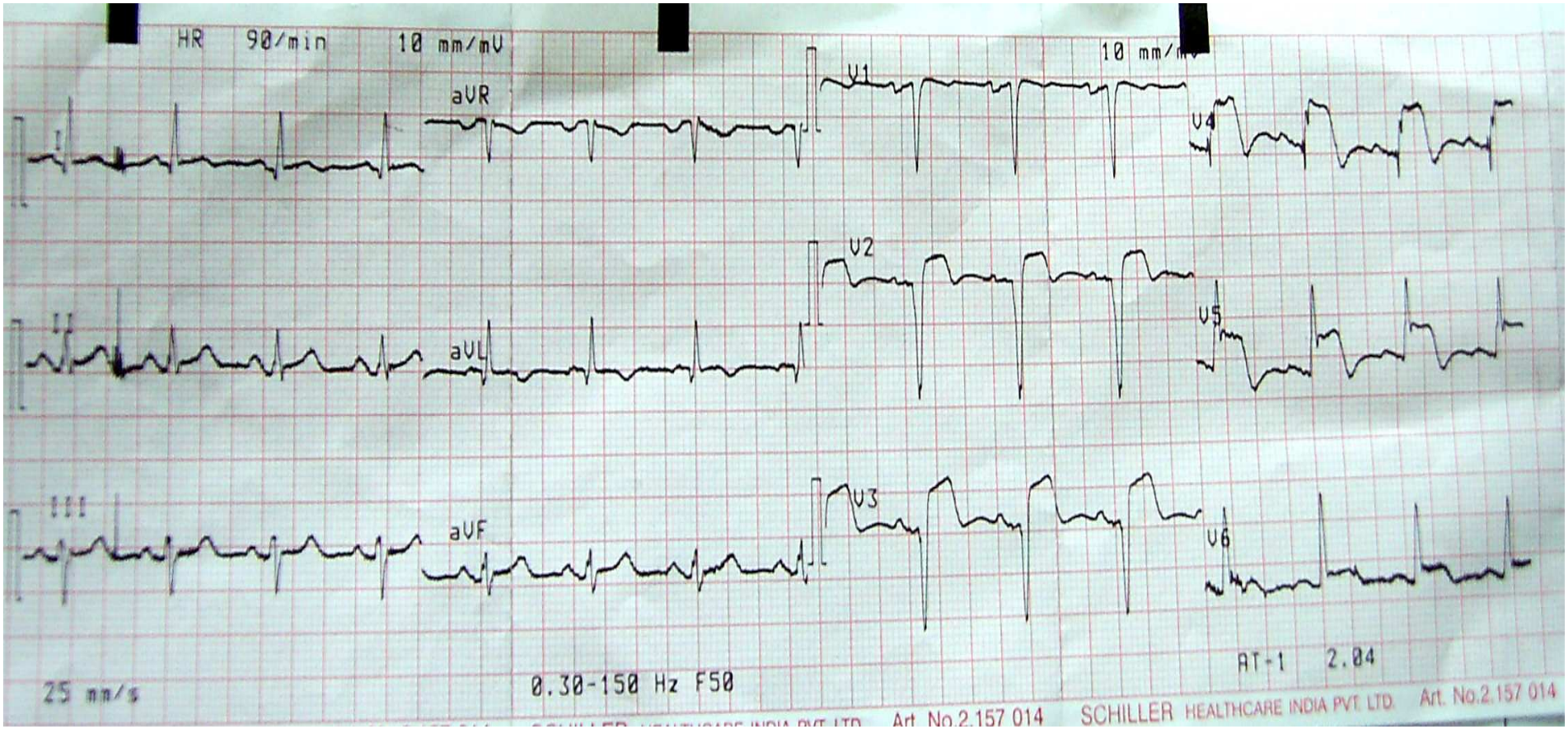
ECG SHOWING ACUTE INFERIOR WALL MYOCARDIAL INFARCTION

THAMARAI 68Yrs IP.No.36741

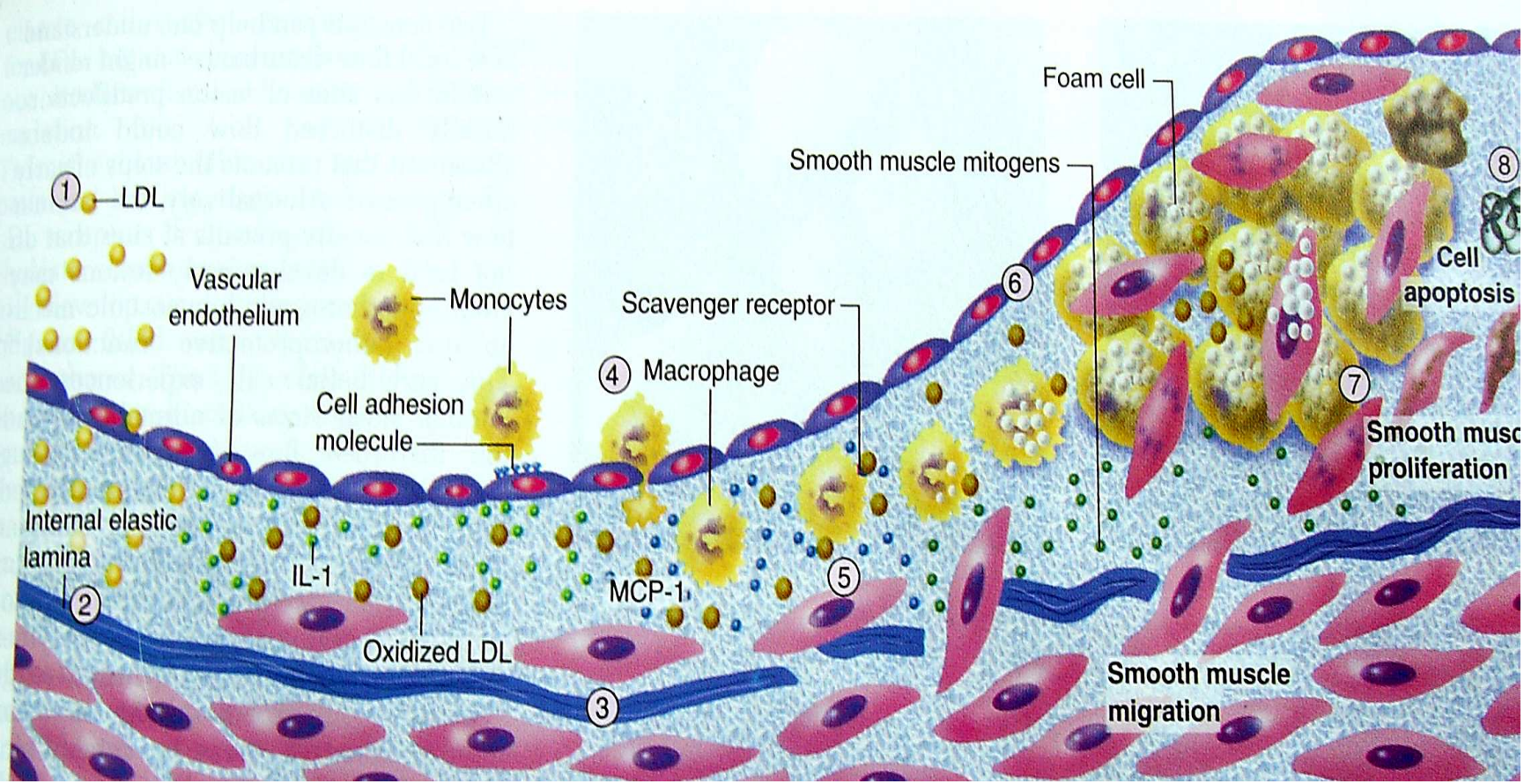


ECG SHOWING EXTENSIVE ANTERIOR WALL MYOCARDIAL INFARCTION

POORNI **44yrs** **47836**



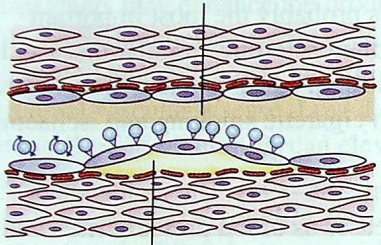
ECG SHOWING ANTERIOR WALL MYOCARDIAL INFARCTION



EVOLUTION OF ATHEROSCLEROTIC PLAQUE

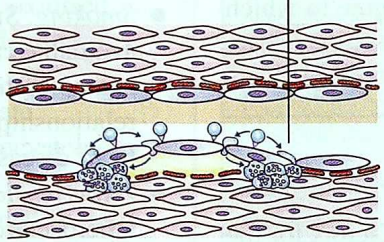
Early atherosclerosis

Activated endothelial cells express adhesion molecules and recruit inflammatory cells, predominantly monocytes

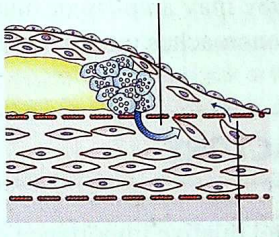


Lipid accumulates in intimal space
Abnormal endothelial cell function

Monocytes migrate into intima, differentiate into macrophages and ingest lipid to form foam cells

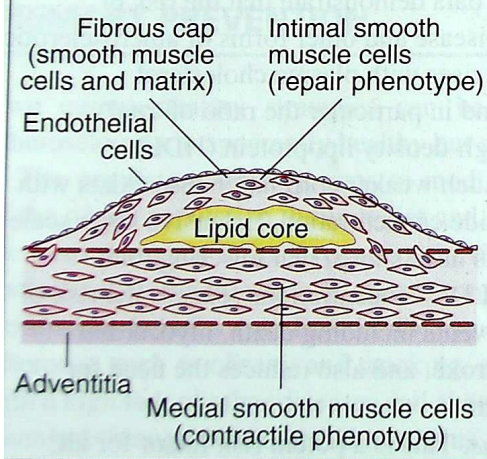


Cytokines and growth factors produced by activated macrophages induce smooth muscle cell migration into the intima



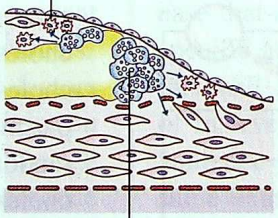
Migrating smooth muscle cells change from contractile to repair phenotype

Stable atherosclerotic plaque



Advanced atherosclerosis

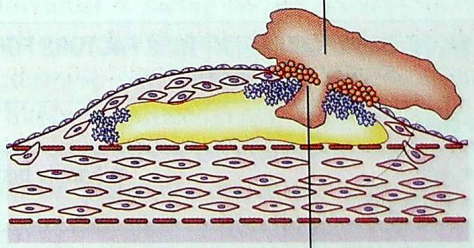
Intimal smooth muscle cells become senescent



Activated macrophages induce intimal smooth muscle cell death and degrade matrix in the fibrous cap

Unstable coronary artery disease

Thrombus forms and extends into the lumen and the plaque



Platelets aggregate at site of rupture/erosion

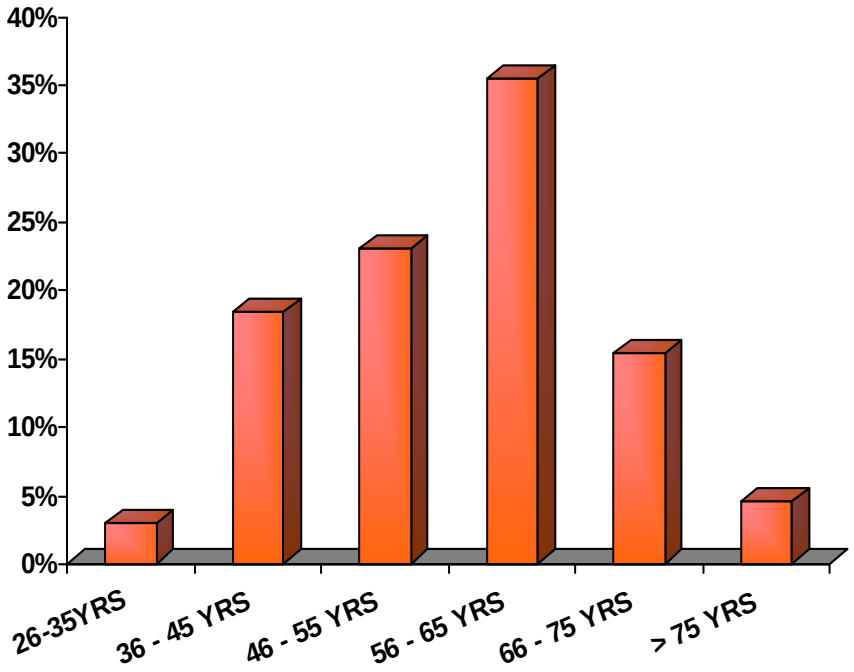


FIG 2: INCIDENCE OF MYOCARDIAL INFARCTION FOR DIFFERENT AGE GROUPS IN WOMEN

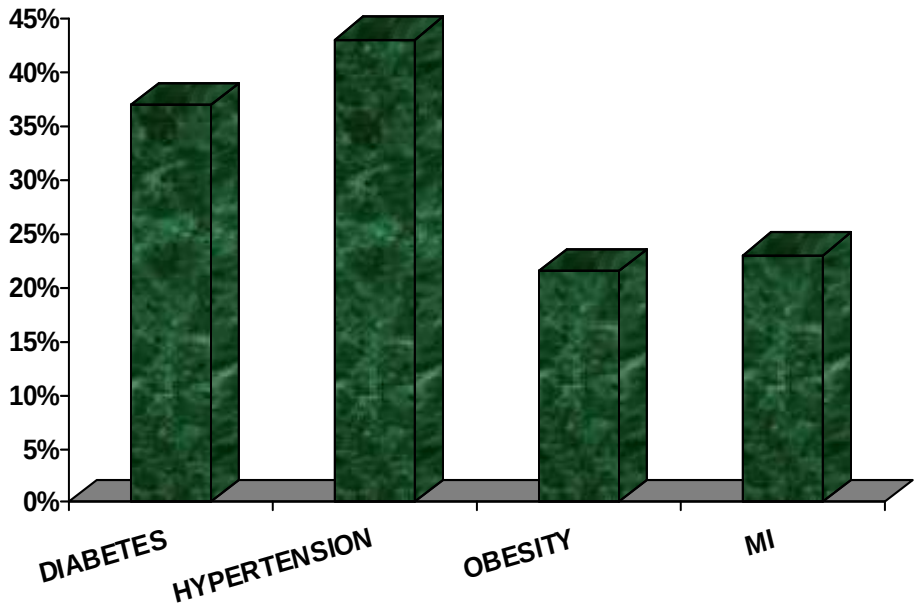
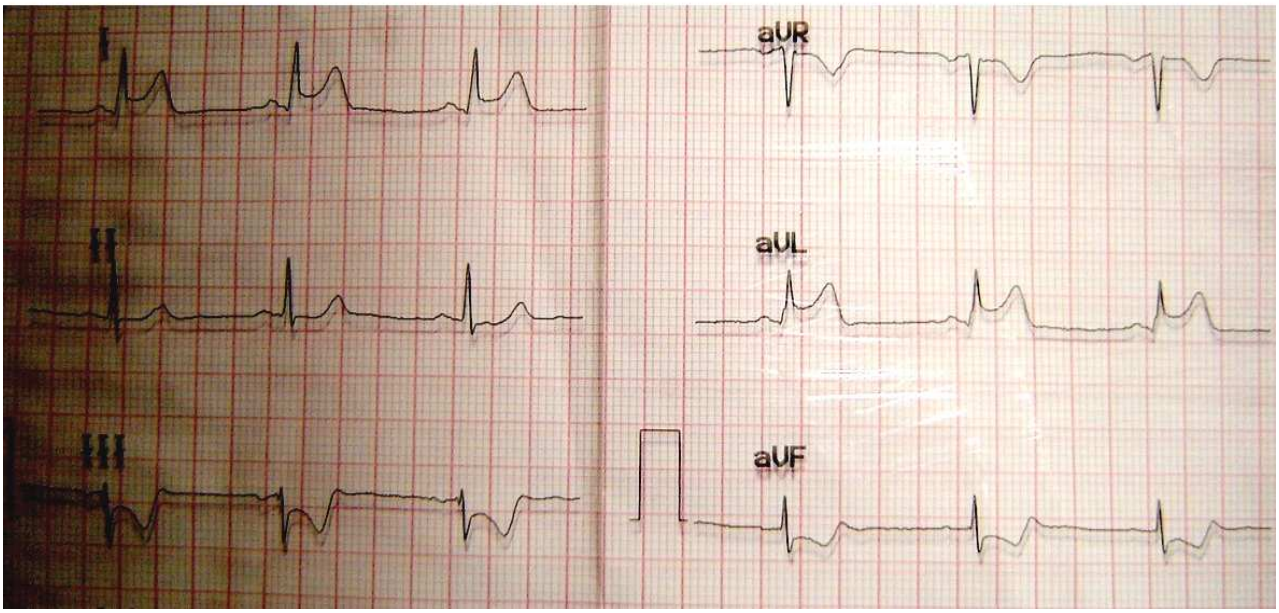
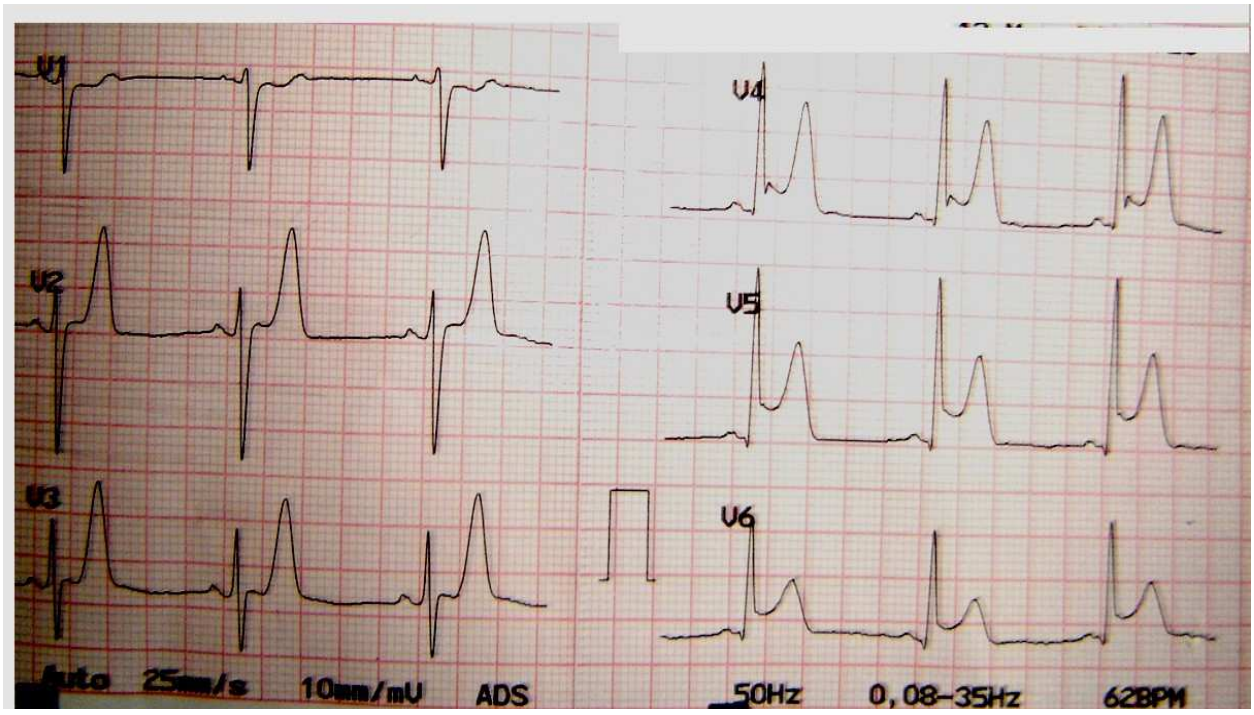


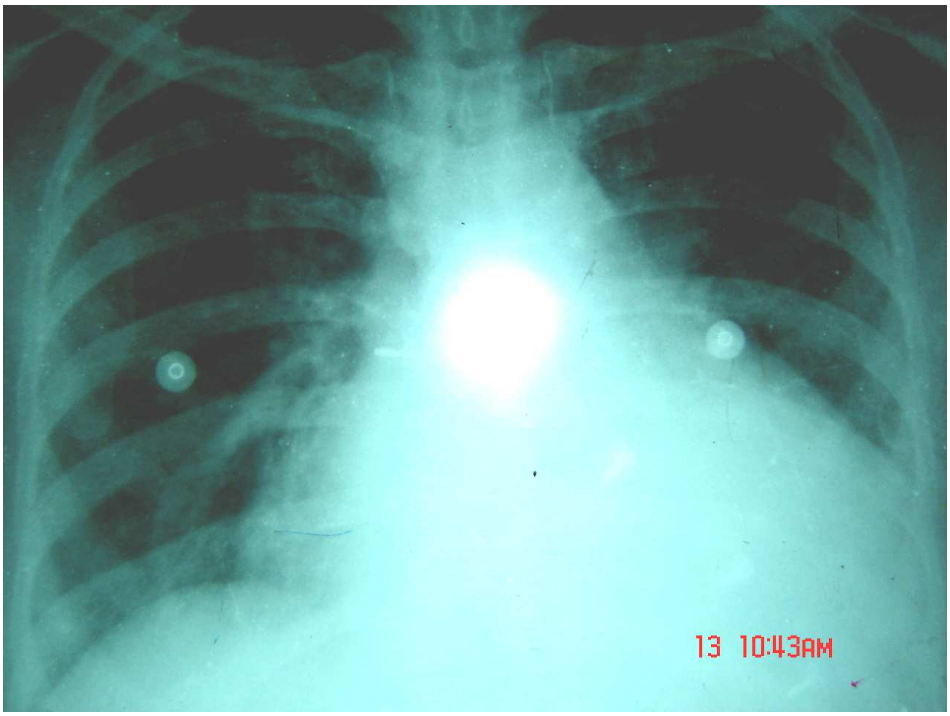
FIG 3: INCIDENCE OF POSITIVE FAMILY HISTORY FOR MI IN WOMEN

CHINNAKANNU 65Yrs IP.No.35786

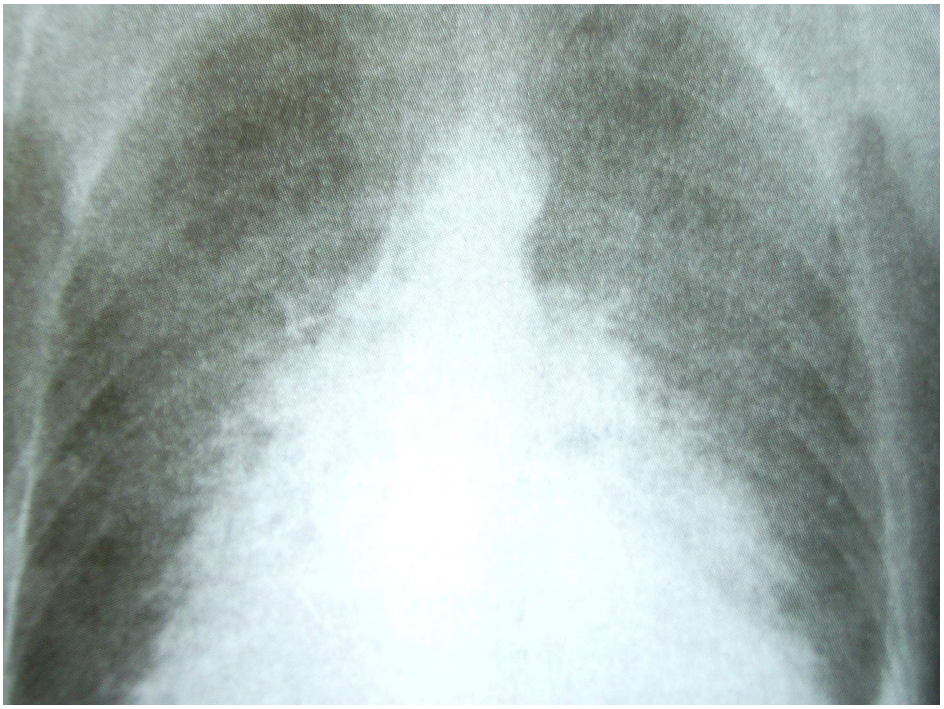




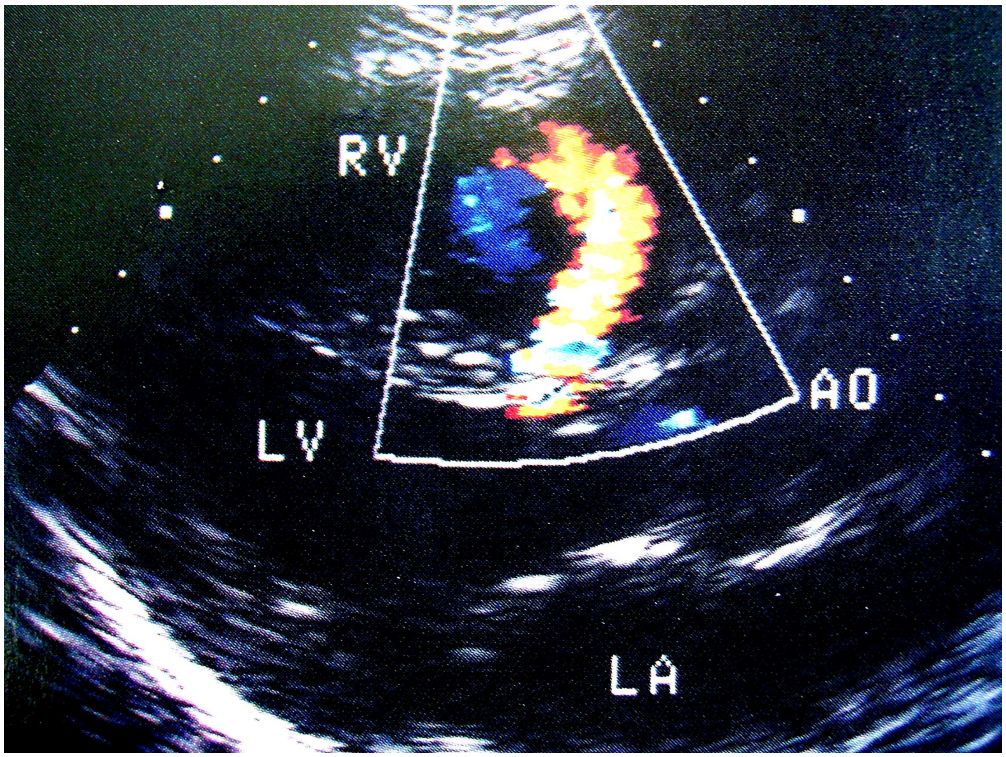
ECG SHOWING ANTEROLATERAL MYOCARDIAL INFARCTION



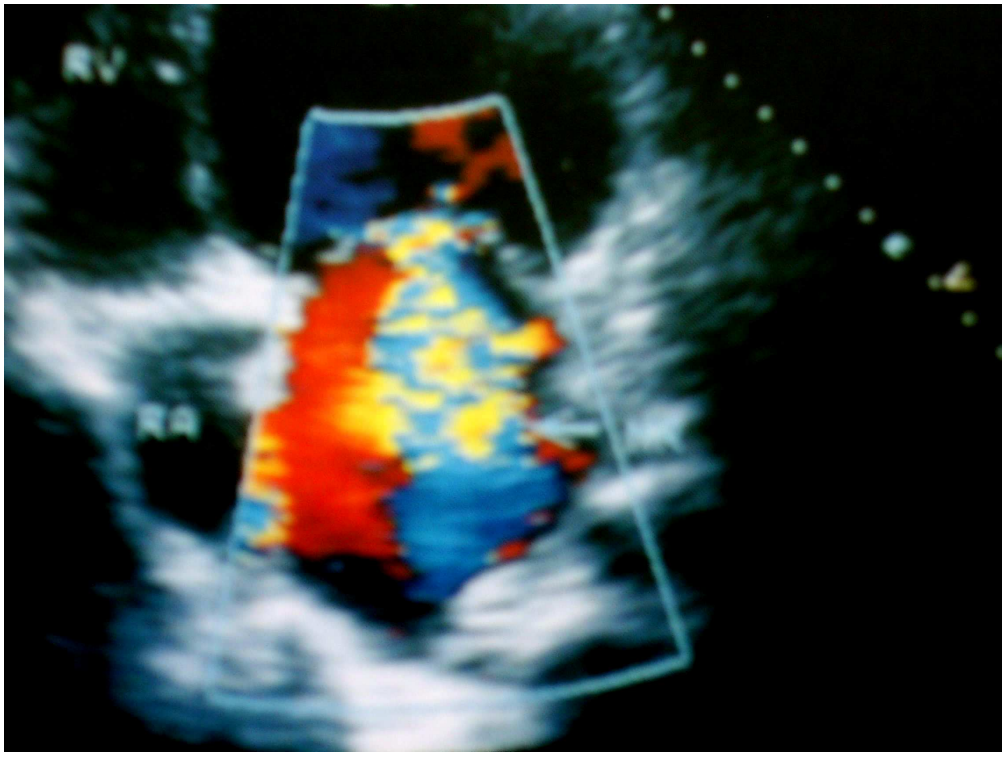
X-RAY CHEST SHOWING CARDIOMEGALY



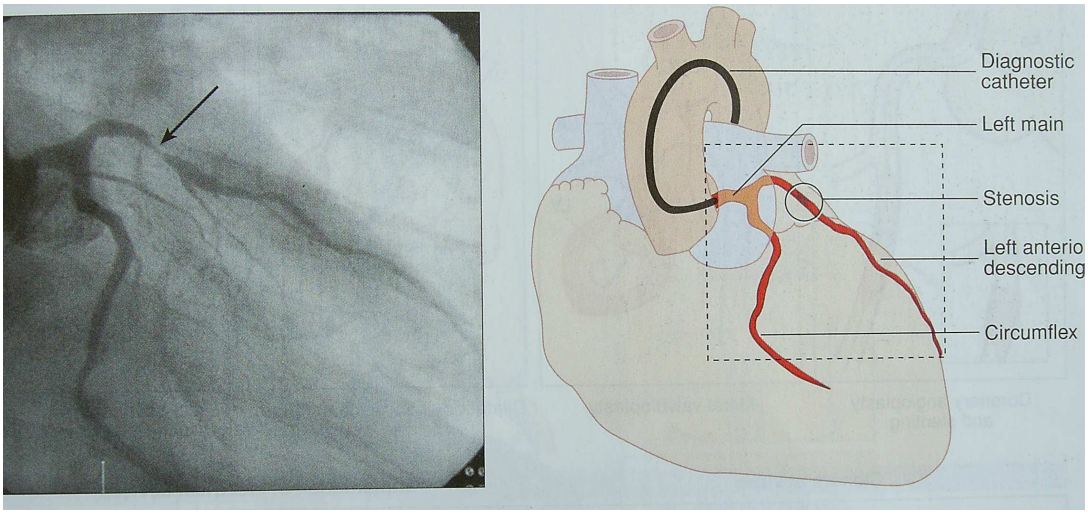
X-RAY CHEST SHOWING FEATURES OF HEART FAILURE



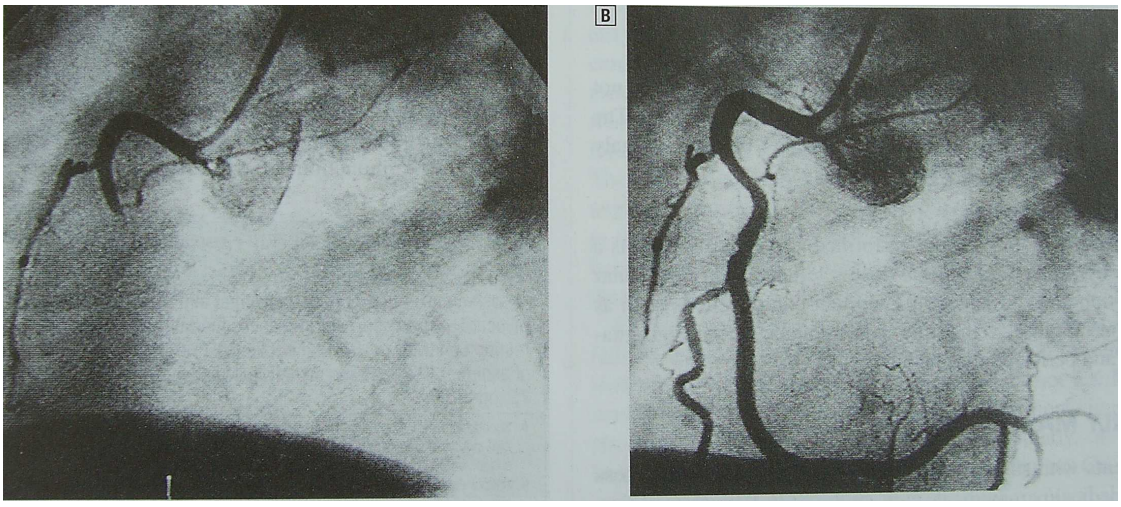
ECHO SHOWING VSD



ECHO SHOWING MITRAL REGURGITATION



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



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

PROFORMA

MYOCARDIAL INFARCTION IN FEMALES

NAME **AGE** **SEX**

ADDRESS **OCCUPATION** **INCOME**

DIAGNOSIS **IP. NO.** **D.O.A.** **D.O.D.**

COMPLAINTS

H/O PRESENT ILLNESS

CHEST PAIN-TYPE, RADIATION
SWEATING
PALPITATION
SYNCOPE
DYSPNOEA
PND, ORTHOPNOEA
GIDDINESS
NAUSEA & VOMITING

PAST HISTORY

SYSTEMIC HYPERTENSION ***DIABETES MELLITUS***

COPD ***TUBERCULOSIS***

PERSONAL HISTORY:

SMOKING, ALCOHOL.

DIET: *VEG/NONVEG/MIXED*

FAMILY HISTORY:

IHD/OBESITY/DIABETES/HYPERTENSION/HYPERLIPIDEMIA

TREATMENT HISTORY: *DRUG INTAKE*

MARITAL HISTORY

MENSTRUAL HISTORY

OBSTETRIC HISTORY

GENERAL EXAMINATION

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

CARDIOVASCULAR SYSTEM

APICAL IMPULSE S1 S2 S3

PERICARDIAL RUB PERIPHERALVASCULAR SYSTEM

RESPIRATORY SYSYEM

ABDOMEN

CENTRAL NERVOUS SYSTEM

FUNDUS

INVESTIGATION

- ***URINE ROUTINE***
- ***BLOOD SUGAR***
- ***BLOOD UREA***
- ***SERUM CREATININE***
- ***SERUM CHOLESTEROL***
- ***SERUM CPK-MB***
- ***ECG***
- ***CHEST X-RAY***
- ***ECHO***