# "A STUDY OF SERUM LIPID PROFILE OF YOUNG ADULTS WITH ACUTE MYOCARDIAL INFARCTION"

**M.D. Degree Examination**

**Branch 1 - General Medicine**

**Coimbatore Medical College,**

**Coimbatore.**

**Dissertation Submitted to**

**THE TAMILNADU**

## DR. M.G.R. MEDICAL UNIVERSITY

September 2006

# CERTIFICATE

This is to certify that the dissertation titled A study of lipid profile in young adults with acute myocardial infarction, is a bonifide work done by Dr. D.Muthukumaran. It is a regular, systematic study done under my guidance and supervision during the period of January-2000-December-2005, and submitted for the ensuing M.D Branch 1 General Medicine examination, September 2006 of the Tamil Nadu Dr. M.G.R Medical university Chennai.

Place:

Date

Unit Chief and Professor of Medicine
Coimbatore Medical College Hospital
Coimbatore

Professor And Head of Department of Medicine
Coimbatore Medical College Hospital,
Coimbatore

The Dean
Coimbatore Medical College,
Coimbatore.

## DECLARATION

I solemnly declare that the dissertation titled, A study of lipid profile in young adults with acute myocardial infarction was done by me at Coimbatore medical college hospital Coimbatore during January 2005-December-2005 under the guidance and supervision of professor Dr.M. Ramasamy MD. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any university, board either in India or abroad.

The dissertation is submitted to the Tamilnadu DR. M.G.R Medical University towards the partial fulfillment of the requirement for the award of MD Degree (Branch1) in General Medicine.

Place:

Date:

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# ABBREVIATIONS

| | | |
|---|---|---|
| TC | - | Total Cholesterol |
| LDL-C | - | Low Density Lipo protein Cholesterol |
| TGL | - | Triglycerides |
| VLDLC | - | Very low density Lipo protein Cholesterol |
| HDLC | - | High density Lipo protein Cholesterol |
| MI | - | Myocardial Infarction |
| CAD | - | Coronary Artery Disease |
| NCEPATPIII | - | National Cholesterol Education Programme Adult Treatment Panel. |
| ECG | - | Electro Cardio Graph |
| ECHO | - | Echo Cardio Graph |

# CONTENTS

# PROFORMA

LIPID PROFILE STUDY IN PATIENT WITH YOUNG MYOCARDIAL INFARCTION

NAME AGE SEX

ADDRESS OCCUPATION INCOME

I.P. NO D.O.A D.O.D

DIAGNOSIS

COMPLAINTS

H/O PRESENT ILLNESS

CHEST PAIN

PALPITATION

SYNCOPE

DYSPNOEA

PND

ORTHOPNOEA

PAST HISTORY

SYSTEMIC HYPERTENSION DIABETES MELLITUS

COPD PULMONARY TUBERCULOSIS

PERSONAL HISTORY: SMOKING

ALCOHOL

DIET:

FAMILY HISTORY

H/O IHD/SHT/DIABETES/HYPER CHOLESTROLEMIA

MYXOEDEMA/LIVER DISEASES/RENAL DISORDERS/

TREATMENT HISTORY DRUG INTAKE

MARITAL HISTORY

MENSTRUAL HISTORY/OBSTETRIC HISTORY

GENERAL EXAMINATION

1. OBESITY
2. DYSPNOEA
3. CYANOSIS
4. CLUBBING
5. PEDAL EDEMA
6. FEATURES OF CRF/THYROID DISEASES
7. XANTHELESMA
8. XANTHOMA RETINALIS
9. TENDON XANTHOMAS
10. ERUPTIVE XANTHOMA
11. ARCUS SENILIS

PULSE

BLOOD PRESSURE

WEIGHT

HEIGHT

BMI

WAIST/HIP RATIO

CARDIO VASCULAR SYSTEM

APICAL IMPULSE S1 S2

GALLOP (S3)

PERICARDIAL RUB

PERIPHERAL VASCULAR SYSTEM

RESPIRATORY SYSTEM

ABDOMEN

CENTRAL NERVOUS SYSTEM

FUNDUS

INVESTIGATION

URINE ALBUMIN SUGAR DEPOSIT

BLOOD SUGAR UREA

SERUM CREATININE

ECG

ECHO

LIPID PROFILE

TOTAL CHOLESTEROL

HDL-C

LDL-C

VLDL-C

TGL

BLOOD CPK-MB

# INTRODUCTION

In Earlier times, starvation consigned languishing bodies to death, now on the other hand prosperity plunges them into the grave.

Lucretivs Ca 50 BC (Hurst)

Coronary heart disease has been defined as impairment of heart function due to inadequate blood flow to the heart compared to its needs caused by obstructive changes in the coronary circulation to the heart. The most common cause of coronary heart disease is obstructive atherosclerotic disease of epicardial coronary arteries.

Myocardial infarction is the end result of acute massive myocardial ischemia due to total interruption of blood supply leading to death of cardiac muscle cells. Irreversible injury occurs in the area at risk and when occlusion is sustained for 4 to 6 hours but most of the damage occurs in first 2 to 3 hours.

Although myocardial infarction mainly occurs in patients older than 40 years young men and women less than 40 years can also suffer myocardial infarction. The disease carries significant morbidity, psychological effects and financial constraints for the person and the family when it occurs at a young age. The protection offered by young age has been slowly taken away by the increased prevalence of risk factors for coronary heart disease in adolescents such as Dyslipoproteinemia, smoking, obesity and lack of physical activity. Most epidemiological data in single and cross cultural populations have strongly linked untreated hypertension, smoking, elevated total and low density lipoprotein cholesterol levels to subsequent development of coronary heart disease.

Evidence supporting the relation between blood cholesterol and coronary heart disease has been strengthened by numerous animal studies showing progression and regression of atherosclerotic lesion as cholostrol level rise and fall by natural history studies of genetic hypercholestremias in which marked cholesterol elevation caused premature atherosclerosis even in the absence of other risk factors[1].

The lipid research clinics coronary primary prevention trial (LRC – CPPT) study, Multiple risk factor intervention trial, Oslo trial, who multifactorial trial, north karelia project, life style heart trial, Stanford coronary risk intervention project have provided strong evidence that the increased total cholesterol level in association with increased incidence of coronary heart disease.

## AIM OF THE STUDY

To study the serum lipid profile and the pattern of dyslipoproteinemia of young adults of 40 years and below with acute myocardial infarction.

# REVIEW OF LITERATURE

## ANATOMY OF CORONARY CIRCULATION

Heart is supplied by two coronary arteries arising from the ascending aorta. The left main coronary artery and right coronary artery arised from the left and right coronary sinus just distal to the aortic wall within the 2.5 cms of it origin. The left main coronary artery divides in to left anterior descending artery (LAD) which runs in the anterior interventricular groove and the left circumflex artery (LCX) which runs posterior atrio ventricular groove.

The LAD gives branches to the left ventricle, the apex and anterior part of septum. LCX gives marginal branches to supply the posterior and inferior surface of the left ventricle. The RCA runs in the right atrio ventricular groove giving branches to supply the right atrium, right ventricle and inferior posterior aspect of the left ventricle. The SA node is supplied by the RCA in about 60% individuals and AV node in 90%. Abrupt occlusion of LCA or LCX causes infarction in the corresponding territory but occlusion of the left main coronary artery is usually fatal. The venous system mainly follows the coronary arteries but drains to the coronary sinus and then to right atrium, small Eustachian veins drains directly into right atrium.

# CORONARY ARTERY DIEASE

## Epidemiology :

Epidemics of coronary heart disease begins at different times in different countries. In United States epidemics began in the early 1920 and in Britan in the 1930. The developing countries are catching up. The countries were the epidemic began earlier are now showing the decline. The decline in coronary artery heart disease mortality in U.S has been attributed to changes in life style and related risk factors. The WHO has initiated project known as MONICA (Multi National Monitoring of Trends and Determinants in Cardio Vascular disease) To elucidate this crisis. To summarise in many developed countries coronary artery heart disease still poses the largest public health problem but even in those showing decline CAD is still a health problem.

## Coronary Artery Disease in India :

A large body of data exists on the occurance of CAD in hospital patients. However there are only two studies on its prevalence in the general population. In study conducted in Chandigargh urban population the prevalence was founded to be 65.4 and 47.8 per 1000 males and females respectively. In village in Hariyana the prevalence was 22.8 and 17.3 per 1000 males and females respectively. However CAD less than 40 years was found to be less than 3% of all patients with CAD.

The pattern in CAD in India has been reported to be as follows.

a) CAD appears a decade earlier compared with the age incidence in developed countries. The peak period is attained between 51 and 60 years.
b) Males are affected more than females
c) Hypertension and diabetics accounts for about 40% of all cases.
d) Heavy smoking is responsible etiologically in a good number of cases.

From epidemiological studies information available on the association of risk factors with CAD lead to the formation of number of risk factors. The risk factors classified into non modifiable and modifiable factors (Framingham study)[2].

| **Non Modifiable** | **Modiafiable** |
|---|---|
| Age | - Cigarette smoking |
| Sex | - Hyper tension |
| Family History | - Elevated Serum Cholesterol |
| Genetic Factors | - Diabetes mellitus |
| Personality | - Obesity |
| | Sedentary Habits |
| | Stress |

The prevalence of risk factors is on the rise in young adults and children. The incidence of risk factors like smoking, hyperlipidemia, diabetic mellitus, obesity, is on the rise in young population. Thus it becomes clear that the prevelance of the CAD is bound to rise in patients less than 40 years in the years to come. The under reporting of the illness and the challenge involved in risk factors modification in this subject of patients would make the management of the potentially growing problem more difficult.

**Bio Chemistry of Lipids** :

The blood lipids include mainly Triglycerides, phospholipids, cholesterol and small amounts of free fatty acids. Blood lipids present an unique problem in transportation because of their isolubility in aqueous media. This problem is solved by

combination of lipids with the protein factions of plasma results in the formation of lipo protein[3].

**Cholesterol**

Cholesterol is an essential component of mammalian cell membrane and furnishes the substrate for steroid hormones and bile acids. Exists in two forms free and esterified forms. Most of the cholesterol in plasma circulates in the form of esters in the core of lipoprotein particles. The enzyme lecithin : Cholesterol acyltransferase forms cholesterol esters by transferring a fatty acyl chain from phosphatidyl choline to cholesterol.

**Triglycerides (TGL)**

These form major fraction of lipids in the plasma lip protein complex. These are glycerol with fatty acids. TGL may be exogenous or endogenous in origin. Exogenous TGL is derived from dietary lipids and is carried in the chylomicron fraction of the lipoprotein. Endogeneous TGL is synthesized in the liver mostly from carbohydrates and is carried in the pre-beta fraction of the lipo protein.

**Phpholipids (PL)**

The PL in the plasma are lecithin, cephalin and Sphingomyelin. The PL exists in the plasma mostly in association with alpha and beta globulins and are carried with the alpha and beta lipo proteins.

**Plasma lipoproteins**

Lipoproteins are complexes of lipids and proteins that are essential for the transport of cholesterol, triglycerides and fat soluble vitamins. There are four major classes of lipoproteins namely Chylomicrons, Pre beta lipo proteins (VLDL). Beta Lipo proteins (LDL) and Alpha Lip Proteins (HDL) and new class of Lipo protein –a

The proteins carriers are known as Apo proteins. The types are A,B,C referred as Apo-A, Apo-B, Apo-C.

**Chylomicrons**

It rich in TGL and derived from intestinal absorption of fats secreted from intestinal mucosal cell into the intra cellular fluids and then enter the lacteals and the lymphatics and finally into the systemic circulation. The apo protein components complex is Apo-B.

**VLDL –C :** - It rich in TGL and derived from liver and secreted by intestines in a manner similar to that of chylomicrons. The apo proteins include Apo-B, Apo-C, The function of VLDL is transport of TGL from the liver to the Extra hepatic tissues.

**LDL-C :** - This is particularly rich in cholesterol. Most LDL derived from intra vascular degradation of VLDL and chylomicrons. Approximately 70% of plasma cholesterol is present in the LDL form. The higher the level of LDL, the greater is the risk of CAD. Its Apo protein is Apo-B.

**HDL-C :** - HDL contains maximum amount protein in cholesterol and phospho lipids. HDL and secreted from both liver and intestinal mucosa. HDL from intestines contains apo-A and from the liver contains apo-C. The HDL cholesterol level is elevated are less likely to develop CAD.

**Lipo Protein – A**

Lipo Protein –A is similar to LDL in lipid and protein composition but it contents additional lipo protein called Apo lipo protein-a. Apo – a is synthesized in liver and it is attached to Apo-B 100 by a disulfide linkage. The mechanism by which lipo protein –a is removed from the circulation is not known.

**Lipo Protein metabolism** :

Lipo Protein metabolism can be thought in terms of three interconnected transport pathways focusing on the liver[4]. One exogenous pathway is responsible for the digestion, absorbs and tissue dissemination of dietary through in each day flows about 100 mg of TGL and 0.5 mg of cholesterol. Digestive enzymes in the intestinal lumen hydrolyse these fats to free cholesterol fatty acid mono and diglycerides which combine with bile salts to form the water soluble miscells responsible for carrying lipids to absorptive sites in the intestine.

Following their absorption into the intestinal enterocytes the components parts of the dietary fats are reconstituted to reform TGL and cholesterol ester which are packaged in chylo microns and secreted in the intestinal lymphatics through which they reach blood stream via thoracic duct. In the circulation TGI is gradually removed by action of enzymes Lipoprotein lipase located on the endothelial surface of the capillary beds in adipose tissue and cardiac and skeletal muscle. This process evicerates the chylomicrons leaving a remnant which is taken up rapidly by the liver depositing in the process dietary cholesterol in that organ. These sterol may be incorporated into hepatocytes membranes oxidized through bile acids, are repackaged into the endogenous TGL rich counter part of the chylomicron, the VLDL.

The liver is responsible for the continuous production of VLDL which in the fasting stage represents body's primary source of circulating TGL energy. The particle is subject to the same lipase mediated digestion process as the chylomicron, except that in this case the resulting particle is not cleared by the liver but instead undergoes additional remodeling to produce a cholesterol enriched particle (LDL) with a plasma

half-life of about three days. LDL is ultimately removed from the circulation by the high affinity LDL receptor particle or by other less well understood scavenger mechanisms which are thought to lead to the incorporation of LDL cholesterol into the atheromatous plaques. Raised level of LDL cholesterol predisposed to CAD.

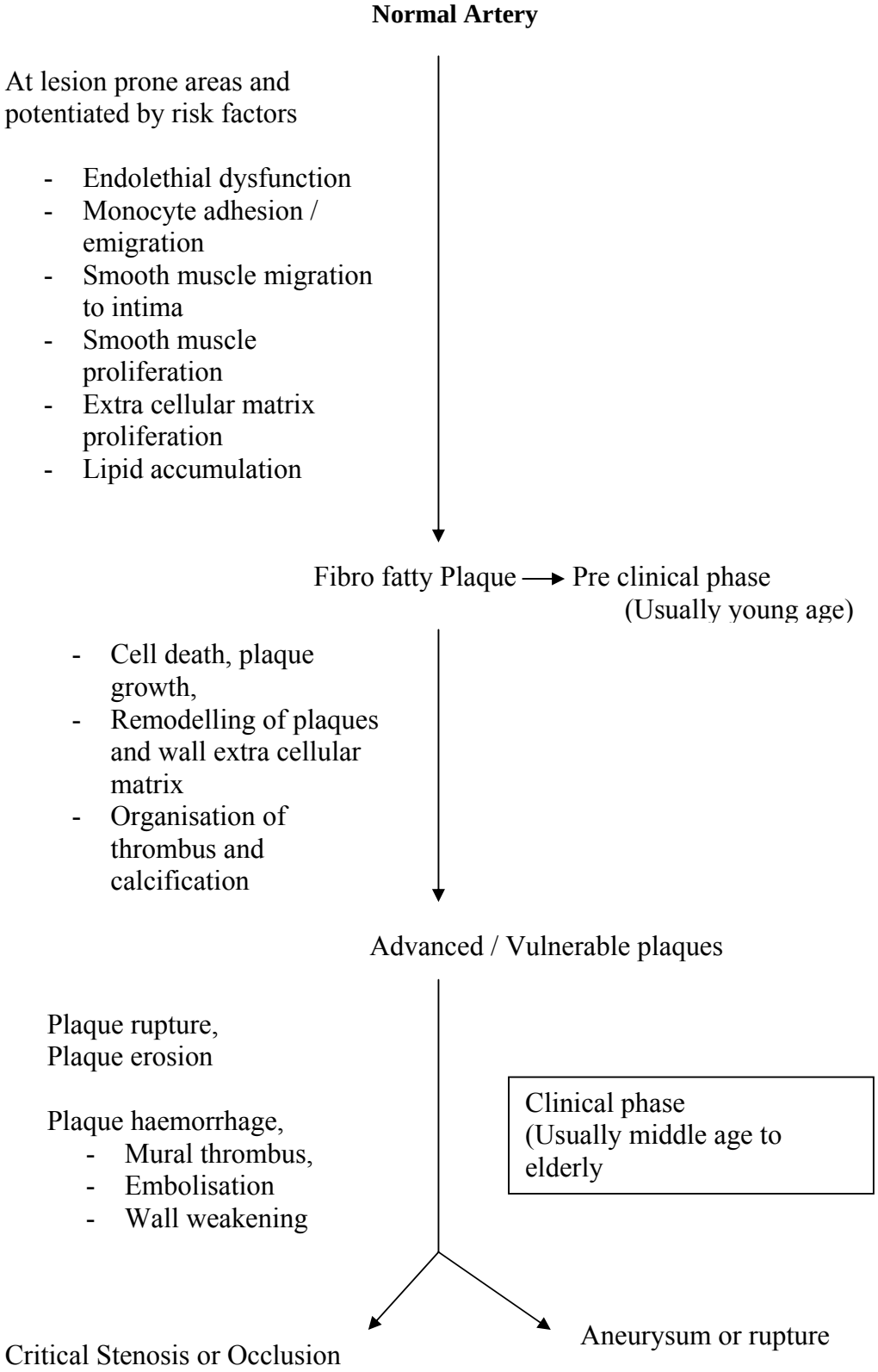
In contrast to LDL, cholesterol rich HDL particles protect against CAD. They are synthesized in the liver and intestine and also generated in part by lipolysis of chylomicron and VLDL. The actual function of HDL is to remove cholesterol from peripheral tissues and transport for hepatic excretion. This process is thought to be anti atherogenic consistent with the observation that raised levels of circulating HDL reduces the risk of CAD.

**Atheroscelorosis** :

This is a slowly progressive disease of arteries marked by elevated intimal fibrofatty plaques formed by lipid deposition smooth muscle cell proliferation and synthesis of extra cellular matrix. Large to medium sized muscular and large elastic arteries are involved principally in the abdominal aorta, coronary arteries, circle of Willis.

**Pathogenesis of Atherosclerosis** :

Historically two hypo thesis for atherogenesis were dominant one emphasized on cellular proliferation in intima, where as the other emphasized on organization and repetitive growth of thrombus. The contemporary view of pathogenesis of Atherosclerosis incorporates element of both theories and accommodate risk factors.




**Flow Chart**
Summary of Pathology, Pathogenesis, Complications and Natural history of Atherosclerosis

The concept called response to injury hypothesis[5], considers atherosclerosis to be a chronic inflammatory response of the arterial wall initiated by injury to endothelium. Central to this thesis

1) Chronic endothelial injury usually subtle with resultant endothelial dysfunction, yielding increased permeability leucocyte adhesion, thrombotic potential.
2) Insudation of lipo proteins into the vessel wall mainly LDL with its high cholesterol content.
3) Modification of lesional lipo protein by oxidation.
4) Adhesion of blood mono cytes to endothelium followed by their migration into the intima and their transformation into macrophage and foam cells.
5) Adhesion of platelets.
6) Release of factors from activated platelets and macrophages or vascular cells, that cause migration of smooth muscle cell from media to intima, proliferation of smooth muscle cell in the intima and elaboration of extra cellular matrix, leading to accumulation of collagen and proteaglycans.
7) Enhanced accumulation of lipids within cells and extra cellularly

**The role of Lipids in Atherosclerosis**

Dyslipoproteinemia results either from primary and secondary causes. The major evidence of implicating hypercholesterolemia in genesis of atherosclerosis includes following :

1) Genetic defect in lipo protein mechanism causing hyperlipo proteinemia are associated with accelerated atherosclerosis.

2) Other genetic factors or acquired disorders like diabetes mellitus, hypo thyroidisn, causes hypercholesterolemia leads to premature and severe atherosclerosis.
3) Major lipids in atheroma are plasma derived cholesterol and esters.
4) Epidemiological analysis demonstrate a significant correlation between the onset of atherosclerosis and levels of total cholesterol and LDL.
5) Lower levels of serum cholesterol by diet and drugs slows the rate of progression of atherosclerosis, causes regression of some plaques, reduces the risk of cardiovascular events.

**The Mechanism by which Dyslipoproteinemia contributes to Atherogenesis**

1. Chronic hyperlipidemia may directly impair endothelial cell function through increased production of oxygen free radicals, that deactivate nitric oxide, the major endothelial relaxing factor.
2. Chronic hyperlipidemia causes lipo protein accumulation within the intima and at the site of increased endothelial permeability.
3. Chemical change of lipid induced by free radicals generated by macrophage of endothelial cell in the arterial wall yield oxidized LDL. Oxidized LDL is
    a) Ingested by macrophages through the scavenger receptor distinct from LDL receptor forming foam cells
    b) Increased monocyte accumulation in lesions
    c) Stimulates release of growth factors and cytokines
    d) Cytotoxic to endothelial cells and smooth muscle cells.
    e) Can induced endothelial dysfunction

**Causes of Myocardial Infarction**

1. Atherosclerosis
2. Non Atherosclerotic causes

**Atherosclerosis and Myocardial infraction** :

Atherosclerotic lesions constitute fatty sheathes and fibrous plaques. The fibrous plaques constitute a central core of extra cellular lipid and necrotic cell debris covered by a fibro muscular layer containing large number of smooth muscle cells macropages and collagen. In the coronary arteries raised lesions are most prominent in the stem, the highest incidence being a short distance beyond the ostia and it is nearly always found in the extramural arteries while the intramural arteries are spared. Selective involvement of the coronary artery may relate to unique low dynamic forces resulting from greater flow in diastole than systole[6].

**Non- Atheroscelerotic Causes of CAD**

A) CAD other than Atherosclerosis

1) Arteritis
2) Trauma to coronary arteries
3) Coronary mural thickening with metabolic disease of intimal proliferative disease .
4) Luminal narrowing by other mechanisms
    - Spasm of Coronary Heart Disease
    - Dissection of Aorta, Dissection of Coronary Artery

**Emboli to Coronary Arteries**

- Infective endocarditis
- Non Bacterial thrombotic endocarditis
- Prolapse of mitral valve
- Mural thrombus from left atrium, left ventricle
- Cardiac myxoma
- Paradoxical emboli

**Congenital Coronary Artery Anomaly**

- Oxygen demand supply disproportion like Aortic stenosis

**Hematological**

- Polycythemia
- Thrombocytosis
- Disseminated intravascular coagulation
- Hypercoagulability states

**Miscellaneous**

- Cocaine abuse
- Myocardial contusion
- Complication of cardiac catheterization

**Risk Factors for Atherosclerosis**

It can be divided in to modified and non modified[7].

**Non modifiable (fixed)**

1. Age
2. Sex
3. Positive family history of CAD

**Modifiable**

1. Hypertension
2. Diabetes mellitus
3. Dyslipoproteinemia
4. Smoking
5. Obesity
6. Physical inactivity
7. Atherogenicdiet

**Emerging risk factor [8]**

Lipoprotein – a

Homocysteinuria

Prothrombotic factors

Pro inflammatory factors

Impaired fasting glucose

Sub clinical Atherogenesis

**1. Age**

Age has a dominant influence on the development of clinically significant atherosclerosis. The process is found to progress with age. In men age > 45 years, women age > 55 years are associated with increased risk of atherosclerosis. In MI less

than 40 years various studies have shown that the highest incidence occurred between 35 and 40 years.

**2. Sex**

CAD is a leading cause of death in males after 35 years and in females after 45 years. Various studies shows the males are increased risk of atherosclerosis after 55 years and in females after 65 years. In women it is due to hormonal protection offered by oestrogen.

**3. Family History of Premature CAD**

CAD often runs in families this may be due to genetic factors or the effects of shared environment. At present it is estimated that about 40% of the risk of developing IHD is condoled by genetic factors and 60% by environmental factors. Dyslipo proteinemia Hyper fibrinogenimia and abnormalities of other coagulation factors are often genetically determined. Family history of premature CAD in first degree in male relatives less than 55 years and in females first degree relatives less than 65 years are definite risk factor for atherosclerosis.

**4. Smoking**

A uniquely human habit smoking has been identified as major CAD risk factor with several possible mechanisms. Carbon monoxide induced atherogenesis. Nicotinic stimulation adrenergic drive risking both blood pressue and myocardial oxygen demand. Lipid metabolism with fall in 'Protective'. It has been calculated that in countries where smoking has been a wide spread habit, it is responsible for 25% of CAD deaths under 65 years of age in men.

Cigarattes seems to be particularly important causing sudden death from CAD, especially in men under 50 years of age.

The degree of risk of developing CAD is directly related to the number of cigarettes per day. Filter cigarettes are probably not protective. It is not only independent but also synergestic with other risk factors.

The risk of death from CAD decreases as cessation of smoking. The risk declines quite substantially within one year of stopping smoking and more gradually thereafter until after 10-20 years.

**5. Hypertension**

The Blood pressure is the single most useful test for identifying individual at a high risk of developing CAD. Hypertension accelerates that atherosclerotic process, especially if Dyslipoproteinemia is also present and contributes importantly to CAD. In the Framingham study, individual with blood pressure more than 160/195 mm Hg had a risk of coronary artery disease is five times more than in normotensive man. Hypertensive men and women are affected equally with the diastolic pressue perhaps more important.

In the MRFIT study increased death rates were associated with systolic Blood Pressure above 110 mm Hg and diastolic BP more than 70 mm Hg. Recent internventional studies show that reduction of diastolic Blood Pressure levels that have been greater than 105 mm Hg significantly reduced the incident of IHD. However various studies have shown that the incidence of hypertension in young myocardial infarction varied from 10-20%[9].

**6. Diabetes Mellitus**

In known diabetics both type 1 and type 2 there is atleast two fold increasing incidence of MI. Diabetes mellitus is a CAD risk equivalent, most patients with diabetes mellitus die of atherosclerosis and its complications. The abnormal lipoprotein profile associated with insulin resistance known as diabetic dyslipidemia accounts for part of the elevated cardiovascular risk in patients with type 2 diabetes mellitus. While diabetic patients often have LDL cholesterol near average, the LDL particles tend to be smaller and denser and thus more atherogenic. Other features of diabetic dyslipidemia include low HDL and TGL. Hypertension also frequently accompanies obesity insulin resistance and dyslipidemia. Indeed the NCEPATP III guidelines now recognize this cluster of risk factors and provide criteria for diagnosis of the metabolic syndrome. The role of glucose in atheroma formation is poorly understood. Sorbitol a product of insulin independent aldose reductase pathway of glucose metabolism (Polyol pathway) accumulates in the arterial wall in the presence of high glucose concentration. Glycosylated LDL can be formed which may be more susceptible to oxidation and more redily deliver cholesterol to arterial walls than native LDL

**7. Dyslipoproteinemia**

Although the term hyperlipidemia has long been used in clinical practice, the term dyslipoproteinemia more appropriately reflects the disorder of lipid and lipoprotein transport pathway associated with arterial disease. Abnormalities in plasmo lipo proteins and derangement in lipid metabolism rank as the most firmly established and best understood risk factors for atherosclerosis[10]. Current NCEPATP III guidelines recommend cholesterol screening in all adults > 20 years. The screen

should include fasting lipid profile (Total cholesterol, TGL, LDLC, and HDLC) repeated every 5 years.

NCEPATP III[11] Classification of Total, LDL and HDL cholesterol suggest optimal TC < 200 mg, with levels >240 mg as high, LDL cholesterol < 100 mg as optimal with values > 190 mg as very high risk, HDL cholesterol value < 40 mg as low value.

**Total Cholesterol (mg)**

| | | |
|---|---|---|
| < 200 | - | Desirable |
| 200 – 239 | - | Borderline high |
| > 240 | - | High |

**LDL Cholesterol (mg)**

| | | |
|---|---|---|
| < 100 | - | Optimal |
| 100 – 129 | - | Near Optimal |
| 130 – 159 | - | Borderline high |
| 160 – 189 | - | High |
| > 190 | - | Very high |

**HDL Cholesterol (mg)**

| | | |
|---|---|---|
| < 40 | - | Low |
| > 60 | - | High |

In the Framingham study, cholesterol men below 40 years were clearly related to the future development of IHD between 35-45 years of age serum

cholesterol of 265 mg or over are associated with 5 times higher risk of developing CAD than are levels below 220 mg. In the Multiple risk factor interventional trial – Men with increased cholesterol more than 240 mg had a three fold increase in IHD compared to men who had less than 200 mg. LDL is found to be more atherogenic than VLDL while. VLDL has also shown to be associated with prematured atherosclerosis, it is more strongly associated with peripheral vascular disease. HDL has a protective effect and the removes cholesterol from arterial walls. Dyslipoproteinemia[12] is found to rank second among the risk factors next to smoking among young MI patients.

Lipoprotein a has been established as an independent CAD risk factor the increased level of lipoprotein a is found to inhibit thrombolytic activity of naturally occurring tissue plasminogen activity and promote atherosclerosis.

**Classification of Dyslipoproteinemia**

It can be divided into primary and secondary

**TABLE - 1**
**Primary Classification (WHO)**

| Freidrickson type | Genetic classification | Defect | Clinical sequale |
|---|---|---|---|
| I | Lipoprotein Lipase deficiency Apo CII deficiency | Mutated or absent lipoprotein lipase or mutated lipoprotein lipase activator | Pancreatitis |
| II | Familial Hypercholestrolemia Apo B 3500 defect familial combained hyperlipidemia | Deficient LDL receptor binding, reduced LDL catabolism, over production of B 100 containing particles | CAD |
| III | Apo E2 homozygosity | Mutated Apo E | CAD and peripheral vascular disease |
| IV | Familial combined hyperlipidemia | Over production of B 100 Particles | CAD |
| V | Familial hypertryglyceridemia | Unknown | Pancreatitis |

**Secondary cause of Dyslipoproteinemia** [13]

| | | |
|---|---|---|
| Metabolic | - | Diabetic Mellitus |
| | - | Lipodystrophy |
| | - | Glycogen storage disorder. |
| Renal | - | Chronic renal failure, |
| | - | Glomerulolnephritis |
| Hepatic | - | Obstructive liver disease |
| | - | Cirrhosis |
| Hormone | - | Estrogen, Progesterone, growth hormone, hypo Thyroidism, Corticosteroids |
| Life style | - | Physical inactivity, obesity, diet rich in fats, and Saturated fats. |
| Medications | - | Retinoic acid derivatives, Glucocorticoids, anti viral Drugs etc. |

**Markers of Hyper Lipidemia**

1. Tendon Xanthomas : These are due to both intracellular and extra cellular deposits of cholesterol most commonly involved Achilles tendons and the extensor tendons of the knuckles and are found in 75% of adults with familial hyperlipoproteinemias.
2. Tuberous Xanthomas which are softer, painless nodules on the elbows and buttocks.
3. Xanthelasmas which are barely elevated deposits of cholesterol on the eye lids are more common in Heterogeneous familial hyperlipoprotinemias.
4. Eruptive Xanthomas
5. Xanthoma retinalis

**`TABLE - 2**

**SUMMARY OF MAJOR DRUGS USED FOR DYSLIPOPROTEINEMIA**

| Drug | Mechanism | Lipoprotein effect | Indication |
|---|---|---|---|
| HMG – coA reductase inhibitors<br>Lovastation<br>Pravastatin<br>Simvastatin<br>Atorvastatin<br>Rosuvastatin | ↓ Cholesterol Synthesis<br>↓ Hepatic LDL receptor<br>↓ VLDL production | LDL ↓ 18-55%<br>HDL ↑ 5-15 %<br>TGL ↓ 7-30 % | Elevated LDL |
| Bile acid sequestrants<br>Cholestyramine<br>Colestipol | ↑ Bile acid excretion,<br>↑ LDL receptor | LDL ↓ 15-30%<br>HDL ↑ 3-5 %<br>TGL no change | Elevated LDL |
| Nicotinic acid | ↓ VLDL hepatic synthesis | LDL ↓ 5-25%<br>HDL ↑ 15-35 %<br>TGL ↓ 20-50% | Elevated LDL,<br>Low HDL<br>Elevated TGL |
| Fibric Acid derivatives<br>Fino fibrate<br>Gem fibrozil | ↑ LPL ↓ VLDL Synthesis | LDL ↓ 5-20%<br>HDL ↑ 10-20 %<br>TGL ↓ 20-50% | Elevated TGL,<br>Elevated remnants |
| Cholesterol Absorption inhibitors | ↓ Intestinal cholesterol absorption | | Elevated LDL |

**Management of Dyslipoproteinemia**

According to NCEP – ATP III identify elevated LDL cholesterol as the primary target of cholesterol lowering therapy. Three categories of risk that modify LDL Cholesterol goals are :

1.CAD and CAD risk equivalent, - < 100 mg

2. Multiple 2+ risk factors-<130 mg

3. Zero to one risk factors - < 160 mg

For category one lifestyle changes at > 100 mg, Drug therapy at > 130 mg, category two lifestyle changes at >130 mg drug therapy at > 160 mg, for category three lifestyle changes at > 160 mg drug therapy > 190 mg.

**Elevated Triglycerides**

Two approaches lifestyle changes and drug therapy. First reduction of LDL cholesterol and addition of nicotinic acid or fibric acid.

**Lower HDL cholesterol**

In all patients with Low HDL cholesterol the primary target of therapy is LDL cholesterol and there is isolated low HDL cholesterol drugs for raising HDL cholesterol is considered and is mostly reserved for persons with CAD and CAD risk equivalent.

**8. Obesity**

It is the most common nutritional disorder in affluent societies. Its significance requires constant emphasis since it is associated with increased mortality. The presence of obesity detected in myocardial infarction patient less than 40 years ranged from 5-20%[14]. Central obesity was more important as it is associated with insulin resistance of peripheral tissues and compensatory Hyper insulinaemias, enhanced production of TGL and cholesterol rich proteins by liver. Obesity is related to atheroscelarosis both directly and via Hypertension Hyper Tryglyceridaemia, Hyper cholesterolaemia and hyper glycaemia. Obesity is assessed by bodymas index. BMI > 30 is considered as obesity. Obesity is assessed by waist circumference measurement. In Men > 40” and women > 35” indicates abdominal obesity.

**9. Physical Inactivity**

Among prospective studies, the Framingham data indicates that the less sedentary an individual, is the less susceptible to sudden death following CAD.

Exercise ameliorates the hyper lipidemia by increasing caloric expenditure[15]. Extensive clinical experience have revealed the importance of exercise affecting both left ventricular function and the atherosclerotic process and improving the prognosis after the coronary event. Physical activity increased the level of HDL.

**10. Personality A**

Type A behaviour is associated with competitive drive, restlessness, hospitality, and a sense of urgency or impatience. Type A individuals are more prone to CAD than calmer, more philosophical type B individual.

**11. Diet**

Diet rich in fat and cholesterol increases the chances of getting CAD Plasma cholesterol and LDL levels are sensitive to the amount of saturated fat and cholesterol in diet. The average cholesterol level in most closely related to the amount of animal fats (Meat, egg, milk). Decreased intake of complex carbohydrates and vegetable fibre leads to hyper cholesterolaemia. The dietary poly-unsaturated / saturated fat ratio is gaining importance. Low levels of Vitamin C, Vit.E and other anti oxidants enhance the prediction of oxidized LDL.

**Emerging Risk Factors**

A large body of literature suggest a relationship between hyperhomocysteinemia and CAD. Several mutations in their enzyme pathway causes accumulation of homocysteine which is associated with coronary risk and thrombosis.

An accumulation of clinical evidence shows that markers of inflammation correlates clinical risks. For example elevated C reactive protein and fibrinogen are associated with increased risk of CAD. One source of inflammatory stimulus could arise from infectious agent.

**Issue in Risk Assessment**

A growing panel of markers of coronary risk presents perplexing array to the practitioner. Markers measured in peripheral blood includes size fractions of LDL particles, concentration of homocysteine, LP(A), fibrinogen, C reative protein. In general such special test add little to the information available from careful history and physical examination, combined with measurement of plasma lipoprotein profile and fasting blood sugar. Similar concerns pertain to the use of specialized radio graphic estimation of coronary artery calcification.

**The challenge of Implementation**

We must learn how to help individuals adopt a healthy life style, and learn to deploy over increasingly powerful pharmacology tools most economically and effectively. The obstacles to implementation are economics education, physician awareness and patient adherence to recommended regime.

## Clinical presentation of myocardial infarction in the young patient

The clinical presentation of acute MI in young adults differs from their older counter parts. The classic presentation of worsening angina culminating in MI is rare in younger patient. The first onset of angina that rapidly progress to fully evolved MI is often the case in patient less than 40 years of age[16].

The prevalence of stable angina in young adults with reported coronary artery diseases was only 24% in one study[17]. In a series of patients who had their MI less than 40 years denied chest pain before MI[18]. The duration of symptoms was found to be less than a week in most of the patient. Careful history taking would be important clues as to the differential diagnosis of chest pain. In every young patient presenting with MI use of recreational drugs in the recent times should be recorded. Family history of premature CAD, smoking, obesity, diabetes and dyslipidemia[19] would give better clue to the likely hood of atheromatous coronary diseases. History of recurrent venous and arterial thrombosis should also be reported [20].

Initial clinical examination should concentrate on hemodynamic stability, evidence of sympathetic hyperactivity such as tachycardia and sweating, and evidence of previous injected drug used. Stigmata of dyslipidemia such as xanthelesma arcus senilis and tendon xanthomata should be looked for in every person the precordium is usually quiet and there may be abnormal systolic pulsations in the apical area, sign of ventricular dysfunction like S3 and S4, decreased intensity of heart sounds and an apical systolic murmur due to mitral regurgitation. There may be pericardial friction rub. Mild elevation of temperature and small drop in blood pressure may also be noted in some cases.

## Complication of myocardial infarction:

1. Rhythm disturbance-Sinus Brady cardia, AF, VF
2. Heart failure

3. Mitral Regurgitation
4. Pericarditis
5. Thromo embolism
6. Ventricular septal rupture
7. Post infarction extension
8. Dressler's syndrome
9. Ventricular aneurysm
10. Cardiac rupure

**Ventricular Function in myocardial infarction**

It has been found that after a brief episode of ischaemia, prolonged dysfunction with gradual return of contraction activity occurred, a condition termed myocardial stunning. It has become clear that chronically depressed myocardial function would be ameliorated, probably by relief of ischemia termed 'Myocardial hibernation'

**Systolic Dysfunction**

The fundamental pathological alteration underlying LV dysfunction in MI is loss of functioning myocardium. Cessation of blood flow to a region of mycaordium produces four sequential abnormal contraction pattern.

1. Dysynchrony – disassociation of the time course of contraction of adjacent segments of myocardium.
2. Hypokinesis – reduction in the extent of shortening
3. Akinesis – Cessation of shortening.
4. Dyskinesis – Paradoxial expansion and systolic bulging.

**Diastolic Dysfunction**

Ventricular diastolic properties are altered in infarction and ischaemic myocardium, leading initially to an increase but later to a reduction in left ventricular compliance. These changes are associated with an initial rise in Left Ventricle end Diastolic pressure over a period of two weeks. This pressure tends to fall towards normal.

**Right Ventricular infarction**

It generally associated with obstructive lesions of the right coronary artery. However it occurs less commonly than would be anticipated form the frequency of atherosclerotic lesions involving the right coronary artery. This discrepancy can be explained by the lower oxygen demands of the right ventricle. More over the inter coronary collateral system is richer and thinner of the right ventricular wall allows the chamber to derive some nutrition from the blood within its cavity.

**Ventricualr remodeling and infarct expansion**

As a consequence of myocardial infarction changes in left ventricular in left ventricular size, shape and thickness involving both the infarcted and non-infarcted segments of the ventricle often occur. These changes referred to as ventricular remodeling can influence LV function and prognosis. An increase in the size of the infarcted segments knows as ‘infarct expansion’ is defined as acute dilatation and thinning of the area of infarction explained by additional myocardial necrosis.

## MATERIALS AND METHODS

Seventy patients with established acute myocardial infarction in the age group of 40 years and below in Coimbatore medical college hospital admitted under cardiology unit and its corresponding medical units were studied during the period of January2005 to December 2005. All the cases were studied in detail from admission to discharge and followed regularly for one month after the admission. 30 healthy age and sex matched persons (controls) were studied simultaneously. Detailed history was taken from all the patients. Enquiries were made regarding the site, nature, duration of the chest pain, accompanying symptoms like breathlessness, vomiting, nausea, fatigue and palpitation.

The diagnosis of myocardial infarction was made by the classic world health organization (WHO)[21] criteria for the diagnosis of myocardial infarction which requires that at least two of the following three elements to be present, history of ischemic type chest discomfort, evolutionary changes on serially obtained ECG tracing, rise and fall in serum cardiac markers.

### On General Examination

Anaemia, Cyanosis, Sweating, Pulse, B.P, JVP, Height, weight in kgs, features of hyperlipidaemia like xanthalesma, Xanthoma, Pedal edema.

Complete systemic examination of CVS, RS, Abdomen and CNS done. Complete history about risk factors analysis was taken with regard to smoking, DM, family history of CAD, Hypertension, Hyper Lipidemia, Lifestyle and physical activity, obesity, or drug intake.

The BMI was calculated from the formula weight in Kgs/Height in $M^2$. The waist circumference is the minimum circumference measured between the costal margin and iliac crest and hip circumference over the buttocks.

**The following investigation were done for all patients.**

1. Urine – Albumin, Sugar, Deposits
2. Blood – Urea, Sugar, Lipid profile (Fasting)
3. Serum- Creatinine, CPK- MB [> 8 IU/dl is abnormal]
4. ECG
5. X-Ray Chest PA View
6. Echo Cardiography

Localisation of myocardial infarction from ECG [22]

Lead I, a VL, V1- V6- Extensive anterior

V1 – V6- Antero Lateral

V1-V4- Antero septal

Lead 1, a VL, V4-V6-Antero lateral

V5-V6 – Apical

Lead II, Lead III, a VF- Inferior wall

V3R, V4R – RV infarction

Echocardiography was done for all patients for assessing the ventricular function, regional wall motion abnormalities, presence of thrombosis and pericardial effusion. X-Ray PA View was taken to detect cardiomegaly features of pulmonary edema and pleural effusion.

**Lipid profile**

Lipid profile was done with standard procedure. Blood was drawn in the morning before breakfast (Fasting 12 hours). It is preferable to collect blood in tubes without anti coagulant (for serum) but it is possible to use tubes containing EDTA. The lipid fractions were done in the following methods [23].

**Total Cholesterol (SALKOWSKI'S METHOD)**

The principle of this reaction is that cholesterol reacts with ferric chlorideions in acetic acid followed by sulphuric acid. This method is modified by 'WYBENGA' and this method is used to estimate total cholesterol level.

**HDL Cholestrol (Loper and Virella)**

In this procedure, the VLDL, Chylomicrons, LDL were precipitated by phosphotungstate in the presence of Mg+ ions and the cholesterol is estimated from the supernatant. All measurements were taken using highly sophisticated modulab system of Boheinger Knoll West Germany.

**Triglycerides (Foster and Dunn Hantzsh Reaction)**

In this reaction TGL are extracted by Haptane and Saponified by potassium hydroxide. The glycerol is oxidized to formaldehyde which combines with acetyl acetone in the presence of ammoniumions to give a dihydrobutidine derivative which is measured calorimeterically at 415nm.

The LDL cholesterol is calculated from the above lipid profile values using the formula – Friedwald.

Totalcholesterol – HDL Cholesterol – TGL/5=LDL Cholesterol

**Test of Significance**[24]

1. Normal deviate or Critical Ratio

$$\text{ND} = \frac{X - \mu}{\sigma / \sqrt{n}}$$

ND – Normal Deviate

X – Character of Variable Tested in Sample

$\mu$ - Character of Variable Tested in Population

$\sigma / \sqrt{n}$ - Standard Error of Sample Character

If – Normal Deviate > 2 indicates P < 0.05, test is significant

2. Standard Error of Difference between two proportions

$$\text{SE} = \sqrt{\frac{p1q1}{n1} + \frac{p2q2}{n2}}$$

p1 - % of patients with risk factor

q1 - % of patients of without risk factor

p2 - % of controls with risk factor

q2 - % of controls without risk factor

n1 – Number of study population

n2- Number of control population

If observe difference between the two groups is more than twice that of standard error, the test is significant.

3. Standard Error of Difference between two means

S.E $\sqrt{\frac{\sigma 1^2}{n1} + \frac{\sigma 2^2}{n2}}$

$\sigma 1$ - Standard Deviation of Study population

n1 – Number of study population

$\sigma 2$ - Standard Deviation of control population

n2 – Number of control population

If the actual difference between the two means is more than twice that of standard error of difference between, two means then the test is significant.

## RESULTS AND OBSERVATIONS

The results and observations of the study were presented below

### Age Distribution

The mean age of study group was 35.271 from 20-40 years. The mean age of control group was 35-64 ranging from 22-40 years. The pattern of distribution was

**Table - 3**

**Age Distribution**

| | Patient | |
|---|---|---|
| Age in years | Number | Percentage |
| <20 | - | - |
| 20-30 | 10 | 14.28 |
| 31-35 | 20 | 28.56 |
| 36-40 | 40 | 57.14 |

Most of the cases were in age group of 31-40 years (85.64%)

### Sex Distribution

Among 70 patient 68 were males and 2 were females.

**Table - 4**

**Sex Distribution**

| | Patient | |
|---|---|---|
| Sex | Number | Percentage |
| Male | 68 | 97.15 |
| Female | 2 | 2.85 |

**Anthropometry**

The mean height of our study cases were 166.43cm and weight 67.16kg. The mean height of control group were 166.2cm and weight was 64.44kg. The mean body mass index of the study cases was 24.27 and of control was 23.26

**Incidence of Smoking**

According to the number of beedies, cigarettes smoked per day they are divided in to 3 groups

**Table - 5**

**Incidence of smoking**

| | Patient | |
|---|---|---|
| Cigarettes or beedies per day | Number | Percentage |
| None | 49 | 70 |
| <20 | 12 | 17.15 |
| >20 | 9 | 12.85 |

All the smokers were male, smokers form 30% among study group. The duration of smoking was more than 10-15 years

**Incidence of obesity**

There were about 28 patient who had a body mass index more than 30

**Table - 6**

**Incidence of obesity**

| | Patient | |
|---|---|---|
| Body mass index | Number | Percentage |
| >30 | 28 | 40 |

**Incidence of Hypertension**

There were 8 cases established Hypertension among the study cases who were on treatment

**Table - 7**

**Incidence of Hypertension**

| No of cases | Percentage |
|---|---|
| 8 | 11.42 |

**Incidence of Diabetes Mellitus**

There were about 12 cases of established diabetes mellitus among study group.

**Table -8**

**Incidence of Diabetes Mellitus**

| No of cases | Percentage |
|---|---|
| 12 | 17.14 |

**Family History of CAD**

About 21 of the study group had family history of MI among blood relatives

**Table - 9**

**Incidence of Family History of CAD**

| No of cases | Percentage |
|---|---|
| 21 | 30 |

**Sedentary Life style**

Among the study group 18 of the cases were in sedentary life style

**Table - 10**

**Incidence of Sedentary Life Style**

| No of cases | Percentage |
|---|---|
| 18 | 25.7 |

## LIPID PROFILE

### a. Total Serum Cholesterol

The mean total serum cholesterol of study group was 217.2mg and that of control was 177.46mg

**Table - 11**

**Mean total Serum Cholesterol**

| Study Group in mg% | Control group in mg% |
|---|---|
| 217.2 | 177.46 |

The standard error of difference between two means of two groups was 15. The observed difference between two means was more than twice that of standard error of difference between means of two groups there fore the result was significant .

### b. Incidence of Hypercholesterolemia

There is about 50% increase in the level of cholesterol > 200 mg in the study group, compared to about 26.66 % in control.

**Table - 12**

**Incidence of Hypercholesterolemia**

| Study Group | | Control group | |
|---|---|---|---|
| Number | Percentage | Number | Percentage |
| 35 | 50 | 8 | 26.66 |

Normal deviate is 2.4, it implies $P<0.05$, there is significant incidence of Hypercholesterolemia in study population. Standard error of difference between 2

proportions is 10.03, the observed difference is more than twice that of standard error of difference, there fore the result was significant.

**c. Serum Low Density Lipoprotein Cholesterol**

The mean level of LDL-C in study group was 132 mg and the control group was 1.5 mg.

**Table - 13**

**Mean Level of LDL Serum Low Density Lipoprotein Cholesterol**

| Study Group in mg % | Control group in mg% |
|---|---|
| 132 | 105 |

The standard error of difference between two means of two groups was 10. The observed difference between two means was more than twice that of standard error of difference between means of two groups there fore the result was significant.

**d. Incidence Of High Risk Low Density Lipoprotein Cholesterol**

There is about 35.71 % increase in the level of LDL cholesterol > 130 mg in the study group, compared to about 23 % in control.

**Table - 14**

**Incidence of High Risk Low Density Lipoprotein Cholesterol**

| Study Group | | Control group | |
|---|---|---|---|
| Number | Percentage | Number | Percentage |
| 25 | 35.7 | 5 | 16.6 |

Normal deviate is 2.6, it implies P<0.05, there is significant incidence of High Risk LDLC in study population. The standard error of difference between two proportions was 8.88, the observe difference is more than twice that of standard error of difference, there fore the result was significant.

**e. Serum Triglycerides**

The mean level of TGL in study group was 167.5 mg and the control group was 110 mg.

**Table - 15**

**Mean Level of Triglycerides**

| Study Group in mg% | Control group in mg% |
|---|---|
| 167.2 | 125.65 |

The standard error of difference between two means of two groups was 15. The observed difference between two means was more than twice that of standard error of difference between means of two groups there fore the result was significant.

**f. Incidence of Hyper Triglyceridemia**

There is about 47.14 % increase in the level of TGL in the study group, compared to about 26.6 % in control.

**Table - 16**

**Incidence of Hyper Triglyceridemia**

| Study Group | | Control group | |
|---|---|---|---|
| Number | Percentage | Number | Percentage |
| 33 | 47.14 | 8 | 26.6 |

Normal deviate is 2.8, it implies P<0.05, there is significant incidence of Hyper Triglyceridemia in study population. The standard error of difference between two proportion is 10.03, the observed difference was more than twice that of standard error of difference between two proportions, indicating the result was significant.

**g. High Density Lipoprotein Cholesterol**

The mean level of HDLC in study group was 50.9 mg and the control group was 50 mg.

**Table -17**

**Mean Level of High Density Lipoprotein Cholesterol**

| Study Group in mg% | Control group in mg% |
|---|---|
| 50.9 | 47 |

There is no significant difference between the means of 2 groups.

**h. Incidence of Highrisk High Density Lipoprotein Cholesterol**

There is about 40 % increase in the level of High Risk HDLC in the study group, compared to about 20 % in control.

**Table - 18**

**Incidence of High Risk HDLC**

| Study Group | | Control group | |
|---|---|---|---|
| Number | Percentage | Number | Percentage |
| 28 | 40 | 6 | 20 |

Normal deviate is 2.6, it implies P<0.05, there is significant incidence of Highrisk HDLC in study population. The standard error of difference between two proportion was 9.36, the observe difference was more than twice that of standard error of difference between two proportions, indicating the result was significant.

**Table - 19**

**Diabetis Mellitus and Lipid Profile**

| | **No of Patients** | **Total Cholesterol (Mean mg/dl)** | **HDLC Mean Mg/dl** | **LDLC Mean Mg/dl** | **TGL Mean Mg/dl** |
|---|---|---|---|---|---|
| With Diabetes | 12 | 229 | 57.5 | 137.41 | 173.7 |
| Without Diabetes | 58 | 213 | 53.2 | 126.5 | 166.5 |

**Table - 20**

**Hypertension and Lipid Profile**

| | **No of Patients** | **Total Cholesterol (Mean mg/dl)** | **HDLC Mean Mg/dl** | **LDLC Mean Mg/dl** | **TGL Mean Mg/dl** |
|---|---|---|---|---|---|
| With Hypertension | 8 | 258 | 62 | 153 | 200 |
| Without Hypertension | 62 | 210 | 53 | 125 | 163 |

**Table - 21**

**Smoking and Lipid Profile**

| | **No of Patients** | **Total Cholesterol (Mean mg/dl)** | **HDLC Mean Mg/dl** | **LDLC Mean Mg/dl** | **TGL Mean Mg/dl** |
|---|---|---|---|---|---|
| With Smoking | 21 | 225 | 58 | 123 | 175 |
| Without Smoking | 49 | 212 | 52 | 130 | 165 |

**Table - 22**

**Obesity and Lipid Profile**

| | No of Patients | Total Cholesterol (Mean mg/dl) | HDLC Mean Mg/dl | LDLC Mean Mg/dl | TGL Mean Mg/dl |
|---|---|---|---|---|---|
| With Obesity | 28 | 212 | 46.2 | 133 | 162 |
| Without Obesity | 42 | 218 | 59 | 125 | 175 |

**Table - 23**

**Sedentary Life Style and Lipid Profile**

| No of Patients | Total Cholesterol (Mean mg/dl) | HDLC Mean Mg/dl | LDLC Mean Mg/dl | TGL Mean Mg/dl |
|---|---|---|---|---|
| 18 | 282 | 63 | 149 | 198 |

**Table - 24**

**Family History of CAD and Lipid Profile**

| No of Patients | Total Cholesterol (Mean mg/dl) | HDLC Mean Mg/dl | LDLC Mean Mg/dl | TGL Mean Mg/dl |
|---|---|---|---|---|
| 21 | 236 | 56 | 142 | 167 |

**Anatomical Distribution of Myocardial Infarction**

| AREA INVOLVED | NO. | PERCENTAGE |
|---|---|---|
| Extensive Anterior wall | 8 | 11.4 |
| Antero Septal | 20 | 28.5 |
| Antero Lateral | 10 | 14.3 |
| Inferior Wall | 18 | 25.7 |
| Inferior Wall with RV Extension | 6 | 8.6 |
| True Posterior wall | 2 | 2.85 |
| Anterior and Inferior wall | 6 | 8.6 |

**Table - 25**

**Incidence of ST Elevation MI and Non ST Elevation MI**

| Type | Number | Percentage |
|---|---|---|
| ST Elevation | 67 | 96 |
| Non ST Elevation | 3 | 4 |

**Complications During Hospital Stay**

| **Complications** | **No.** | **Percentage** |
|---|---|---|
| 1. Rythm Disturbance | | |
| Ventricular Premature beats | 12 | 17 |
| Sinus Bradycardia | 10 | 14 |
| Left bundle branch block | 6 | 8.5 |
| 2. Left Ventricular failure | 18 | 25.5 |
| 3. Death Mortality | 3 | 4 |

**Echo Cardio Gram**

All patients were subjected to Echo. About 25 patients shows left ventricle systolic dysfunction (35.7%) left ventricular clot 2 patients (3%).

**X-ray Chest PA View**

X- ray Chest PA View was taken to all patients for evaluation of Cardiomegaly and features of congestive cardiac failure. Only 7 patients showed features of failure.

# DISCUSSION

The conquest over infectious disease have enhanced the importance of preventive cardiology, because of the increasing size of adult population and higher incidence of MI in the 4th decade and beyond. The risk factors includes increasing age male sex, Dyslipoproteinemia, Hypertension, Diabetes Melitus, Smoking, Obesity, Family History of CAD and lack of physical inactivity.

Our hospital incidence shows that there is steady increase in MI altogether, especially among young adults of 40 years and below.

## Age Incidence

The maximum incidence of MI is between 36 to 40 years. The data is comparable to the study conducted by K.N Pradeep et al [25] and S. Tewari et al [26].

**Table - 26**

**Age Incidence**

| Series | Year | Mean Age |
|---|---|---|
| K.N. Pradeep et al | 2004 | 36.8 |
| S. Tewari et al | 2005 | 37.4 |
| Present | 2005 | 35.271 |

## Sex Incidence

Among 70 cases studied only two are females. The sex incidence of male and female were 97.15 : 2.85, which was comparable to other studies conducted in India by Chandurkar[27], B.L. Agarwal[28], S. Tewari.

**Table - 27**

**Sex Incidence**

| Series | Year | Male Percentage | Female Percentage |
|---|---|---|---|
| Chandurkar | 1971 | 95.2 | 4.8 |
| B.L. Agarwal | 1978 | 93.5 | 6.5 |
| S. Tewari | 2005 | 92 | 8 |
| Present | 2005 | 97.15 | 2.85 |

**Diabetes Melitus**

There were 12 cases of established diabetis melitus who were taking treatment in study group. This was comparable to the studies conducted by S. Sharma et al [29], S. Ravishankar et al [30] on young MI patients in India.

**Table - 28**

**Incidence of Diabetes Melitus**

| Series | Year | Male Percentage | Female Percentage |
|---|---|---|---|
| Sharma et al | 2002 | 12 | 20% |
| S. Ravisankar et al | 2002 | 23 | 22% |
| Present | 2005 | 12 | 17.14 |

Overall diabetes melitus did not formed major risk factor for young MI.

**Hypertension**

There were eight patients with established systemic hypertension. The incidence of hypertension is 11.42% in the present study which is comparable to other studies conducted in India by Chandurkur et al, S. Sharma et al, J. Ismail et al[31], in

young MI patients. This study shows that hypertension does'nt appears to be primary cause of advanced atherosclerosis in the population of young.

**Table - 29**

**Incidence of Hypertension**

| Series | Year | No. | Percentage |
|---|---|---|---|
| Chandurkur et al | 1971 | 9 | 14.5 |
| S. Sharma et al | 2002 | 9 | 15 |
| Ismail et al | 2004 | 20 | 14 |
| Present | 2005 | 8 | 11.42 |

**Smoking**

There were about 21 patients of smokers in the present study. This was comparable to other studies conducted by D.K. Baruah et al[32], Gurupar Singh et al[33], K.N. Pradeep et al, on young MI patients.

**Table - 30**

**Incidence of Smoking**

| Series | Year | No. | Percentage |
|---|---|---|---|
| D.K. Baruah et al | 2004 | 89 | 32.2 |
| Gurupar Singh et al | 2002 | 20 | 40 |
| K.N. Pradeep et al | 2004 | 19 | 30 |
| Present | 2005 | 21 | 30 |

**Obesity**

The number of obese patients in the study group was 38. This was comparable to other studies conducted by K.N. Pradeep et al and K.W. Alwash et al [34].

**Table - 31**

**Incidence of Smoking**

| Series | Year | No. | Percentage |
|---|---|---|---|
| K.N. Pradeep et al | 2004 | 22 | 34 |
| K.W. Alwash et al | 2003 | 61 | 28 |
| Present | 2005 | 28 | 40 |

**SERUM LIPID PROFILE**

**a. Serum Mean Total Cholesterol**

The Mean Serum Total Cholesterol was significantly elevated in the study group when compared to controls. This values are comparable to studies conducted by J.K. Mishra et al [35], and Santanu Guha et al [36].

**Table - 32**

**Serum Mean Total Cholesterol**

| Series | Year | Study group mg%. | Control group mg%. |
|---|---|---|---|
| J.K. Mishra et al | 2001 | 224 | 180 |
| Santanu Guha et al | 2001 | 216 | 185 |
| Present | 2005 | 217.2 | 177.46 |

These values were also comparable to other studies conducted on young MI patients by Nevas et al [37], and Corvilan. B et al [38],

**b. Incidence of Hyper Cholesterolemia**

There is significant increase in the incidence of patients with High Risk Cholesterol ($P < 0.05$) which was comparable to the studies conducted by Rajeev Gupta et al [39], S. Tewari et al, on young MI patients.

**Table - 33**

**Incidence of Hyper Cholesterolemia**

| Series | Year | Study Group | | Control Group | |
|---|---|---|---|---|---|
| | | No | Percentage | No | Percentage |
| Rajeev Gupta et al | 2001 | 253 | 48.8 | 100 | 15 |
| S. Tewari et al | 2005 | 44 | 40 | 12 | 12 |
| Present | 2005 | 35 | 50 | 8 | 26.66 |

In over 20 prospective studies in difference countries, total serum cholesterol level has been shown to be related to the development of CAD as in Multiple Risk Factor, Intervention trials (MRFIT) by Stamler J et al [40]. In the study group the incidence of Hyper Cholesterolemia was noted in 50 % thus there is significant elevation of total cholesterol in study group when compared to controls, which is comparable to other studies conducted by Chowdry et al [41] on MI in young patients.

**c. Serum Low Density Lipoprotein Cholesterol**

The mean level of High Risk LDLC in the study group was 132 mg which is significantly elevated then control, which was 98 mg. This increased level of LDLC was comparable to other studies conducted by Jacob V Jose et al [42] and S. Tewari et al.

**Table - 34**

**Mean Serum Low Density Lipoprotein Cholesterol**

| Series | Year | Study group mg% | Control group mg% |
|---|---|---|---|
| Jacob V Jose et al | 2004 | 134 | 100 |
| S. Tewari et al. | 2005 | 121 | 96 |
| Present | 2005 | 132 | 105 |

Increased LDLC is Atherogenic which was proved in many studies like studies conducted by Kwame O Alwash et al and Rodney G Bowden et al [43].

**d. Incidence of High Risk Low Density Lipoprotein Cholesterol**

There is significant increase in the incidence of High Risk LDLC ($P<0.05$) which is comparable to other studies conducted by Jacob V Jose et al, S. Tewari et al, UHL GS et al, M Egred [44] on studies of Risk Factors in young MI.

**Table - 35**

**Incidence of High Risk Low Density Lipoprotein Cholesterol**

| Series | Year | Study Group | | Control Group | |
|---|---|---|---|---|---|
| | | No | Percentage | No | Percentage |
| Jacob V Jose et al | 2004 | 121 | 41 | 80 | 20 |
| S. Tewari et al | 2005 | 45 | 41 | 20 | 21 |
| Present | 2005 | 25 | 35.71 | 5 | 16.6 |

**e. Serum Triglycerides**

The Mean Serum Triglycerides is significantly elevated in study groups comparing to control, which is comparable to other studies conducted by Issar et al [45], Jacob V Jose et al, G. Morgan et al [46], Zimmerman FH et al[47],

**Table - 36**

**Mean Serum Triglycerides**

| Series | Year | Study group mg% | Control group mg% |
|---|---|---|---|
| Issar et al | 2001 | 202 | 148 |
| Jacob V Jose et al | 2004 | 142 | 120 |
| Present | 2005 | 167.5 | 125.65 |

**f. Incidence of Hyper Triglyceridemia**

There is significant increase in the incidence of High Risk TGL (47.14%, P<0.05) in the study group, which was comparable to studies conducted by Jacob V Jose et al, S. Tewari et al, A.L. Yusuf A.R.[48] and curren PJ et al [49].

**Table - 37**

**Incidence of Hyper Triglyceridemia**

| Series | Year | Study Group | | Control Group | |
|---|---|---|---|---|---|
| | | No | Percentage | No | Percentage |
| Jacob V Jose et al | 2004 | 127 | 43.8 | 74 | 30 |
| S. Tewari et al | 2005 | 45 | 41 | 24 | 32 |
| Present | 2005 | 33 | 47.14 | 8 | 26.6 |

**g. High Density Lipoprotein Cholesterol**

The level of HDLC < 40 mg is definite Risk Factor for Atherosclerosis however in our present study there is no significant reduction in HDLC. But in studies conducted by Issar et al, S. Tewari et al, Showed reduced HDLC.

**Table - 38**

**Mean High Density Lipoprotein Cholesterol**

| Series | Year | Study group mg% | Control group mg% |
|---|---|---|---|
| Issar et al | 2001 | 30.16 | 46.8 |
| S. Tewari et al | 2005 | 33 | 60 |
| Present | 2005 | 50.9 | 47 |

**h. Incidence of High Risk High Density Lipoprotein Cholesterol**

There is significant increase in the incidence of High Risk HDLC in the study group 40% ($P<0.05$) which was comparable studies conducted by S. Tewari et al Usula S. et al [50], Siwash et al [51].

**Table - 39**

**Incidence of High Risk High Density Lipoprotein Cholesterol**

| Series | Year | Study Group | | Control Group | |
|---|---|---|---|---|---|
| | | No | Percentage | No | Percentage |
| S. Tewari et al | 2005 | 69 | 62.7 | 32 | 40 |
| Present | 2005 | 28 | 40 | 6 | 20 |

**Family History of Coronary Artery Disease**

Family History of CAD forms a significant Risk Factor in the development of early atherogenisis and MI in the present study the incidence was 30% which was comparable to other study conducted by Issar et al.

**Table - 40**

**Incidence of Family History of CAD**

| Series | Year | No. of Cases | Percentage |
|---|---|---|---|
| Issar et al | 2001 | 12 | 24 |
| Present | 2005 | 21 | 30 |

**Sedentary Life Style**

The incidence of sedentary life style was 27% in the present study which was comparable to study conducted by Chadurkur et al, on MI in young.

**Table - 41**

**Incidence of Sedentary Life Style**

| Series | Year | No. of Cases | Percentage |
|---|---|---|---|
| Chadurkur et al | 1971 | 20 | 28.2 |
| Present | 2005 | 18 | 27 |

**Diabetes Melitus and Lipidprofile**

The pattern of Lipidprofile in Diabetic patients in the present study was

**Table - 42**

**Diabetes Melitus and Lipidprofile**

| No. of patients | Percentage | Total Cholesterol Mg. | TGL Mg | HDLC Mg | LDLC Mg |
|---|---|---|---|---|---|
| 12 | 17.14 | 229 | 173 | 57.5 | 137.41 |

This is comparable to study conducted by Vusal H et al [52], which shows Serum Cholesterol – 233 mg, TGL – 173 Mg, HDLC – 38 mg, LDLC – 137 mg. There is increase incidence of Hypercholesterolemia, Hypertriglyceridemia, increased level of

LDLC as shown in study conducted by Mohamed Ishak et al [53] on Hyperliipidemia in Diabetis Melitus.

**Hyper Tension and Lipidprofile**

The pattern of lipid profile in the present study with hypertensive patients was

**Table - 43**

**Hyper tension and lipid profile**

| No. of patients | Percentage | Total Cholesterol Mg. | TGL Mg | HDLC Mg | LDLC Mg |
|---|---|---|---|---|---|
| 8 | 11.42 | 258 | 200 | 62 | 153 |

There is increased incidence of elevated total cholesterol, TGL, LDLC. It is comparable to study conducted by Malhotra et al [54], Isolated lipid abnormalities in rural and urban hypertensive patients in north India and study conducted by AB Bhavani et al [55] on lipid profile and apolipoprotein E polymorphism on essential hypertension.

**Smoking and Lipidprofile**

The pattern of lipid profile in the present study with smoking in the presence study was

**Table - 44**

**Smoking and Lipidprofile**

| No. of patients | Percentage | Total Cholesterol Mg. | TGL Mg | HDLC Mg | LDLC Mg |
|---|---|---|---|---|---|
| 21 | 30 | 225 | 175 | 58 | 123 |

There is increased incidence of elevation total cholesterol TGL and low density lipoprotein cholesterol. In the study conducted by Ns Neki et al [56], lipid profile in chronic smokers, shows increased incidence of total cholesterol (181 mg) Triglycerides (173mg), Low density lipoprotein cholesterol (103mg) when compared to control. It is also comparable other studies conducted by Restogi et al [57], On lipid profile in smokers and Muscat je et al [58] on cigarettes smoking and plasma cholesterol.

**Obesity and Lipidprofile**

The pattern of lipid profile in the present study shows elevated total cholesterol, TGL and LDLC.

**Table - 45**

**Obesity and Lipidprofile**

| No. of patients | Percentage | Total Cholesterol Mg. | TGL Mg | HDLC Mg | LDLC Mg |
|---|---|---|---|---|---|
| 28 | 40 | 212 | 162 | 46.2 | 133 |

The pattern was seen in studies conducted by Abdulhamid et al [59] which shows total cholesterol 215mg, TGL- 189 mg, LDLC-135mg. It was also comparable to the study conducted by DK Baruah et al

**Sedentary Lifestyle and Lipidprofile**

There is significant increase in total cholesterol, TGL and LDLC in this group

**Table - 46**

**Sedentary Life Style and Lipid Profile**

| No. of patients | Percentage | Total Cholesterol Mg. | TGL Mg | HDLC Mg | LDLC Mg |
|---|---|---|---|---|---|
| 18 | 27 | 282 | 198 | 63 | 149 |

It was comparable to the study conducted by Rodney G Bowden et al on lipid level of sedentary university students.

**Family History of CAD and Lipidprofile**

There is significant increase in total cholesterol, TGL and LDLC in this group

**Table - 47**

**Family History of CAD and Lipidprofile**

| No. of patients | Percentage | Total Cholesterol Mg. | TGL Mg | HDLC Mg | LDLC Mg |
|---|---|---|---|---|---|
| 21 | 30 | 236 | 167 | 56 | 142 |

This was compared to study conducted by Chandurkar et al which was shows similar pattern

**Anatomical Distribution of Myocardial Infarction**

The pattern of Anatomical distribution of myocardial infarction in the presence study shows most of the involvement of anterior wall, which is comparable to the study conducted by S. Sharma et al which shows anterior wall 35 patients (58.3%), Inferior wall 23 patient (38.3%), Right ventricle 2 patients (3 %)

**Table -48**

**Anatomical distribution of myocardial infarction**

| Area involved | Number | Percentage |
|---|---|---|
| Extensive Anterior wall | 8 | 11.4 |
| Anteroseptal wall | 20 | 28.5 |
| Anterolateral wall | 10 | 14.3 |
| Inferior wall | 18 | 25.7 |
| Inferior wall and right ventricle | 6 | 8.6 |
| True posterior wall | 2 | 2.85 |
| Anterior and inferior wall | 6 | 8.6 |

**ST Elevation and non ST elevation Myocardial Infarction**

Most of the cases were ST elevation myocardial infarction in the present study (96%)

**Table - 49**

**ST Elevation and Non ST Elevation Myocardial Infarction**

| Type | Number | Percentage |
|---|---|---|
| ST Elevation | 67 | 96 |
| Non ST Elevation | 3 | 4 |

This is comparable to the study conducted by CVN Murthy et al [60] which shows ST elevation MI incidence of 93%

**Mortality**

In the presence study 3 cases died of severe left ventricular dysfunction (4%) which was comparable to the study conducted by Chandurkar et al which shows mortality of 4 cases (6.7%)

# CONCLUSION

There was significant incidence of dyslipoproteinemia in the form of elevation of  Incidence of elevated serum  total cholesterol, low density lipoprotein cholesterol, Triglycerides and elevation in the incidence of high risk high density lipoprotein cholesterol in patients of 40 years and below with acute myocardial infarction in the study population, when compared to matched controls.

# SUMMARY

Seventy cases of proved myocardial infarction in patients with 40 years and below were studied. Controls were studied simultaneously.

The male to female ratio was 68:2 in the study group and all the controls were males.

Age group ranged from 19 years to 40 years of which maximum incidence was noticed between 35-40 years.

12 percent of the study group were smokers, 40 percent were Obese, 11 percent had hypertension, 17 percent were diabetic mellitus 30 percent had family history of CAD.

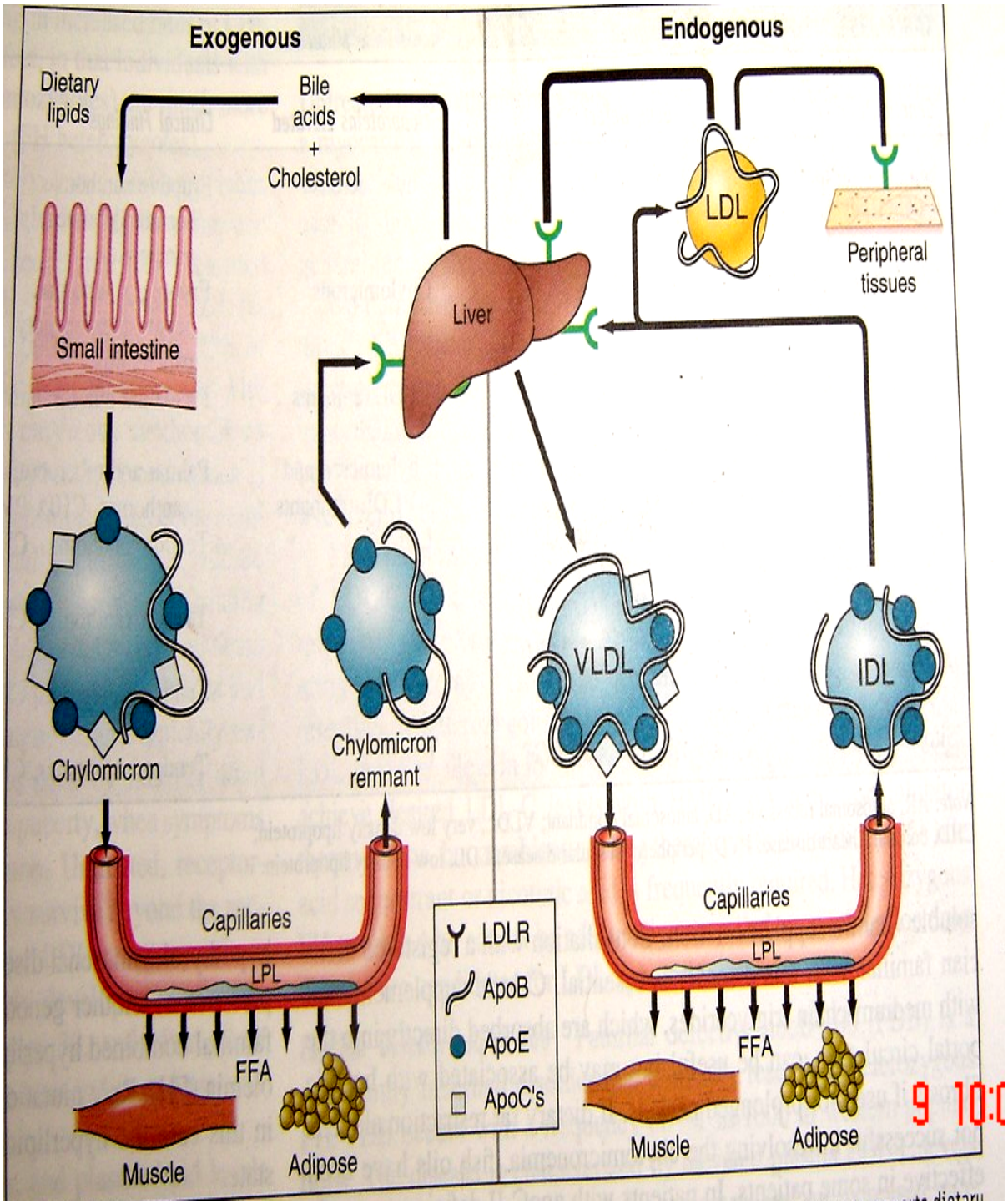
Mean Serum cholesterol, TGL, LDLC were elevated in the study group when compared to controls.

There were 50% incidence of hypercholesterolemia , 35.7% incidence of elevated LDLC, 47.14% incidence of elevated TGL, 40% incidence of high risk HDLC in the study group, which all were significant when compared to controls.

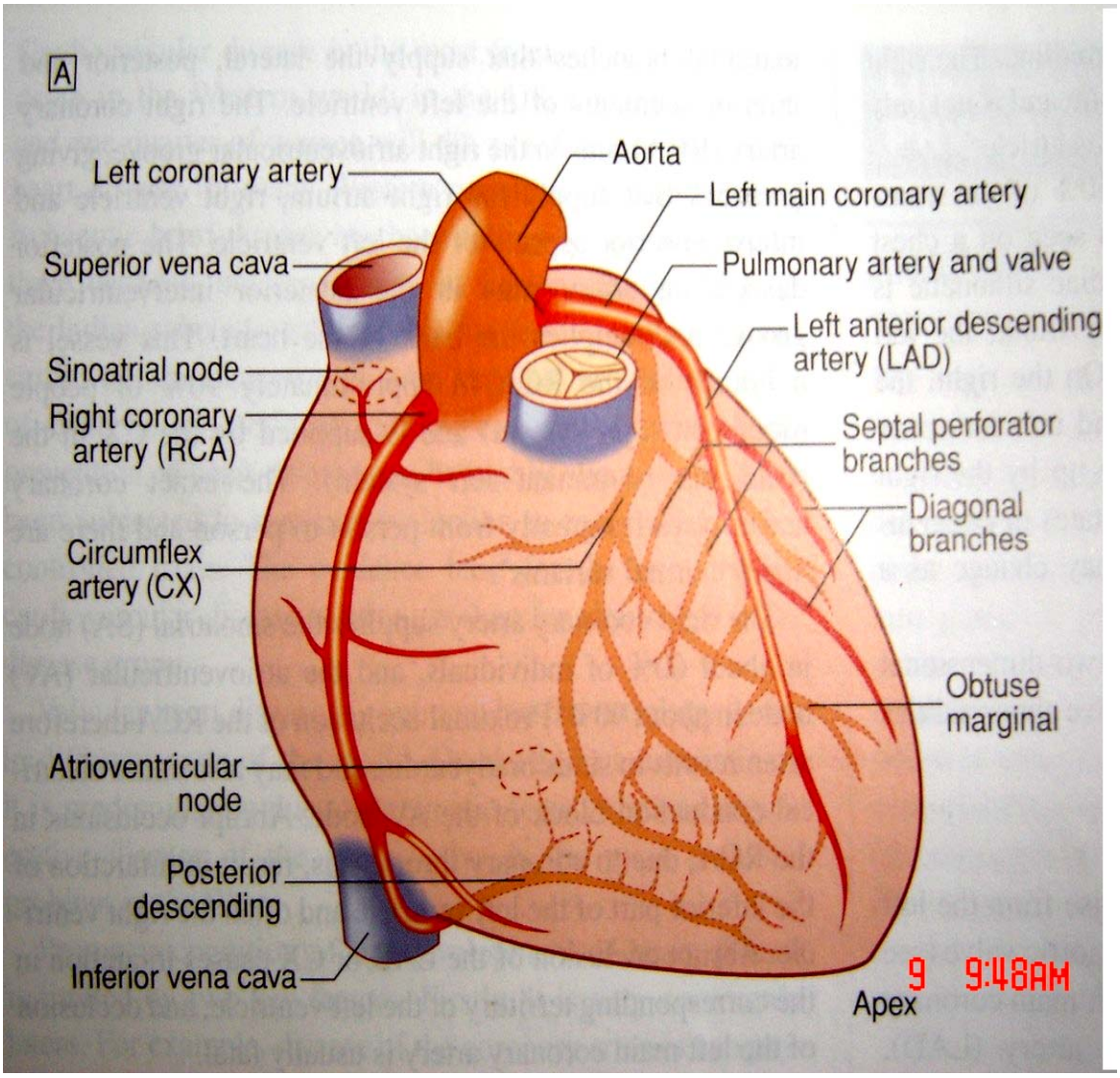
Obesity, diabetes mellitus, hypertension, smokers showed the pattern of dyslipoproteinemia in the study group

Anterior wall MI was seen in 54%, Inferior wall 33%, Posterior wall, 2.85%, combined anterior and inferior wall 86% Incidence of ST elevation MI was 96%. Rythymic disturbances was noted in 40% and features of left ventricular failure were seen in 25.5%. Mortality was 4% . The incidence of left ventricular systolic dysfunction was 35.7%.

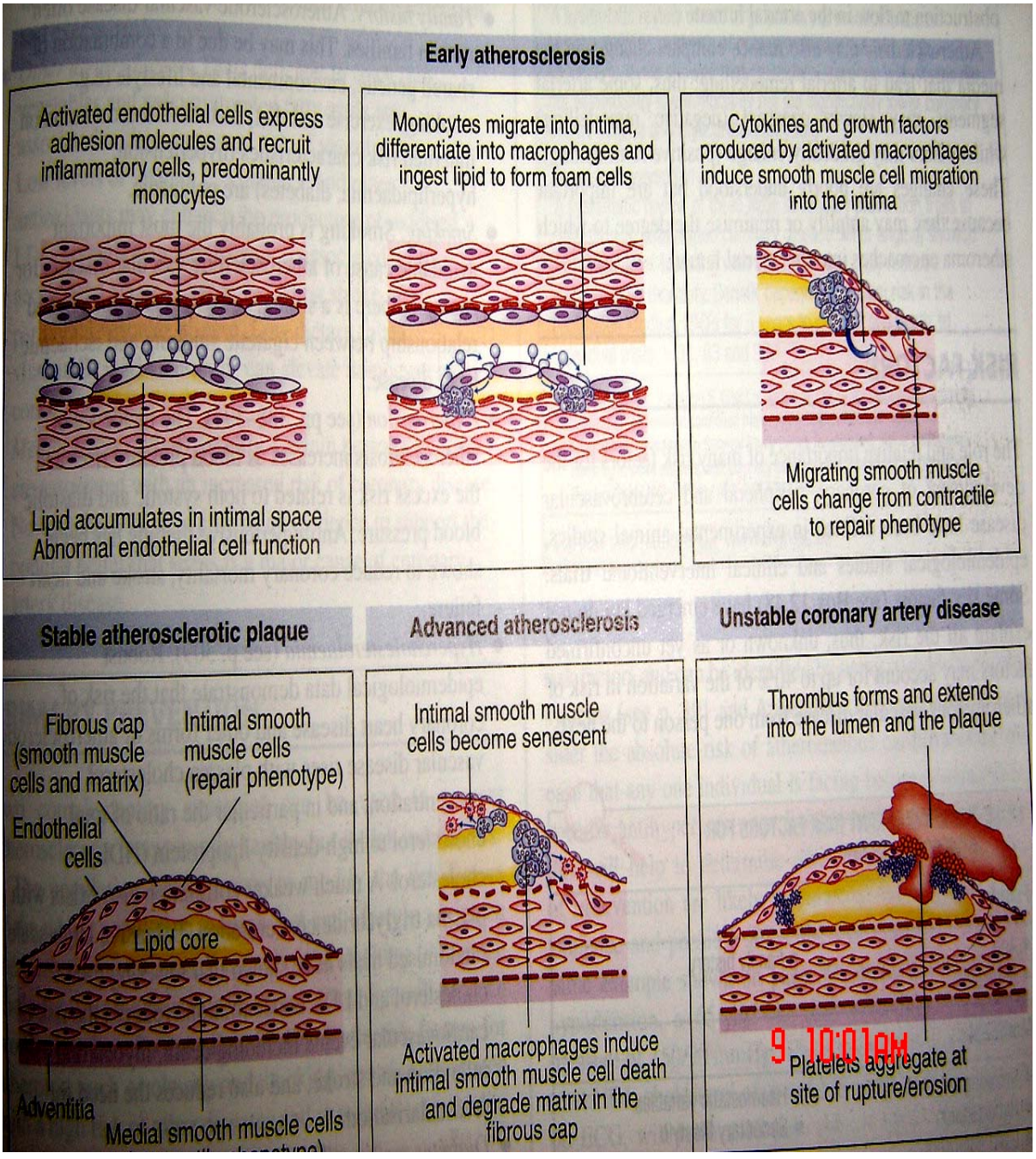
The results were compared with earlier works in the field. Relevant literature were reviewed.



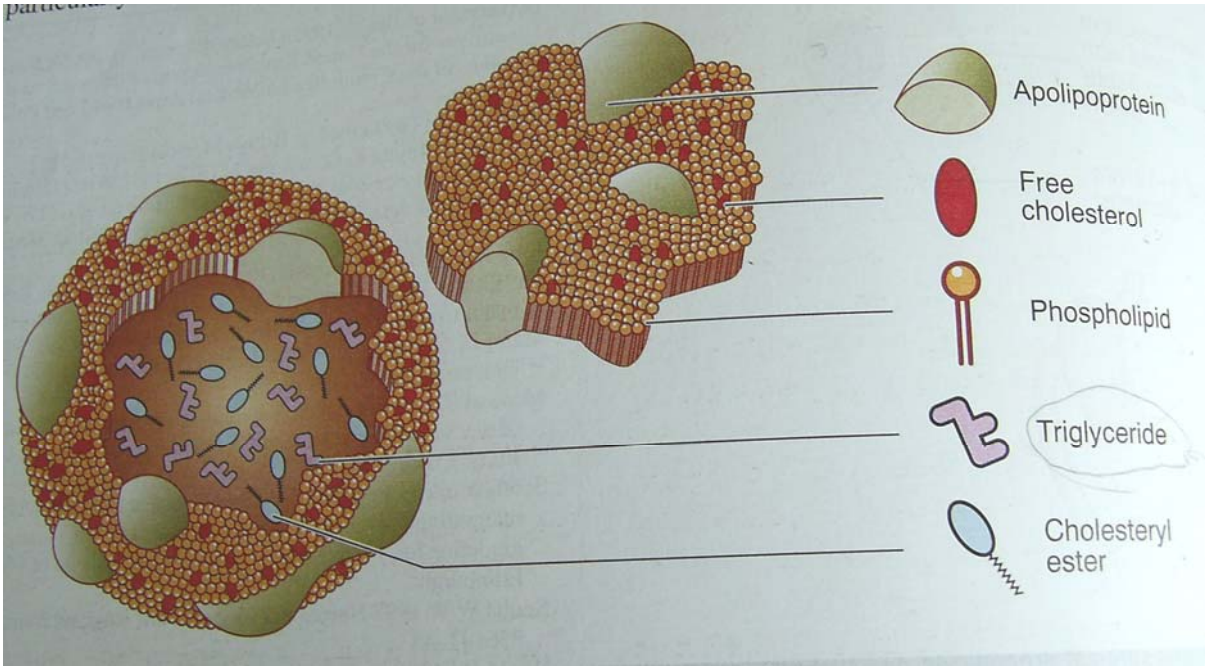

**METABOLISM OF LIPIDS**



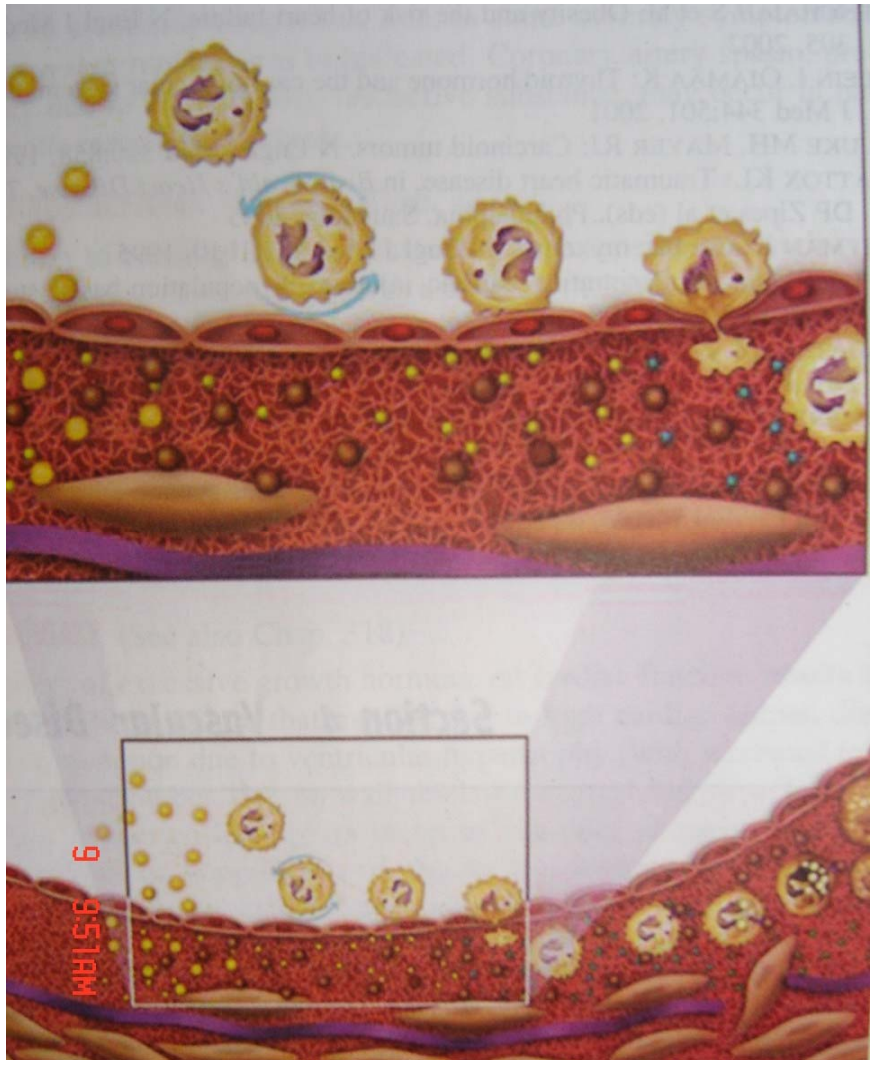

**ANATOMY OF CORONARY CIRCULATION**



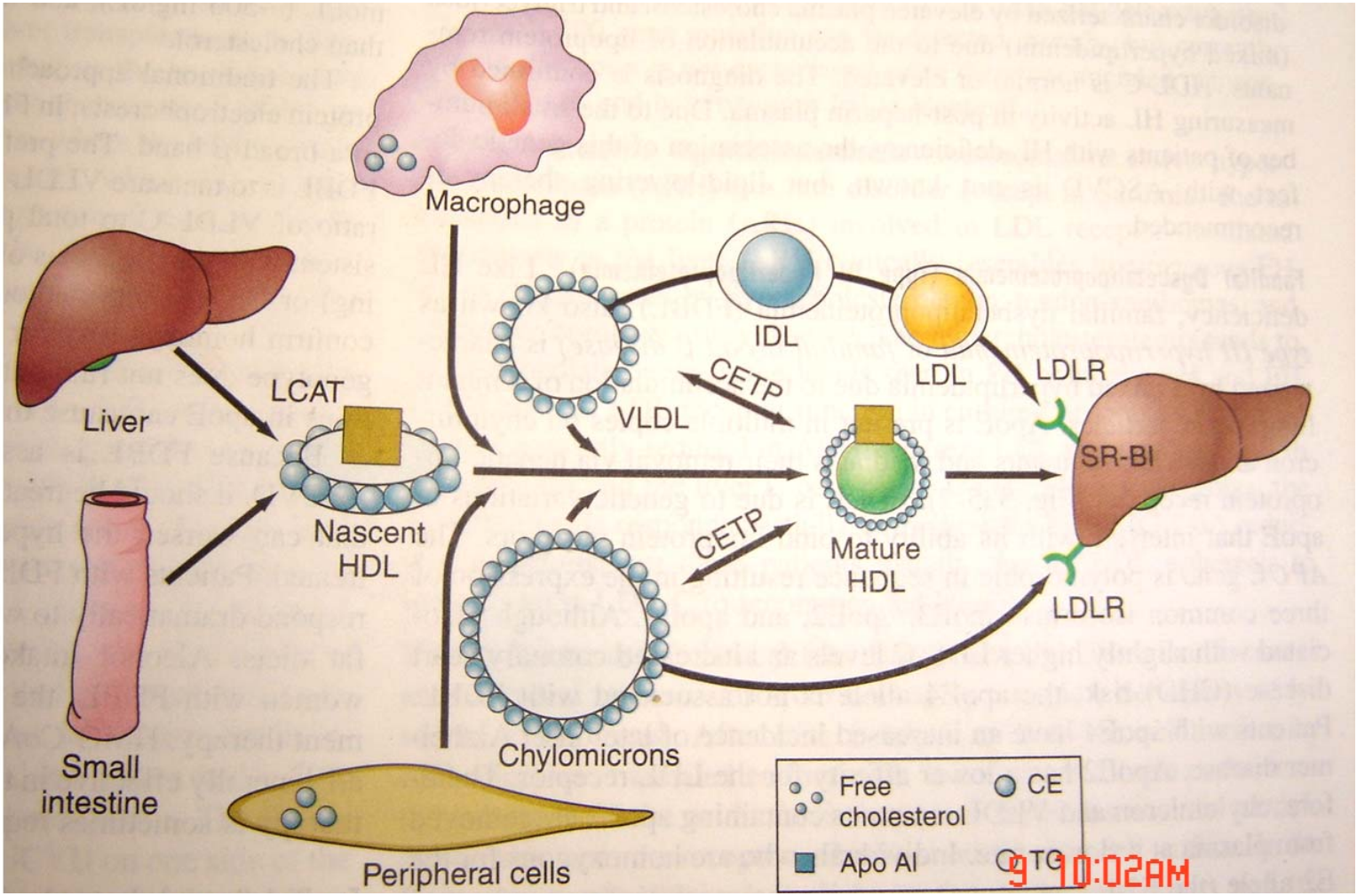

**EVOLUTION OF ATHEROSCLEROSIS**



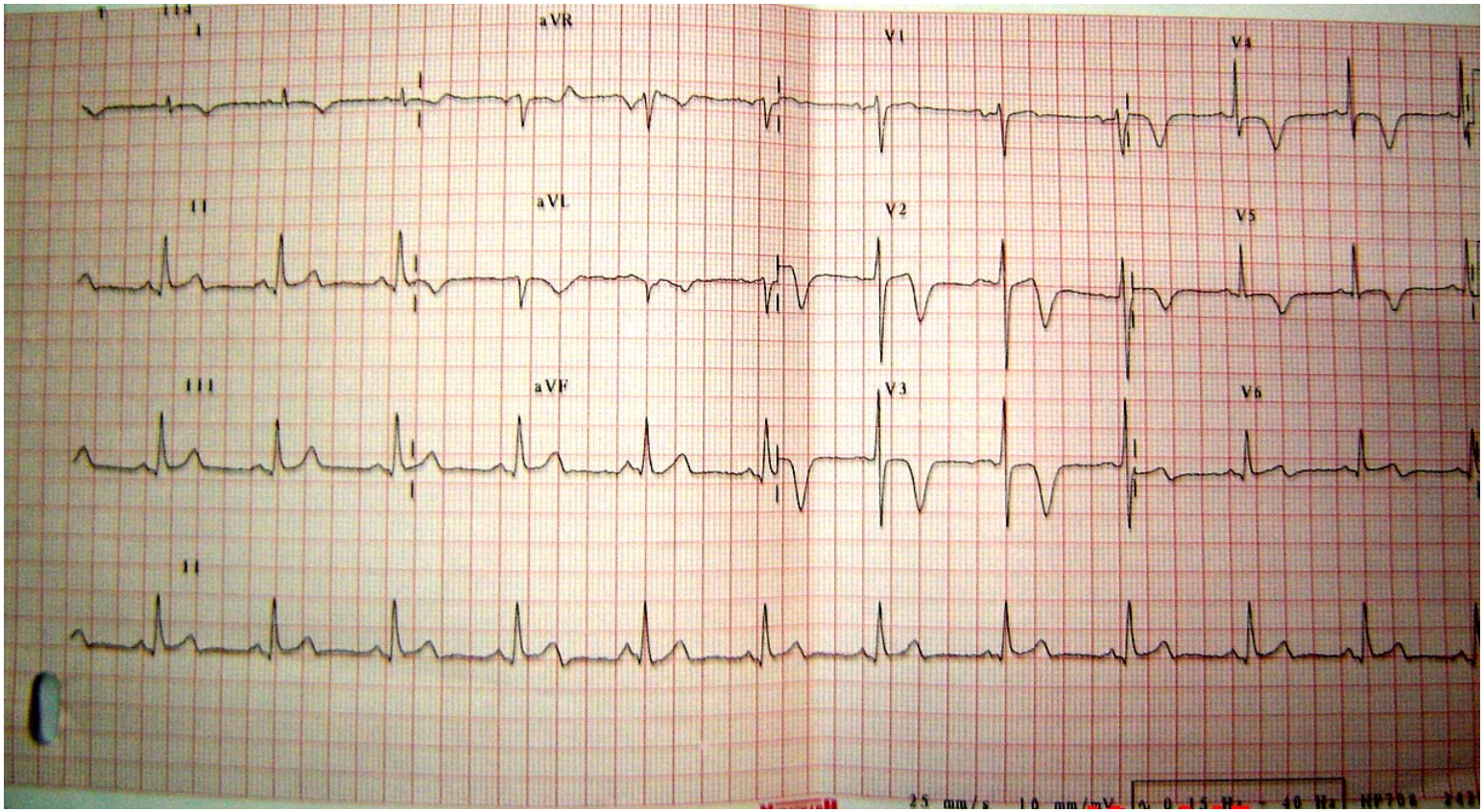

**STRUCTURE OF BASIC LIPOPROTEIN**



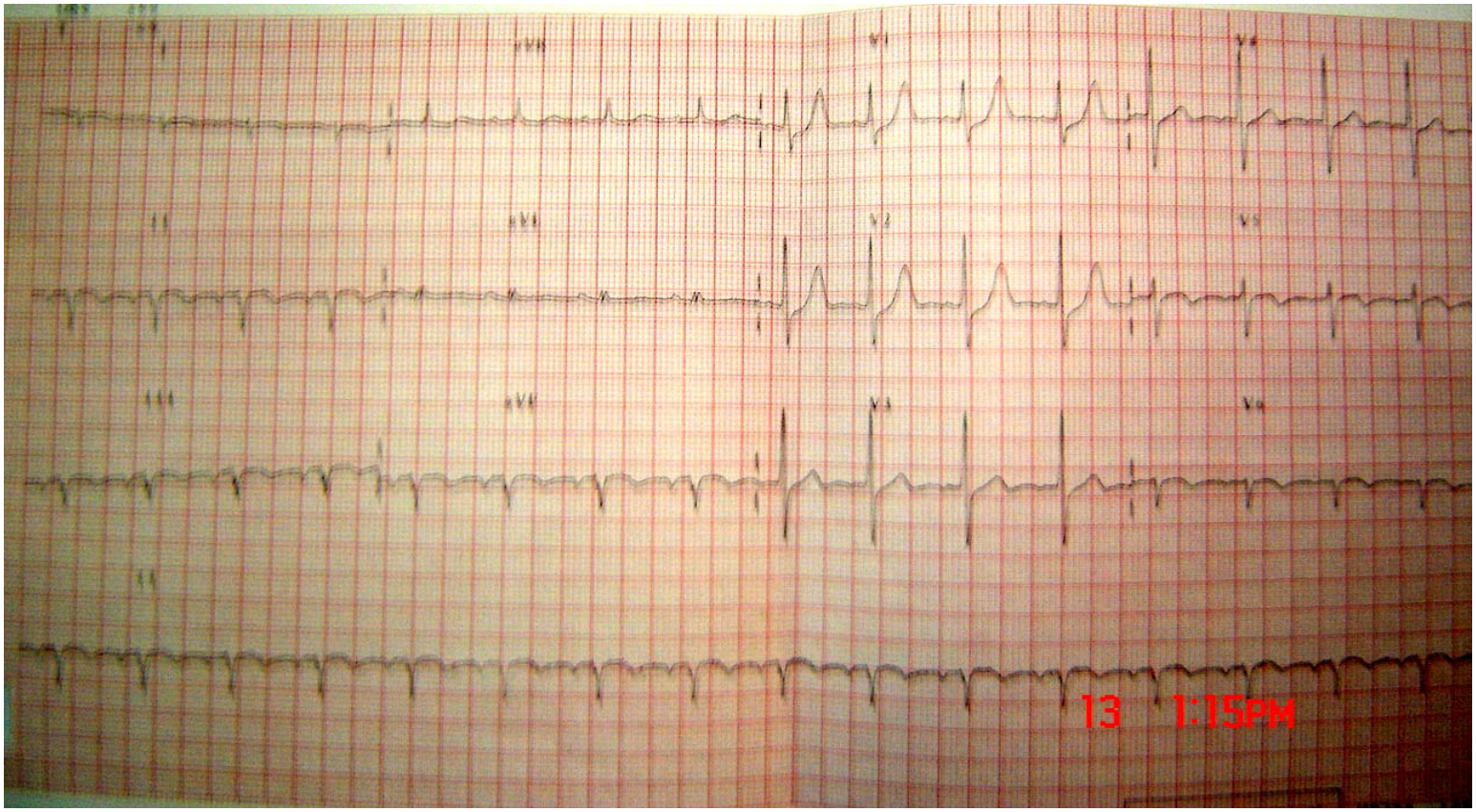

**PATHOGENESIS OF ATHEROSCLEROSIS**



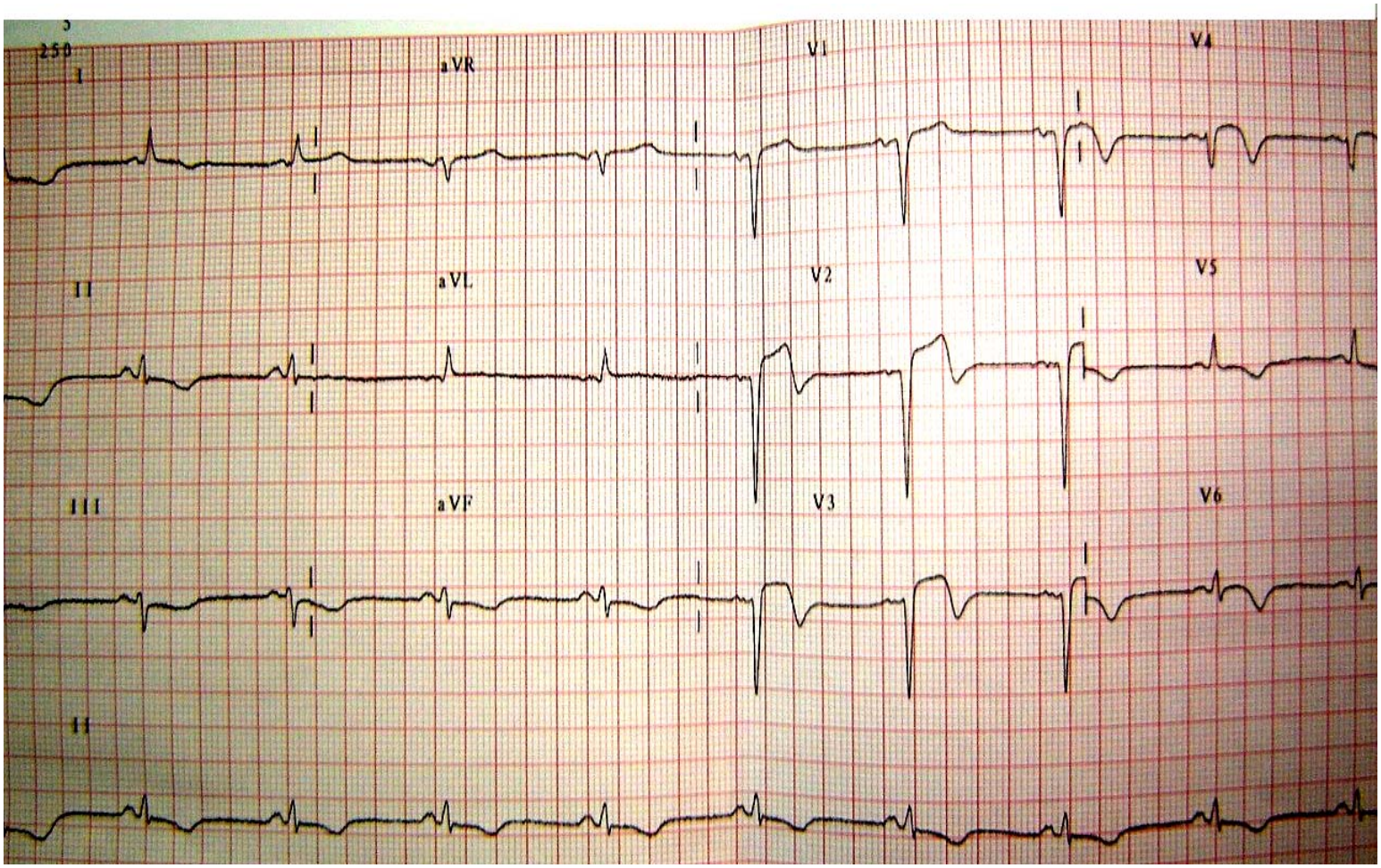

**CHOLESTEROL EXOGENOUS & ENDOGENOUS PATHWAY**



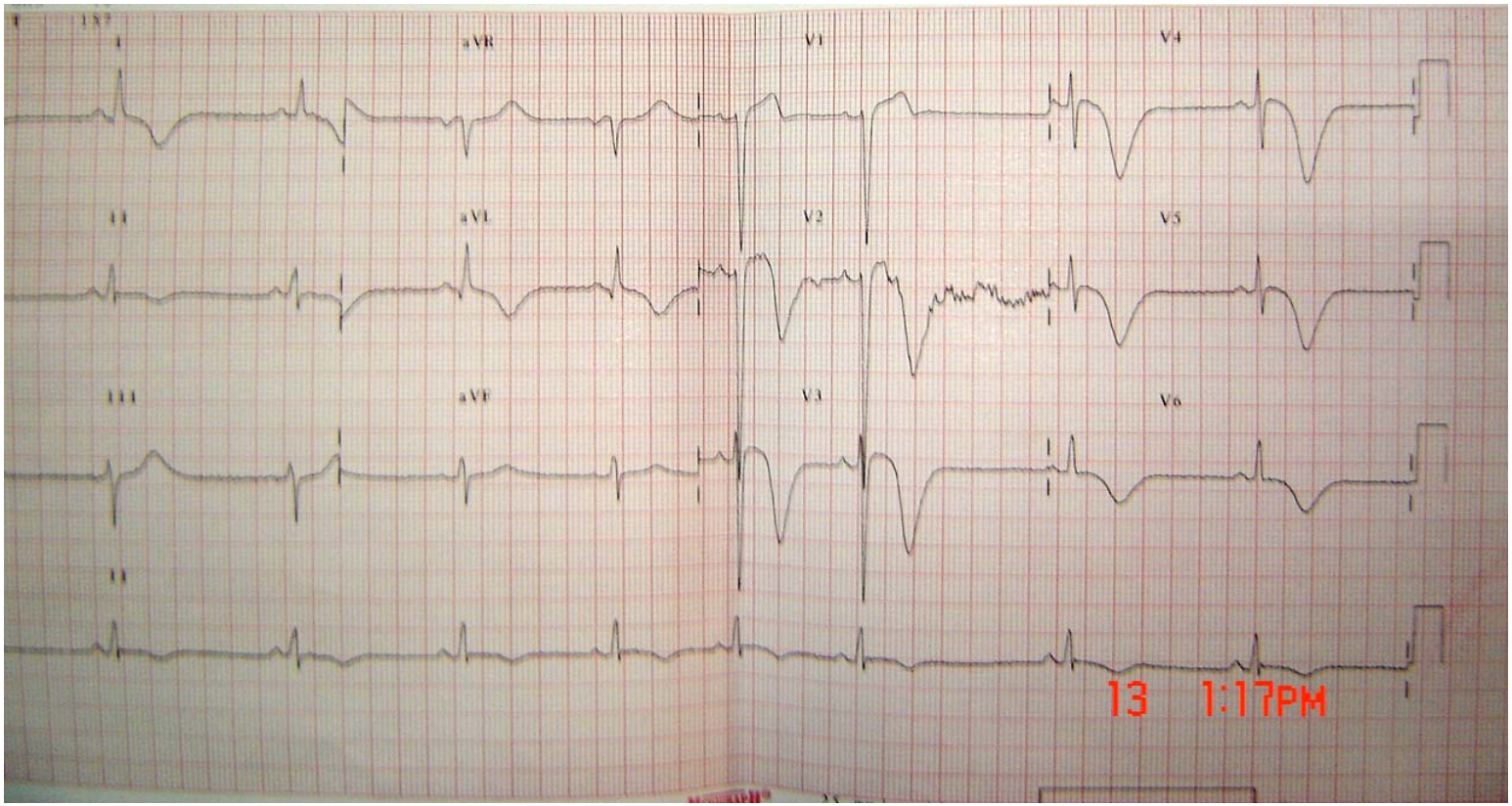

**ECG SHOWING NSTEMI ANTERIOR WALL**



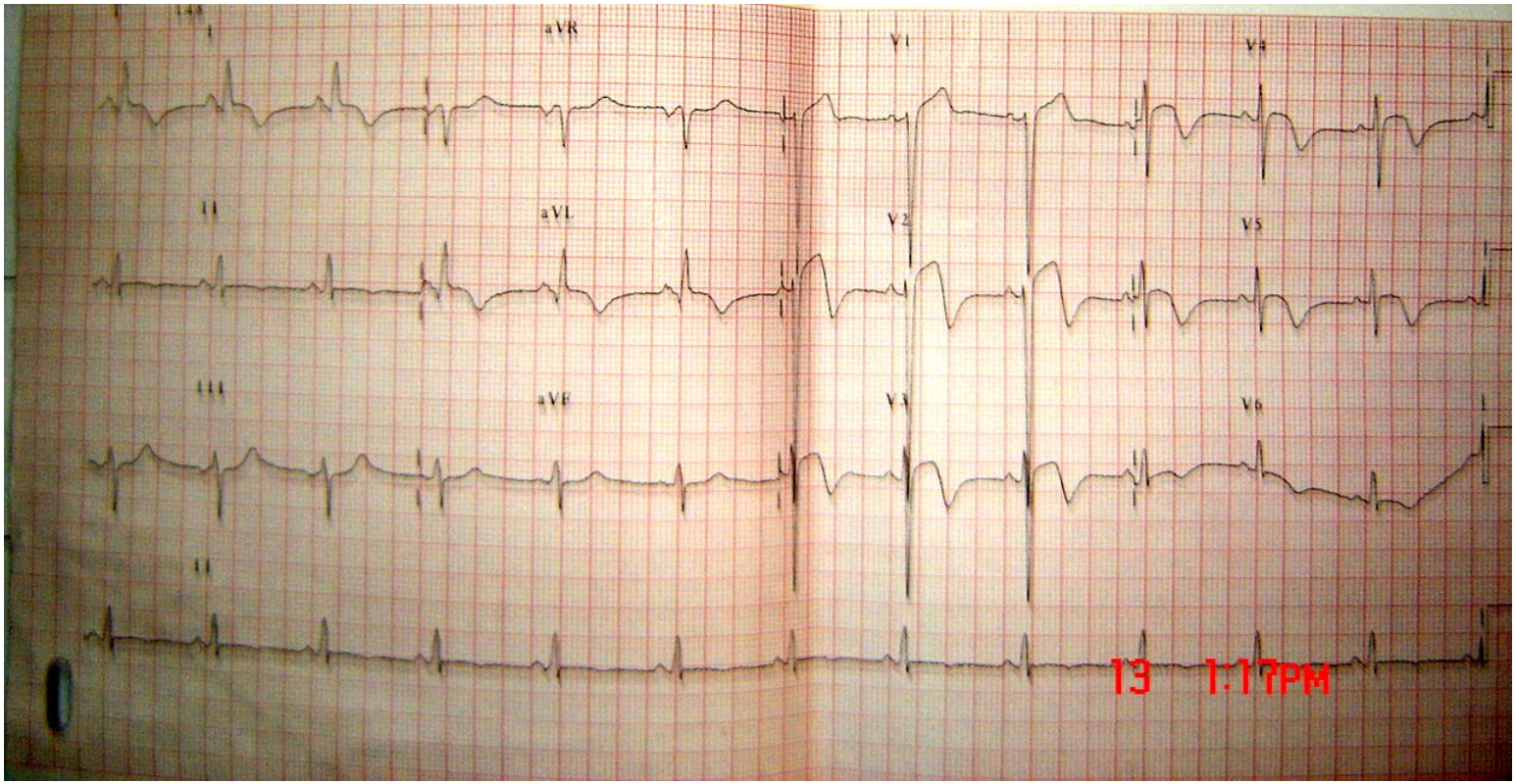

**ECG SHOWING INFERIOR WALL AND POSTERIOR WALL MI**



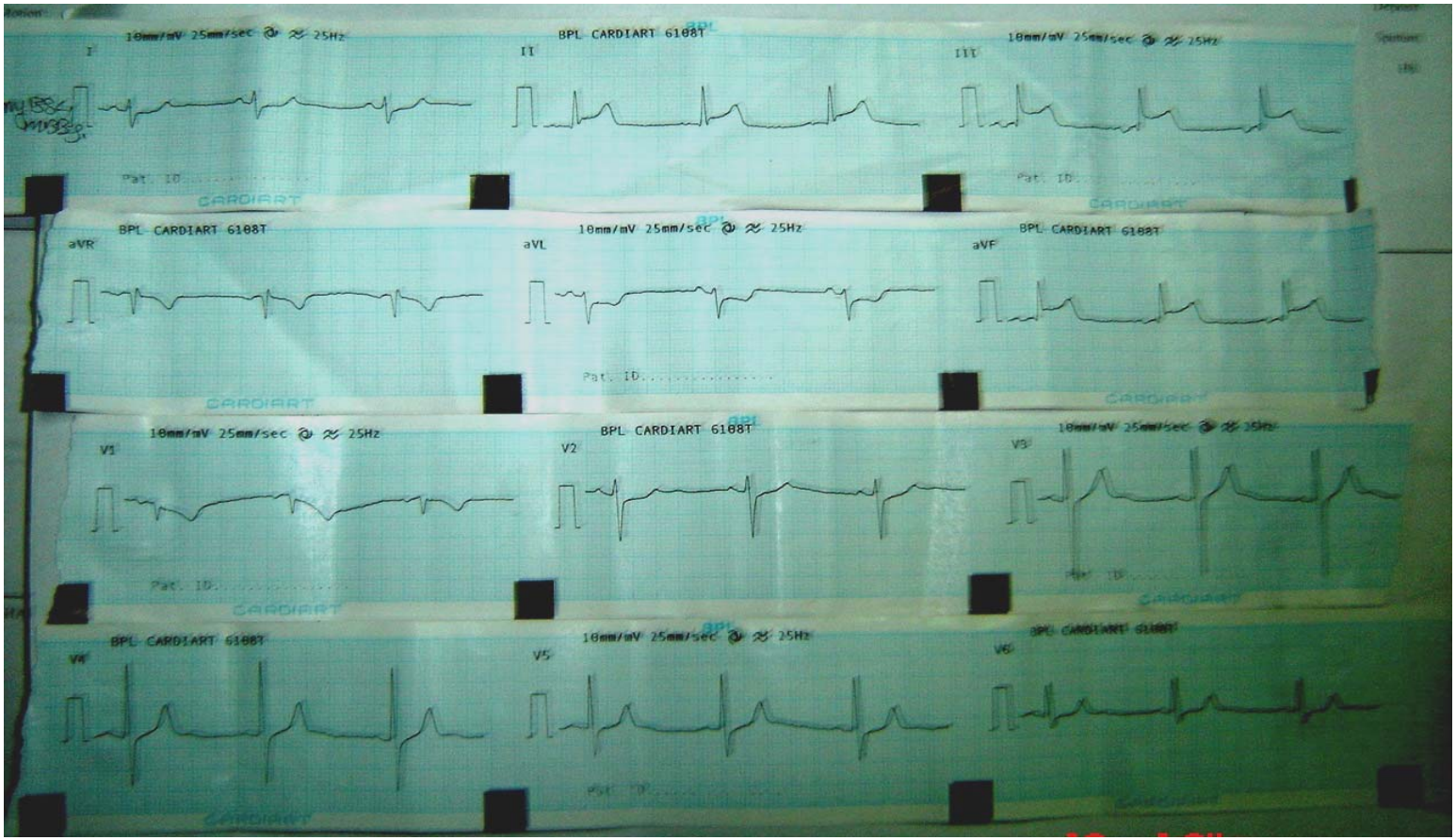

**ECG SHOWING ANTERIOR WALL MI**




**ECG SHOWING ANTERIOR WALL MI**




**ECG SHOWING ANTERIOR WALL MI**




**ECG SHOWING INFERIOR WALL MI**

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