"ASSOCIATION OF LIPOPROTEIN (a) IN CORONARY ARTERY DISEASE IN YOUNG INDIVIDUALS"

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ABBREVIATION

Lp(a)	-	LIPOPROTEIN(a)
CAD	-	CORONARY ARTERY DISEASE
IHD	-	ISCHEMIC HEART DISEASE
ASCVD	-	ATHEROSCLEROTIC CARDIOVASCULAR DISEASE
AMI	-	ACUTE MYOCARDIAL INFARCTION
MI	-	MYOCARDIAL INFARCTION
SVD	-	SINGLE VESSEL DISEASE
DVD	-	DOUBLE VESSEL DISEASE
TVD	-	TRIPLE VESSEL DISEASE
LAD	-	LEFT ANTERIOR DESCENDING ARTERY
LCX	-	LEFT CIRCUMFLEX ARTERY
RCA	-	RIGHT CORONARY ARTERY
LRND	-	LIPID RICH NECROTIC DEBRIS
ECM	-	EXTRACELLULAR MATRIX
ASCVD	-	ATHEROSCLEROTIC CARDIOVASCULAR DISEASE
CVD	-	CARDIOVASCULAR DISEASE
AHA	-	AMERICAN HEART ASSOCIATION
HDL	-	HIGH DENSITY LIPOPROTEIN
LDL	-	LOW DENSITY LIPOPROTEIN
VLDL	-	VERY LOW DENSITY LIPOPROTEIN
IDL	-	INTERMEDIATE DENSITY LIPOPROTEIN
TGL	-	TRIGLYCERIDES
CHD	-	CORONARY HEART DISEASE
VCAM-1	-	VASCULAR CELL ADHESION MOLECULE-1

TGF-B	-	TRANSFORMING GROWTH FACTOR-B
EC	-	ENDOTHELIAL CELL
IL	-	INTERLEUKIN
MCP	-	MONOCYTE CHEMOATTRACTANT PROTEIN
PAI	-	PLASMINOGEN ACTIVATOR INHIBITOR
SMC	-	SMOOTH MUSCLE CELL
TFPI	-	TISSUE FACTOR PATHWAY INHIBITOR
MACE	-	MAJOR ADVERSE CARDIAC EVENTS

ABSTRACT

TITLE: "ASSOCIATION OF LIPOPROTEIN(a) IN CORONARY ARTERY DISEASE IN YOUNG INDIVIDUALS"

Introduction

Coronary artery disease (CAD) is one of the commonest heart diseases, accounting for 5–8% global prevalence. This global data warrants the need for new screening tools and treatment interventions to reduce the incidence/prevalence of atherothrombotic disease.

Methodology

A hospital-based cross-sectional study was conducted on patients admitted in the ICCU and medicine wards at KLES, Dr. Prabhakar Kore Hospital, Belagavi fulfilling the inclusion criteria. Patients between the age of 18 to 49 were the focus of the investigation. Patients with ischemic ECG changes, RWMA (regional wall motion abnormalities) in 2D Echocardiography and/or had symptoms like chest discomfort, dyspnoea or diaphoresis were taken up for the study. Investigations such as a complete hemogram, fasting lipid profile (FLP), Lipoprotein(a), ECG, ECHO, X-RAY, and Angiography were performed and the results were noted. T-test, chi-square and one-way ANOVA were used to evaluate continuous data, and Excel 2007 was utilised for categorical data. By utilising a bar graph and pie charts, the distribution of the variables were depicted. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. P value <0.05 was considered statistically significant. The data was analysed by using SPSS software. Various CAD factors were also examined for

correlations with Lp(a). Age, gender, comorbidities, consumption of tobacco, lipoprotein levels and number of vessels involved (SVD, DVD, TVD) were represented using bar graph & pie charts. Correlations of SVD, DVD, TVD, lipoprotein levels, lipid profiles, total cholesterol, TGL, HDL, and LDL

Results

The average age of subjects in the study population was 41.1 years. Overall average Lp(a) value was 77.98 mg/dl in the study population. 82% (85) males and 18% (19) females contributed to the study, showing male preponderance. Lp(a) and LDL levels were compared and were found to have an average of 77.98 ± 68.22 mg/dl and 82.13 ± 37.20 mg/dl respectively. Majority of the individuals had normal LDL values but high Lp(a) values. Lp(a) was significantly high in subjects who had Lipid profile within the normal ranges. There was a statistical significance seen in the value of Lp(a) in subjects with STEMI, NSTEMI and Unstable Angina with average Lp(a) values of 72.4 mg/dl, 57 mg/dl and 33 mg/dl respectively(p value=0.000) , making the Lp(a) value a very important marker for CAD.

Conclusion

The present study holds significant relevance, as very few Indian studies have evaluated the role of Lp(a) as a risk predictor of CAD in young. The present study adds to the literature evidence advocating the use of Lp(a) as a more specific biomarker of CAD. Routine measurement of Lp(a) helps in the early diagnosis of CAD and detection of myocardial damage. This may help in timely intervention to lower associated morbidity and mortality.

Keywords: Lipoprotein(a), Coronary Artery Disease, Dyslipidemias, Coronary Artery Disease, Acute Coronary Syndrome

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INTRODUCTION

Coronary artery disease (CAD) is one of the commonest heart diseases, which occurs due to building up of plaque in the arterial wall. 5–8 percent of the world's population suffers from CAD, which has a significant economic and human impact. This worldwide data underscores the need for new screening tools and treatment interventions to reduce the incidence/prevalence of atherothrombotic disease.

EPIDEMIOLOGY IN INDIA

India has been experiencing a rise in CAD in last three decades at an alarming rate. This gives us an evidence to seek for the other factors that could be accountable for its snowballing prevalence. There is three-four times higher risk of developing CAD in Indians than Americans, 6 times higher than Chinese and 20 times higher than Japanese[99]. In a very renowned study, Framingham Heart Study the prevalence of CAD was 12.9 per 1000 in men of 30 to 34 age and 5.2 per 1000 in women of 35 to 44 age over a follow-up for 10 years. In another multinational study, Awad et al,[142] studied the patients hospitalized for Acute Coronary Syndrome (ACS) had 23% prevalence of CAD in young adults (<55 years). It has been reported previously that there has been an increase in rates of CAD prevalence in young individuals[100]. According to the investigations in 1990s, it was concluded that prevalence of around 12-16% existed in the young population[101]. It has also been reported, the incidence of acute MI is now as high as 25 to 40% in the young [103]. Analysis of INTERHEART data in South Asians discovered prevalence of acute MI (AMI) in 11.7% in Indians < 40 years of age[102]. In a study by Mohanan et al,[104] in 25,748 ACS patients in Kerala it was observed that prevalence of ACS was 22.2% in the age group of < 50 years,

57.2% in 51-70 years and 20.6% in > 70 years. Further they found similar prevalence of 22.4% in ST-elevation myocardial infarction (STEMI), 22.2% in Non-ST Elevation MI (NSTEMI) and 22% in Unstable Angina (UA) in patients under the age of 50.

The incidence of CAD in the South Asian population is rising at an exponential rate. At an earlier age and with more severe presentations, this cohort is diagnosed with CAD than other demographics, even though they have lower BMI (Body Mass Index) and waist circumferences.[1,2] It is a major cause of death among the population of developed world causing 1 in every 5 deaths.[3] We need a large and comprehensive, all India prospective study to evaluate the prevalence of CAD in young Indians.

Risk factors for coronary heart disease (CHD) were introduced in 1957 by the Framingham heart study (FHS) and have been shown to be correlated with the prevalence of CAD in epidemiological studies. When it comes to modifiable risk factors, Hypertension (HTN), tobacco consumption (smoking), Diabetes Mellitus (DM), obesity, and a sedentary lifestyle are among the most important. Age and gender are the only ones that cannot be altered.[4] Well-established risk factors like tobacco consumption in the form of smoking, DM, HTN and obesity do not completely explain the high incidence of CAD in younger population. Hence, non-conventional risk factors such as elevated lipoprotein(a), homocysteine etc have gained importance in the recent times.[5]

Just over a decade ago, there was very little consensus about whether or not Lp(a) was an independent Atherosclerotic Cardiovascular Disease (ASCVD) risk factor. Presently, majority of available statistics show that Lp(a) is independent of the LDL-C level and is predictive of ASCVD events. The Lp(a) gene is expressed

completely at a very young age and the levels remain steady over a lifetime irrespective of puberty or lifestyle changes. Lp(a) screening is done selectively unlike that of lipid screening which is generally universal. This is well-warranted, given that the gene for Lp(a) is inherited as an autosomal co-dominant trait and is one the most heritable disorders in humans.

A property strikingly unique of Lp(a), is that it is detectable in the serum of newborns, the levels rise and the apo(a) gene is fully expressed by the end of first two years of life.[95] In actual fact, no other lipoprotein level can be traced as effortlessly to adulthood as Lp(a). Lp(a) acts as both proatherogenic and prothrombotic which is due to apo(a) component of the gene.

Patients with normal cholesterol outlines may have difficulty identifying risk factors; in these circumstances, lipoprotein subfractions (LS) might be analyzed to uncover additional dangers.[6] As with other risk factors for CAD elevated lipoprotein levels have been related to an increased risk of the condition.[7,8] The levels of lipoprotein, triglycerides and cholesterol are tend to be increased with elevation of coronary artery disease severity.[9] Lp(a) level is also found to be correlated with the coronary lesions.[10] Small dense low density lipoprotein (LDL) rise has been linked to an increased risk of significant cardiovascular events in persons with diabetes.[11] The high all-cause mortality in CAD patients has significant association with moderately elevation of baseline plasma lipoprotein(a) level of $\geq$15 mg/dL.[12]

At this point in time, there is no single risk factor that can be linked to the development of CAD in young, which supports the role of numerous associated factors. Researchers are trying to find out if there is a link between lipoprotein(a) and other factors associated with CAD.

OBJECTIVES

- To study the association of Lipoprotein (a) and CAD in study population.

REVIEW OF LITERATURE

Coronary artery disease is the narrowing or blockage of the coronary arteries due to the build-up of plaque in the walls of the arteries—a process called atherosclerosis. If a plaque becomes delicate and breaks, a blood clot will quickly form that can block blood flow in the artery and may lead to a myocardial infarction (MI), often referred to as a *heart attack.*

Acute coronary syndromes (ACS) represent a clinical spectrum disease ranging from ST-segment elevation myocardial infarction (STEMI), non–ST-segment elevation myocardial infarction (NSTEMI), or unstable angina (UA).

Coronary artery disease is also called coronary heart disease and ischemic heart disease.[13]

Coronary artery vessels

The blood supply to the cardiac muscle comes from the left and right coronary arteries.

1) **Left Main Coronary Artery branches into:**
    - Left Anterior Descending artery (LAD)- supplies the anterior two thirds of the septum and anterior aspect of the left ventricle.
    - Left Circumflex artery (LCX) - supplies left atrium, lateral and posterior aspect of the left ventricle.
2) **Right Coronary Artery (RCA) branches into:**
    - Right marginal artery - lateral portion of the right ventricle.

- Posterior descending artery - Right atrium, Right ventricle and posterior one-third of the interventricular septum. [14,15,16]

It is possible to have single, double, or even triple vessel disease based on the angiogram representation of the arterial occlusions. The Left main coronary artery and proximal left anterior descending coronary artery are predominantly dangerous.

Atherosclerosis

When fibrofatty lesions form in the intimal layer of artery, they restrict the lumen, leading media deterioration and major repercussions. Early atherosclerosis lesions commonly contain foam cells, which are macrophages that have accumulated excess cholesterol. As the lesion advances, fatty streaks produce smooth muscle cells (SMCs) and lipid-rich necrotic debris (LRND). Lesion rupture can lead to thrombosis, which results in the creation of an embolism, which can lead to a MI or stroke.[17]

- NATURAL HISTORY OF ATHEROSCLEROSIS: The natural history of Atherosclerosis can be studied under 2 headings:

I. 'Pre-clinical phase' (usually at young age)
    1. Fatty streaks
    2. Fibrofatty plaque
    3. Advanced/ Vulnerable plaque

II. 'Clinical phase' (usually middle age to elderly)
    1. Occlusion by thrombus
    2. Critical stenosis
    3. Aneurysm and rupture

Pathophysiology of atherosclerosis

Atherosclerosis can take place through following events:

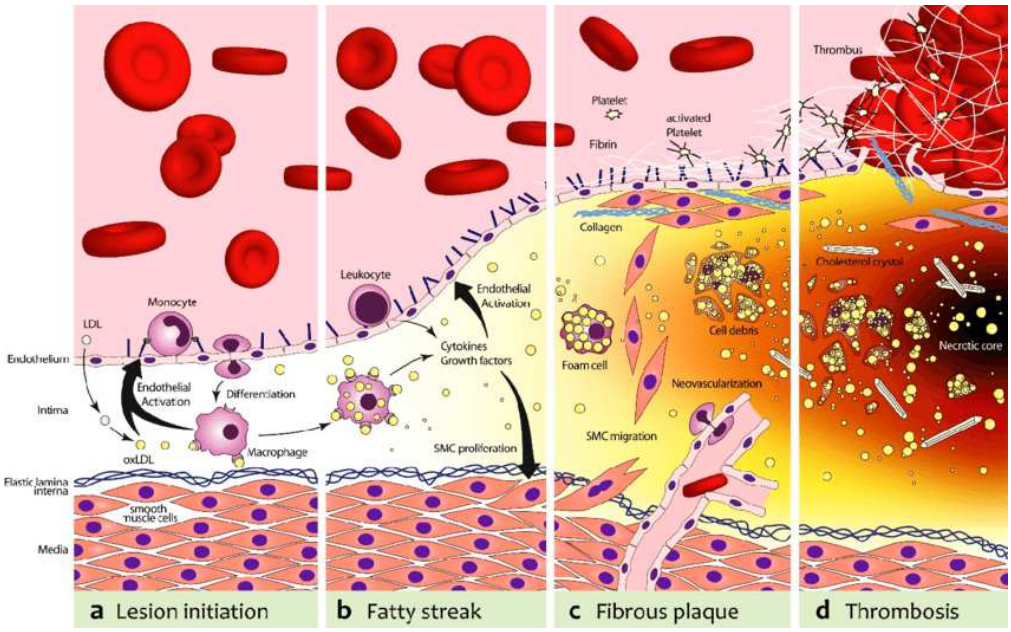
Endothelial injury: Endothelial injury can cause adhesion of leukocytes, thrombosis and increased permeability.

Lipoproteins: The accumulation of lipoprotein can take place in the vessel wall. It is oxidized into LDL, which leads to stimulation of endothelial cells to show adhesion molecules such as vascular cell adhesion molecule-1 (VCAM-1), P-selectin and diverse types of chemokines.

Monocyte adhesion: In the subendothelial space, monocyte transmigrates into the intima, acquiring the characteristics of macrophages, and transforms into foamy macrophages.

Platelet adhesion and lipid accumulation: Smooth muscle cell recruitment is induced by platelet adhesion factor, which is released by activated platelets, vascular wall cells, and macrophages, and this results in ECM and smooth muscle cell proliferation, as well as lipid accumulation in macrophages and smooth muscle cells. Platelet adhesion factor, platelet activation and deposition on a defective endothelium lead to the creation of a pro-thrombotic milieu. Foam cells apoptosis releases lipids and cell debris, which can lead to necrotic core development. Peptides from foam cells destabilise the plaque and ECM components such as collagens, elastin, tissue factor (TF) and von Willebrand factor (vWF), which in turn trigger thrombotic events**.**

Figure 1: Pathogenesis of atherosclerosis[18]



Source: Steinl et al; 2015

Lp(a) contributes to the development of atherosclerosis in several ways:

1) It increases the expression of vascular cell adhesion molecule-1 and E-selectin in endothelial cells;
2) It increases accumulation of peripheral blood mononuclear cells in vessels; and
3) Pro-inflammatory - through its oxidised phospholipids.

Global burden of coronary artery disease

CAD has been predicted to be the leading cause of death globally for the next 20 years.[20] The corresponding mortalities among men and women death due to CAD were around 3.8 and 3.4 million, accounting for 11.1 million deaths globally in 2020.[21,20] In Europe, someone is experiencing coronary events in every 26 seconds,

leading to death every minute.[22] Heart disease is the leading cause of death in 1 in 7 women from Europe, and 16% of all deaths in Europe.[23] It is considered as epidemic due to increased prevalence of CAD across the world.[23] According to a study reported by Jones et al,[24] the corresponding average age adjusted incidence rate of CAD per 1000 person-years were 10.6 for black men, 4.0 for white women and 12.5 for white men.

As per the statistics reported by American Heart Association (AHA) in 2008, 7,70,000 Americans had coronary artery attack, while recurrent attack was experienced by 4,30,000. Around 1,90,000 silent first heart attacks are occurring annually.[22] CAD is also responsible for morbidity and loss of quality of life, in addition to its mortality burden. It also exerts massive economic cost, and it was around EU €45 billion in 2003. The economic loss due to CAD mortality is estimated to be around €11.7 billion, with loss of one million working years and loss of 90 million working days due to CAD morbidity.[25]

In 2016, the Heart Disease and Stroke Statistics report published by the AHA reported that 15.5 million persons ≥20 years of age had suffered CAD in USA, and the prevalence of the disease increased with age. An American is suffering from myocardial infraction in every 42 second.[26] The countries that have reported the highest prevalence of CAD in the last two decades are Latvia, Estonia, Lithuania, Bulgaria, and the Czech Republic; however, the countries with high income such as Denmark, Germany, United Kingdom, Finland, and Italy have come down in ranking. Increased prevalence has been reported in western Europe and was comparatively higher than South Asia and rest of the world.[27]

As of 2030, the incidence of ischemic heart disease (IHD) could rise to 1,845 per 1000,000, with an upper confidence estimate of 1,917 per 100,000, according to the prediction model constructed by Khan et al. Compared to women, men had a higher incidence of IHD, with 1,786 cases per 100,000 men compared to 1,522 instances per 100,000 women. 126 million people, or 1.72 percent of the world's population, have IHD, according to Khan et al, [27]study 9 million people have died as a result of IHD worldwide, they estimate. South Asians countries such as Bangladesh, Bhutan, India, the Maldives, Nepal, Pakistan, and Sri Lanka had higher proportion of atherosclerotic cardiovascular disease (ASCVD) mortality rates compared to other Asian and non-Hispanic whites. The global burden of ischemic heart disease is depicted in table 1.[28]

Table 1: Global burden of ischemic heart disease[27]

Region	Prevalence (rate per 100,000)	Disability - Adjusted Life Years (rate per 100,000)
Global	1,655	2,228
Europe	3,547	3,771
Germany	3,432	2,855
France	2,696	1,237
Italy	3,468	1,831
Spain	2,733	1,503
Netherlands	3,502	1,451
Switzerland	2,581	1,461
Sweden	3,858	2,192
Turkey	2,418	1,960
Russia	4,198	6,568
United Kingdom	3,337	1,864
Asia and Australasia	1,440	2,272
China	1,612	2,131

India	1,197	2,679
Japan	2,928	1,427
South Korea	1,352	704
Taiwan	1,759	1,241
Saudi Arabia	1,509	1,643
Iran	1,599	2,149
Australia	2,576	1,450
Americas	1,990	1,887
United States	2,929	2,470
Canada	2,335	1,837
Brazil	1,685	1,736
Africa	880	1,309
South Africa	1,227	1,184

Burden Of Coronary Artery Disease In India

Since 1990, India has seen a dramatic increase in the number of people suffering from coronary artery disease. In 2016, cardiovascular disease accounted for 28 percent of India's overall mortality, compared to 15 percent in 1990, according to the latest data. In India, Kerala, Punjab, and Tamil Nadu are the states that have the maximum prevalence of CAD, as well as excessive cholesterol and high blood pressure. Between 1970 and 2013, the prevalence of cardiovascular disease among Indians has increased fourfold in rural areas and sevenfold in urban areas, and presently it stands at 14 percent in urban areas and 7 percent in rural areas. Urban men had a CAD of 255 per 100,000, whereas rural men had a CAD of 234 per 100,000. Age-adjusted CVD mortality in India was 325/100,000 in males and 225/100,000 in women, compared to 190/100,000 in the United States. Currently, India has the highest burden of CAD and STEMI in the world.[74] This form of CAD is the most common form of presentation accounting for two-thirds of all AMI in India *vs* one-third in the United

States. Very high rates of CAD have also been reported in Pakistan and Bangladesh. Recent global burden of disease study have estimated that between 1990 and 2010, mortality secondary to CAD in South Asia has increased by 88 percent as compared to a 35 percent decline globally. Unless aggressive preventive efforts are undertaken, the number of deaths due to CAD in South Asian population is predicted to increase by another 50 percent by 2030. In the recent past, it was noted that there was an increased rate of massive infarction, severe ventricular dysfunction and triple vessel disease that have resulted in higher rates of mortality in young Indians. Young Indians between the ages of 35 and 65 are more likely to develop CAD than other people around the world.[29,30,31,32]

Table 2: Burden of ischemic heart disease in India

Regions	**Disability-adjusted life years lost per lakh population**
Highest burden	
Punjab	5758
Tamil Nadu	4,788
Haryana	4,244
Andhra Pradesh	4,023
Karnataka	3,892
Lowest burden	
Mizoram	663
Arunachal Pradesh	957
Meghalaya	957
Nagaland	1,167
Sikkim	1,526

Risk factors for coronary artery disease

Modifiable risk factors:

1. Diabetes
2. Hypertension
3. Dyslipidemia
4. Cigarette smoking
5. Obesity
6. Physical inactivity

Non-modifiable risk factors:

1. Age
2. Gender
3. Ethnicity
4. Family history

Modifiable risk factors:

1. Diabetes: According to the Framingham Heart Study, persons with diabetes have a two- to three-fold greater chance of developing atherosclerotic disease. 68 percent of diabetics aged 65 and over had cardiac disease, according to the American Heart Association (AHA), and 16 percent had died from a stroke. Death rates for diabetic adults were up to 2-4 times higher than for healthy controls. This has led to diabetes being listed as a major risk factor for cardiovascular disease by the American Heart Association.[33]

2. Hypertension: High pressure is exerted in the parts of the vasculature with the development of atherosclerosis, although coronary artery disease had been observed in subjects with diastolic blood pressure of 75 mmHg and systolic blood pressure of 115 mm Hg[34,35]. The risk of CAD rises when hypertension is accompanied by other risk factors such as, dyslipidemia, obesity, etc[36]. Hypertension-induced atherosclerosis is primarily caused by an increase in oxygen free radical production. Atherosclerosis develops when the transcription factor NFkB enters the nucleus, stimulates the production of the 'Vascular Cell Adhesion Molecules (VCAM-1)' and 'smooth muscle growth factors'. Plaque rupture can be facilitated by an increase in blood pressure.[3]

3. Dyslipidemia: The changes in cardiovascular disease incidence rate have been associated with changes in levels of cholesterol.[38] Cardiovascular disease risk can be predicted using total plasma cholesterol. LDL is termed as "bad cholesterol" has also been linked to cardiovascular disease.[39,40,] HDL is termed as "good cholesterol". Second only to smoking as a risk factor for Ischemic Heart Disease (IHD) is hyperlipidemia. Hypercholesterolemia, joint hyperlipidemias, and low HDL have all been related with coronary calcium scores above 55% in a cross-sectional analysis. As a risk factor for CAD, elevated triglyceride levels are also examined.[42,43,44]

4. Cigarette smoking: The higher the number of cigarettes smoked and the younger the smoker, the greater the danger. It is therefore said it is dose dependent. If any additional risk factor is present, the risk increase by two to three times. CRP, platelet adhesiveness, and HDL levels can all be raised in smokers due to the oxidation of low-density lipoprotein (LDL).[45,46] According to Framingham study, there is association between increased risk of MI and smokers and daily cigarette

consumption.[47] There has been double risk of morbidity and mortality due to IHD in smokers compared to non-smokers.[48,49]

5. Obesity: Using the Framingham heart research, Kannel et al,[50] discovered a link between obesity and cardiovascular illness. For all-cause mortality, obesity was recognised an independent risk factor for type 2 diabetes, coronary heart disease, hypertension and sleep apnea. Ades reported hazard ratio of 2.00 (95% CI 1.67-2.40) in obese patients indicating twice as likely of having CAD.[51]

6. Physical inactivity: Exercise can be an important preventive measure in the development of CAD. A case-control study, conducted across 52 countries with involvement of 15,152 cases and 14,820 controls, has found that physical inactivity has 12.2% of attributable risk for MI.[52] Berlin et al,[50] reported that subjects with inactive lifestyle had (Relative Risk = 1.9, 95% CI: 1.6-2.2) higher risk of death due to coronary heart disease when compared to active people.

Non-modifiable risk factors

1. Age: The prevalence for CAD increases in men and women post 35 years with a lifetime risk of developing CAD, 49% in men after age of 40 and 32% in women after age of 40.[53]

2. Gender: Men are found to be at an increased risk for CAD compared to women.[4]

3. Ethnicity: Few ethnic groups are specific to having an increased morbidity and mortality due to CAD such as Blacks, Hispanics, Latinos, and Southeast Asians.[54,55,28]

4. Family history: A family history of coronary artery disease is a significant risk factor. In families with a history of CAD, early onset of CAD is common. Early

detection of family members with CAD history can help avoid CAD, especially if a parent or sibling died from CAD or developed CAD at a young age. A study by Slack et al.[56,57] found that there was a 2.5 to 7 times greater risk of developing CAD in first-degree relatives of patients with CAD.

Plasma lipoproteins:

Plasma lipoproteins are of 7 types based on their size, lipid composition and apolipoprotein depending on the density:

- **High density lipoprotein (HDL):** HDL is important in transporting cholesterol from the peripheral tissues to the liver, which helps in removal of excess cholesterol. HDL care rich in cholesterol, phospholipids and constitutes apolipoproteins.[64]

- **Low density lipoprotein (LDL):** LDL is derivative of VLDL and IDL particles and is richer in cholesterol. LDL carries the major part of the cholesterol present in the circulation. The predominant apolipoprotein is B-100 and each LDL particle contains one Apo B-100 molecule. Small dense LDL particles have a low affinity for the LDL receptor resulting in a prolonged retention time in the circulation. These have a tendency to enter the blood vessel wall with no trouble and bind more avidly to intra-arterial proteoglycans, which traps them in the arterial wall.[59]

- **Very low density lipoprotein (VLDL):** VLDL are enriched with triglyceride and are produced by liver. They consist of apolipoproteins such as B-100, C-I, C-II, C-III, and E and Apo B-100 molecule.[59]

- **Intermediate-density lipoprotein (IDL)**: IDL are pro-atherogenic particles, formed due to triglycerides removal from the VLDL by muscles and adipose tissue. IDL is enriched with cholesterol and contains apolipoprotein B-100 and E.[59]
- **Chylomicrons:** Chylomicrons are enriched with triglyceride particles produced by intestine and consist of apolipoproteins such as 'A-I, A-II, A-IV, A-V, B-48, C-II, C-III, and E' with Apo B-48 being the core structural protein with each one Apo B-48 molecule having one chylomicron particle. The size of chylomicrons varies depending on the amount of fat ingested.[59]
- **Chylomicron remnants:** Chylomicron remnants are formed by eliminating triglyceride from chylomicrons in the peripheral tissues. These are pro-atherogenic particles, enriched with cholesterol compared to chylomicrons.[59]

Chylomicron remnants, VLDL, IDL, LDL, and Lp(a) are all highly 'pro-atherogenic' while only HDL is 'anti-atherogenic'.

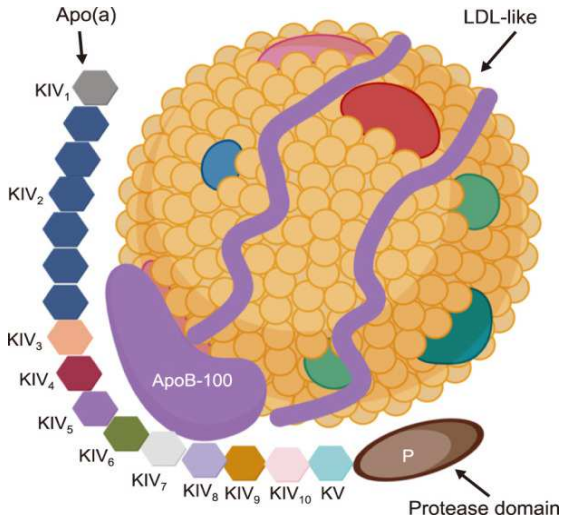
Lipoprotein(a) [Lp(a)]

Lipoprotein(a) [Lp(a)] is a low-density lipoprotein like particle with apoB (apolipoprotein B) which is covalently bound to a specific highly polymorphic glycoprotein apoA [apolipoprotein(a)] via a disulfide bond.[58] The apo(a) component is highly proatherogenic and prothrombotic.

Lipoprotein(a) is expressed in the first year of life and hence does not vary with age. Due to the insolubility of lipids in water, such as cholesterol and triglycerides (TGL), they are associated with protein(lipoproteins) during circulation. In small intestine, it plays a vital role in transportation and absorption of lipid, while transporting lipid

from liver to peripheral tissues and similarly from peripheral tissues to liver and intestine.[59]

Fig. 2: Structure of Lp(a)[60]



Phospholipid molecules and fat droplets form the core of Lp(a) macromolecule complexes, which are formed largely in the liver and the small intestine. Amphipathic fat molecules with phosphorus-containing groups attached to them are known as phospholipids. Phospholipid molecules in Lp(a) face outward to connect with water, which aids in the movement of Lp(a) through the bloodstream. As the name suggests, it is made up of an apoprotein [apo(a)], which is made up of a single copy of plasminogen Kringle 5, plasminogen Kringle 4, along with an inactive protease domain in numerous copies of this protein. In LDL surface apo(a) and apolipoprotein(b) are connected to each other with minimum contact.[61,62,63]

Pathogenic Mechanisms of Lp(a)

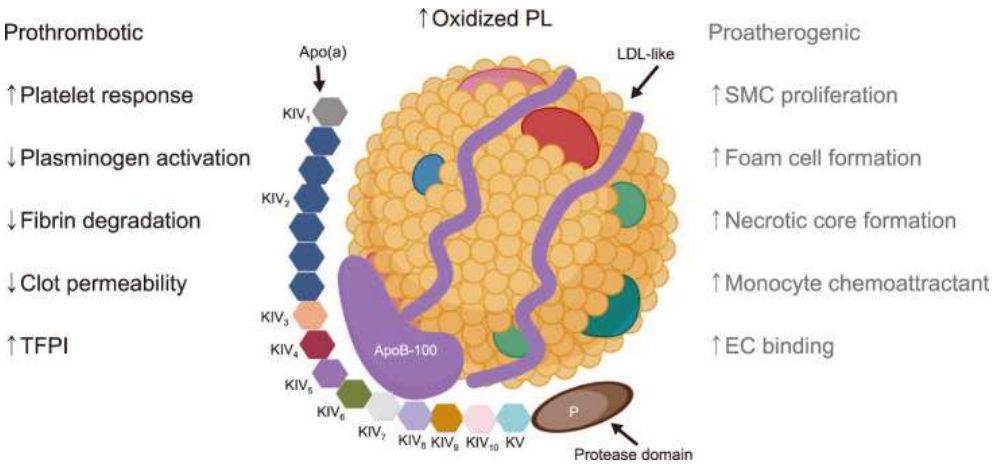
To a great extent, Lp(a) is more atherogenic when compared to LDL-C and the atherogenicity of Lp(a) can be broadly classified in 3 categories:

1. Proatherogenic
2. Proinflammatory, and potentially
3. Antifibrinolytic.

Role of apo(a):

1. Smaller apo(a) isoform favourably binds to vascular intima.
2. Inhibition of activation plasminogen to plasmin due to smaller isoforms that bind strongly to fibrin and inhibits fibrinolysis.

Figure 3: Pathogenic Mechanisms of Lp(a)



EC = endothelial cell; SMC = smooth muscle cell; TFPI = tissue factor pathway inhibitor

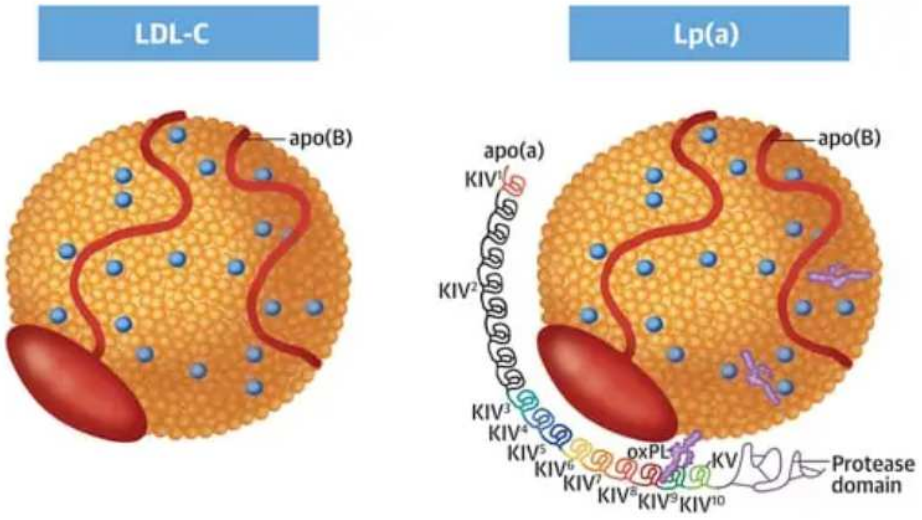
Few animal studies have shown that Lp(a) can be retained in the blood vessel wall to a larger extent than LDL. Lp(a) also has a tendency to bind to triglyceride-rich lipoproteins, a property which may subsidize to the accumulation of lipid in the arterial wall. Like LDL, Lp(a), is also subject to oxidative modification which leads to it becoming a substrate for uptake by macrophages thereby fuelling the formation of

foam cells. Lp(a) also compose inflammatory properties which promote the chemotaxis of monocytes and thereby inducing the expression of vascular adhesion molecules.

In an advanced atherosclerotic lesions Lp(a) is found in areas of calcification. Lp(a) also possess osteoblastic activity as seen when culture of smooth muscle was done and Lp(a) was found to calcify it by stimulating the uptake of calcium by these cells and promoting the change in phenotype. It is due to the presence of Lp(a) within arterial wall that provokes premature changes in the arterial wall before any evident manifestations of atherosclerosis is noted.

Lp(a) also affects the catabolism of other lipoproteins. Lp(a) enhances the burden in previously known hypercholesterolemic patients by inhibiting the clearance of non-HDL cholesterol which is done by the apo(a) kringle.

Figure 4: Structural difference between LDL and Lp(a)



Source: Sigurdsson[8]

Relevant studies on association of Lp(a) with CAD

Lp(a) and CAD have been researched in a range of groups. Retrospective case-control studies encompassing individuals with varied coronary outcomes, including symptomatic angina, MI, and angiographically proven coronary stenosis, have consistently related Lp(a) to coronary heart disease (CHD).[66,67] However, the selection bias and limitation in evaluating the temporal association between outcome and exposure often impair the accurate measurement of the association.

There is substantial literature evidence to validate the association between Lp(a) levels and the risk for major vascular and nonvascular outcomes. The mechanisms of Lp(a) pathogenicity comprise of the following:[68]

- Increased thrombogenesis
- Impaired fibrinolysis
- Plaque destabilization
- Inhibition of transforming growth factor β
- Elevated smooth muscle cell proliferation and migration
- Occlusive thrombus formation
- Impaired formation of collateral vessels
- Increased oxidation uptake and retention of LDL-C
- Upregulation and increased expression of the plasminogen activator inhibitor

Lp(a) is considered to be ten times more atherogenic than LDL cholesterol. Relative risk of CAD increases three-fold if the levels of Lp(a) are more than 30mg/dl[93].

A meta-analysis that was published in 2009, that consisted of 126,634 participants from 36 prospective studies, established that there was a constant high risk of CAD and Stroke with the high plasma level of Lp(a)[94]. It is estimated that with every 3.5-fold higher than usual Lp(a) levels is associated with a 13 percent increased risk of CHD and a 10 percent increased risk of stroke.

Additionally, a recent meta-analysis of 40 studies reported that there was a twofold increase in risk of CAD in subjects with smaller Apo(a) isoforms, which hereby correlated with higher plasma Lp(a) levels.

Another meta-analysis study by Lan Y et al,[130] considered a total of 8 studies with 86,808 participants and 8180 CAD events which showed Lp(a) as a risk factor for future CAD events, especially in the young populations.

Lp(a) and CAD in Diabetes Mellitus

There were 2 prospective studies which stated positive links between Lp(a) levels and risk of CAD in patients having Diabetes Mellitus (DM). It is noted that there is 2-3 fold risk of CAD in the presence of either DM or high Lp(a) compared to people without these two conditions. Few studies have shown an association of high Lp(a) levels with higher risk and severity of CAD in patients with DM, contrary to this, few other studies have shown a paradoxically lower risk of CAD in patients who have both elevated Lp(a) and DM.

As yet, the role of Lp(a) in DM uncertain. There are several inconsistent reports in the literature about the role of Lp(a) in DM, while some studies found no impact of DM on Lp(a) concentrations, others reported an elevation or a decrease of Lp(a) concentrations. Earlier studies were reported to have elevated Lp(a) levels in

patients with type 1 DM [126, 127, 128] as well as in patients with type 2 DM. Furthermore, few recent studies in Asian inhabitants reported an association between raised Lp(a) levels and the incidence DM.

Lp(a) and fibrinolysis

A review by Edelberg and Pizzo et al[71]has noted that the increased risk for arthritis noted in patients with elevated Lp(a) may be attributed to Lp(a)-mediated reduction of fibrinolytic activity. The Lp(a)'s increased homology with the fibrinolytic zymogen plasminogen helps the protein to bind to fibrin. Plasminogen activation is inhibited by Lp(a) due to the competitive binding with the plasminogen for access to plasminogen activators. The protein also prevents the irreversible inhibition of the activators, thereby delaying the plasminogen activation.[71]

According to Cano et al,[141] elevated levels of Lp(a) plasma concentrations may point to a possible antifibrinolytic action source. When Lp(a) levels fluctuate in vivo, a correlation has been shown between the amount of plasmin produced and the plasminogen attached to fibrin's surface. However, the fibrin-binding capacity of apo(a) polymorphs is heterogeneous, and only a small number of isoforms exhibit significant affinity for fibrin. Since small apo(a) isoforms bind fibrin more tightly, they have a stronger impact on the prognostic value of elevated Lp(a) when considered a risk factor. The prevention and treatment of elevated cardiovascular risk associated with high thrombogenic Lp(a) levels could be revolutionized by selectively neutralising Lp(a) antifibrinolytic's activity.

Lp(a) and CAD

Association of Lp(a) with CAD was first observed in 1974. Several prospective studies have corroborated the role of Lp(a) as a significant predictor of premature atherosclerotic vascular disease.[72] Lp(a) levels achieve the adult levels by 1-2 years of life and hence the pathogenesis of atherosclerosis actually begins 15-20 years before the other modifiable risk factors set in. This subsidizes to CAD in younger individuals thus precipitating premature CAD. Elevated Lp(a) values have been noted in both the genders with premature coronary atherosclerosis. Lp(a) has been recognized as an 'independent genetic risk factor' responsible for the development of CAD in men below the age of 45.[73] Enas et al,[2] have identified Lp(a) as a previously unrecognised genetic risk factor in Indian participants with malignant CAD, with considerably higher levels. Diabetes and other recognised risk factors, such as Lp(a) levels, are associated with a 2- to 3-fold increased risk of CAD. An acute ischemic cardiac event is more common in South Asians than in Europeans because of their higher risk of acute MI and second highest Lp(a) content.[74]

Zawacki et al,[132] noted that a subject with a history of early-onset CAD in the family was associated better with a raised Lp(a) level in a child (>50 mg/dL) than an elevated LDL-C level (>190 mg/dL). In such cases a cascade screening and reverse cascade screening can be beneficial and can have a high yield in detection of new cases of CAD in younger population.

Another major prospective study of South Asians to date, is the London Life Science Population Study (LOLIPOPS),[134] which studied the reasons for high vulnerability of Indians to CVD as compared to Europeans. It has proved a twofold prevalence of CAD in South Asians as compared to whites.

Lp(a) in STEMI

In a large single-center by Deora et al,[97] study of patients with ACS in 8268 subjects in India, 820 (10%) were aged <40 years. Outstandingly, 611 (75%) of the subjects aged <40 years had STEMI.

Another study by Rashid et al,[98] noted that patients with STEMI had higher values of Lp(a) than patients with NSTEMI.

Lp(a) in NSTEMI and Unstable angina

A study done by in Nepal by Timalsena et al,[58] showed no significant difference between STEMI and NSTEMI patients.

CAD due to atherosclerosis has been recognized as the most common cause of unstable angina. The prospective study by Stubbs et al,[69] has provided the first evidence on role of Lp(a) pathogenesis in unstable angina. The researchers have identified statistically significant correlation between cardiac troponin T levels and the Lp(a) concentration ($r_s = 0.2798$, $P = 0.0001$). This finding suggests the involvement of Lp(a) in failed plaque rupture stabilization. Yazici et al,[70] investigated the Lp(a) levels in subjects with unstable angina and their association with atherothrombosis and myocardial damage. The study has noted a substantially impaired fibrinolytic activity and elevated coagulation activity in the cardiac troponin-I-positive subjects. The study findings have shown that elevated Lp(a) levels may be a contributing factor for myocardial damage in subjects with unstable angina.

Indians, at a very young age are predominantly liable to premature CAD leading to AMI and eventually death. A study by Kasliwal et al,[96] done in India which consisted of 877 patients with angiographically proven CAD, observed that more than one-half of patients were less than 55 years and one-third of them were less than 45 years, with a mean age of 48 years. Double-vessel disease (DVD), triple-vessel disease (TVD), and left main coronary artery (LMCA) involvement were found in 79% of patients despite the patients being this young. Additionally, coronary atherosclerosis were mostly diffuse with multiple sites of obstruction in most of the blood vessels and 6% of CABG was performed in patients <45 years of age.

Elevated Lp(a), hypertension, and renal insufficiency are all independent predictors of CAD in patients with genetically proven heterozygous familial hypercholesterolemia. Based on the American cholesterol guidelines, those who have a family history of early CAD should be regarded to have an increased risk of developing CAD and should be evaluated for elevated Lp(a). People with a history of cardiac disease in their families, as well as those who have raised Lp(a) levels, are at an increased risk of coronary artery disease.[7]

The 2010 guidelines by the American College of Cardiology Foundation/American Heart Association for assessing cardiovascular risk in asymptomatic subjects have suggested the use of lipoprotein-associated phospholipase A2 for evaluating cardiovascular risk in asymptomatic intermediate-risk adults.[75]

Measuring Lp(a) is more advantageous for young people with a personal or family history of early vascular disease and/or frequent coronary procedures. No effective therapeutic techniques for decreasing protein levels exist because of

insufficient effective research, even though Lp(a) can be utilised to identify individuals who have a higher risk of CVD.

According to European Atherosclerosis Society, Lp(a) screening must be considered in subjects with:[76]

- Premature CAD
- Familial hypercholesterolemia
- Premature CAD and/or increased Lp(a) in the family
- Poor response to statin indicated for lowering LDL-C
- Recurrent CAD, despite receiving lipid lowering medications
- ≥5% 10-year risk of lethal CAD based on SCORE (systematic coronary risk evaluation)

The current genetic investigations confirm the importance of elevated Lp (a) in premature CVD/CHD risk, based on the substantial literature findings on this association. If Lp(a) levels are elevated, they can either trigger an anti-fibrinolytic/prothrombotic impact due to their similarity to plasminogen and plasmin as well as increase the risk of atherosclerosis.[77]

According to a review by Vavuranakis et al,[78] increased levels of Lp(a) have been associated to calcific aortic valve stenosis and atherosclerotic cardiovascular disease in humans. It is, however, possible that oxidised phospholipids can lead to unstable plaques, impaired vascular endothelial cell function, and an increased risk of acute and recurrent cardiovascular events.

There is substantial literature evidence from Asian population validating the role of Lp(a) in CAD. A 10-year prospective cohort study by Lim et al,[79] has

concluded on the role of increased Lp(a) level as a predictable risk factor for CVD in 1,183 Korean subjects with type 2 diabetes (HR, 1.92; 95% CI :1.26 to 2.92; P < 0.001). Another study by Xu et al,[80] evaluated the association of Lp(a) with long-term clinical outcomes in Chinese CKD patients who underwent percutaneous coronary intervention (PCI). The researchers have found a positive association between poor prognosis including recurrent CVD risks and Lp(a).

Elevated Lp(a) levels may alter cardiovascular outcomes in individuals with pre-DM and stable CAD, according to a prospective study conducted by Jin et al,[81] at three Chinese medical facilities. Stable CAD patients with modest glucose metabolism impairment could be stratified using CAD, according to the research. According to the findings of Saeed et al,[82] higher Lp(a) levels have been linked to aortic valve calcification and atherosclerotic cardiovascular disorders. The "Lp(a) hypothesis", in contrast to the thoroughly tested ''LDL theory", has seen very few clinical studies, according to the researchers.

Nordestgaard et al,[77] have advocated the screening for elevated Lp(a) in those at intermediate or high CVD/CHD risk, and use of niacin for Lp(a), thereby to reduce the CVD/CHD risk. Scientific and literature evidence have proposed the desirable levels for LDL and Lp(a) levels in the fasting or non-fasting state and the same has briefed below.

Table 3: Desirable levels for LDL and Lp(a) levels in the fasting or non-fasting state

	Patients with CVD and/or diabetes	Other patients and individuals	Level of evidence for treatment
LDL cholesterol	<2 mmol/L (<77 mg/dL)	<3 mmol/L (<116 mg/dL)	Meta-analysis of randomized, controlled trials of statin treatment
Lp(a)	<80th percentile (<~50 mg/dL)	<80th percentile (<~50 mg/dL)	Meta-analysis of randomized, controlled trials of niacin treatment

Source: Nordestgaard BG, Chapman MJ, Ray K, et al. Lipoprotein(a) as a cardiovascular risk factor: current status. Eur Heart J. 2010;31(23):2844-2853.

In individuals without CVD/CHD or diabetes and having intermediate or high absolute risk of CVD/CHD, treatment of high Lp(a) levels is recommended. If the statin treatment in a subject with Lp(a) >50 mg/dl, but without diabetes or CVD, decreases the risk for fatal CVD to <3% or for fatal and/or non-fatal CHD to <10%, further niacin treatment might not be required.[77]

Lp(a) is a significant risk factor for a wide range of atherosclerotic cardiovascular disorders, including ischemic stroke and MI, as well as for the development of calcific aortic stenosis. Lp(a) has been linked to an elevated risk in several ways, according to the researchers: apo(a) has increased procoagulant effects, while oxidised apolipoprotein B-related phospholipids have atherogenic and proinflammatory effects.[83]

Research by Ashfaq et al,[84] has examined the relationship between Lp(a) levels and the severity of CAD in patients from North India. Compared to the normal coronary group, there was a statistically significant difference in Lp(a) levels (P 0.0001) between the CAD group and the normal coronary group. Research shows that

Lp(a) values of 21mg/dL are linked with coronary atherosclerosis that is more severe than previously thought.

Although there are several studies linking Lp(a) with increased risk for CAD in men, very few studies have explored the association in women. A 2000 study by Foody et al,[85] evaluated the effect of gender on Lp(a) ed in a clinical setting. Lp(a) values were assessed at the outpatient prevention clinic in 918 CAD and 829 non-CAD patients (603 females, 1144 males). Lp(a) was found to be a significant risk factor in both the genders (OR=1.9, CI 1.4–2.6). The increase in risk of CAD was 22 to 35% in women <55 and from 38 to 63% in women >55. Moreover, among high-risk patients with a Lp(a) 20–44mg/dl (third quartile), younger men had a higher rate of CAD (51%) than elderly women (43%). When Lp(a) concentrations were >45 mg/dl, all genders showed significant risk. The study has concluded that elevated Lp(a) was related to a significantly higher risk of CAD in both the genders. The significant cardiovascular risk appeared to be represented by higher Lp(a) concentrations in women than in males in this cohort of high-risk patients.

Lacunae in literature

Despite the fact that numerous worldwide and epidemiological research have found a favourable correlation between Lp(a) levels and CHD, there is a lack of evidence-based data from the Indian subcontinent. Newer diagnostic techniques and management strategies may benefit from understanding the function of Lp(a) as a key risk factor for cardiovascular disease (CVD) in the country. As a result, the current research from India is inadequately thorough to accurately analyse the relationship between Lp(a) and CHD.

MATERIALS AND METHODS

Study site

The present study was done in the Department of General Medicine at Jawaharlal Nehru Medical College, KLES Dr. Prabhakar Kore Hospital, Belagavi.

Study design

The study was a one-year hospital-based cross-sectional study.

Period of study

1STJanuary 2020 to 31ST December 2020

Study population

The study was conducted on patients admitted in the ICCU and Medicine wards at KLES Dr. Prabhakar Kore Hospital, Belagavi fulfilling the inclusion criteria.

The inclusion and exclusion criteria considered are briefed below:

Inclusion criteria

- Patients between 18 to 49 years of age
- Criteria for CAD:

    1. History of MI

    2. Angiography proved coronary artery lesion

Exclusion criteria

- Patients above 49 years of age
- Patients on statins (more than 1 week therapy)
- Known case PCOD
- Pregnancy

Ethical considerations

The institutional human ethics committee approved the study. The participants were included in the study only after getting informed written consent.

Informed consent

All patients were explained about the study and their informed written consent was obtained. The study participant's privacy was protected.

Sample size

$$n = \frac{z_\alpha{}^2 P(1-P)}{d^2}$$

$$= 87 \sim \mathbf{90}$$

z= 1.96 at 95% confidence

p= 35%

q= 65%

d= 10% = p-P = absolute error

Data collection

All relevant parameters were documented in a structured Study Proforma.

Methodology

Patients between the age group 18-49 years of age who were admitted in ICCU and Medicine ward were included in the study. All cases were interviewed using a questionnaire. For each participant, medical history, clinical examination was done, height, weight were measured and BMI was calculated. Complete blood count (CBC), Fasting Lipid Profile (FLP), Lipoprotein(a), Cardiac enzymes, ECG, ECHO, X-RAY, and Coronary Angiography (CAG) reports were performed.

Angiography evaluation

Coronary angiography procedure (CAG) was performed using the Seldinger puncture needle, Judkin's technique and was evaluated by an interventional cardiologist who was blinded by the values of serum Lp(a) in the study patients. Based on CAG, CAD is defined as "the presence of at least a > 50% stenosis of major coronary arteries (left anterior descending, left circumflex, or right coronary arteries) or their major branches (diagonal, obtuse marginal, posterior descending, or posterior left ventricular arteries)."

Measurement of Lp(a)

Particle enhanced immunoturbidimetric assay method was used to determine the Lp(a) levels in the study population. A value of >30mg/dl was considered high in the patients.

Statistical analysis

T-test, chi-square one-way ANOVA were used to evaluate continuous data, and Excel 2007 was utilised for categorical data. By utilising a bar graph and pie chart, the distribution of the variables were depicted such as age, gender, comorbidities, alcohol and tobacco use, lipoprotein levels, and the number of vessels involved (SVD, DVD, and TVD) (excel 2103 16.0.13901.20400). Excel was used to perform correlations of SVD, DVD, TVD, Lipoprotein(a) levels, Total Cholesterol, TGL, HDL and LDL.

RESULTS

A total of 104 participants were included in the final analysis.

Majority of study population 70 (67.31%) had higher level of Lp(a) and 34 (32.69%) had normal Lp(a) level

Figure 5: Overall Lp(a) levels in the study population:

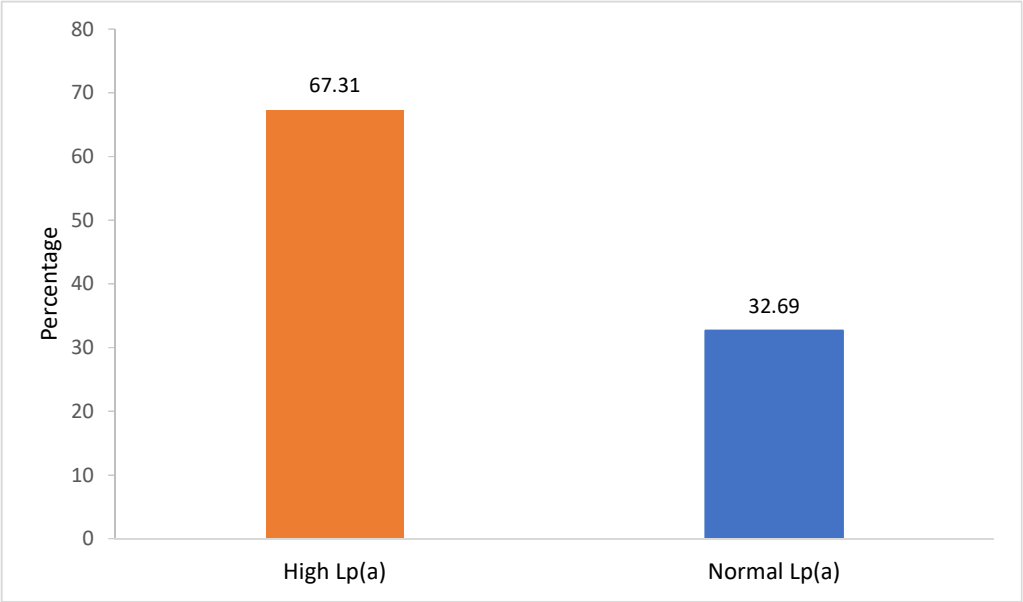


Table 4: Descriptive analysis showing gender distribution in the study population: (N=104)

Gender	No. of subjects (%)
Male	85 (81.73%)
Female	19 (18.27%)

Among the study population, 85 (81.73%) were males and 19(18.27%) were females.

Figure 6: Gender distribution in the study population:

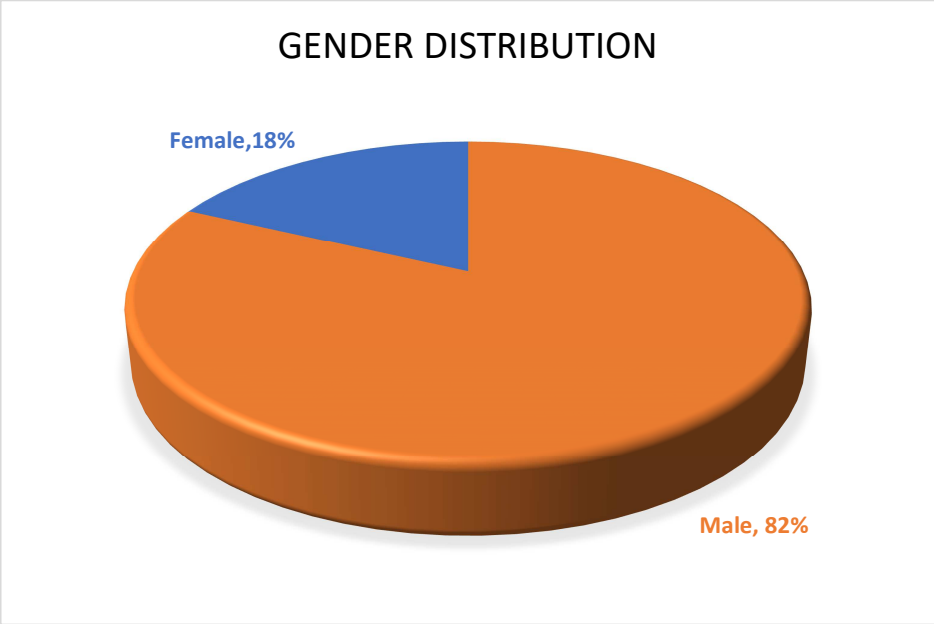


Figure 7: Bar graph depicting the average Lp(a) in males and females in the study population

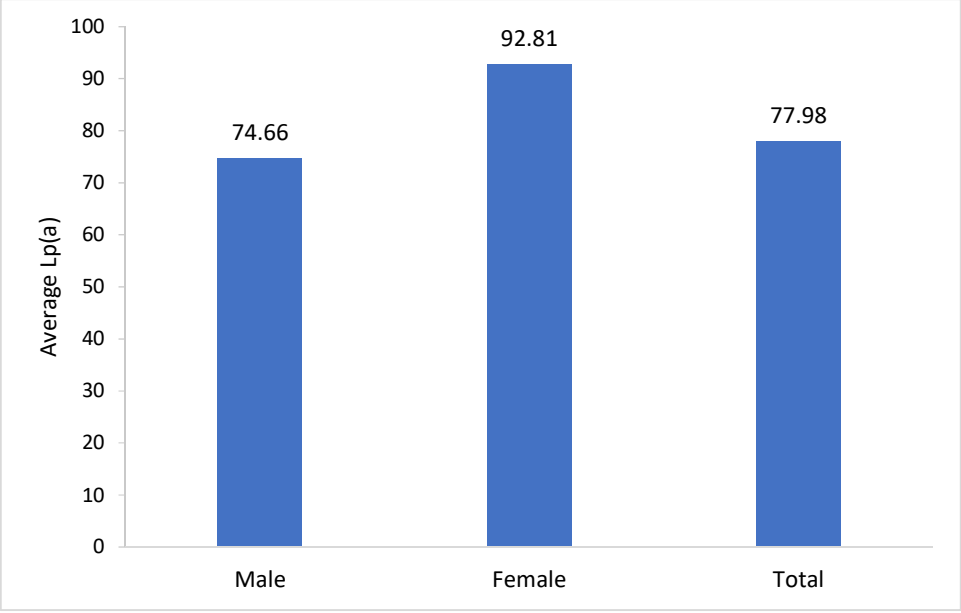


Table 5: Descriptive analysis showing the Lp(a) levels in males and females (N=104)

Sex	Lp(a)				Total
	High		Normal		
	n1	%	n2	%	n
Male	51	60.0	26	30.6	85
Female	12	63.2	4	21.1	19

n1- number of subjects with high Lp(a)
n2- number of subjects with normal Lp(a)
n- total number of subjects

The study showed that among the study population, 63% (12) subjects were female who had high Lp(a) and 60% (51) were male with high Lp(a). Whereas, 21% (4) were females with normal Lp(a), 31% (26) were males with normal Lp(a).

Table 6: Descriptive analysis of male and females with their average Lp(a) value

Gender	Lp(a)	N	Mean ± SD	p-value
Male	**High**	56	105.69 ± 61.22	
	Normal	29	14.74 ± 7.61	
Female	**High**	14	117.34 ± 76.59	
	Normal	5	24.12 ± 5.03	0.297
Total		104	77.98 ± 68.22	

Among the males (85) in the study population, 56 of had high Lp(a) and their average Lp(a) value was found to be 105.69 ± 61.22 and those with normal Lp(a) levels had 14.74 ± 7.61, and among the females (19) in the study population who had high Lp(a) had an average of 117.34 ± 76.59 and those who had normal Lp(a) was 24.12 ± 5.03

Table 7: Descriptive analysis of number of subjects in the age group category (N=104)

Age group (year)	No of subjects	%
18-30	10	9.62
31-40	30	28.85
41-49	64	61.54

10% (10) subjects belonged to the age group 18-30, 29% (30) belonged to 31-40, 62% (64) belonged to 41-49 age group

Figure 8: Bar graph depicting the distribution of patients in age group categories

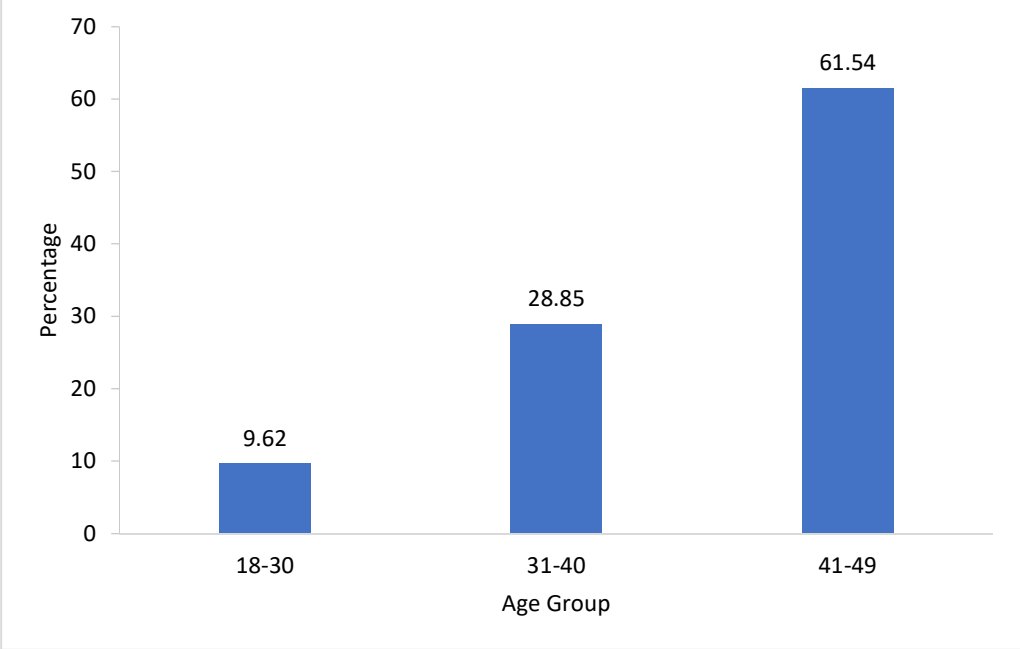


Table 8: Descriptive analysis of the average Lp(a) in the study population according to the age groups

Age group	High Lp(a)		Normal Lp(a)	
	Mean ± SD	p-value	Mean ± SD	p-value
18-30	75.67 ± 18.86		18.08 ± 8.55	
31-40	106.87 ± 54.91		14.7 ± 6.17	
41-49	113.1 ± 71.45	0.412	16.36 ± 8.78	0.773
Total	77.97±68.21			

The analysis showed that the average Lp(a) in the age groups 18-30 years of age was 75.67 ± 18.86 mg/dl in the high Lp(a) category and 18.08 ± 8.55mg/dl in the normal Lp(a) category.

In the age group of 31-40 was 106.87 ± 54.91 mg/dl and 14.7 ± 6.17mg/dl with normal range of Lp(a) and that of age group 41-49 was 113.1 ± 71.45 mg/dl and 16.36 ± 8.78 mg/dl and the overall average value of Lp(a) was 77.97±68.21 mg/dl.

Figure 9: Bar graph depicting the overall Lipid profile in the study population

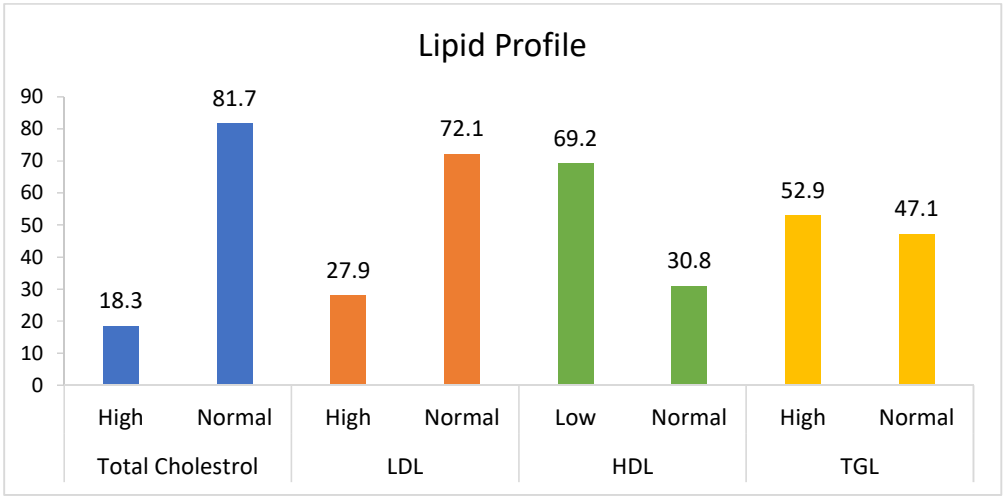


Table 9: Descriptive analysis of average value of Lipid profile in the study population

	Total Cholesterol	**LDL**	**HDL**	**TGL**
N	104	104	104	104
Mean	156.45	82.13	37.40	170.41
Std. Deviation	46.52	37.20	11.25	90.93

N- number of patients

In our study population, the average values of Total Cholesterol was 156.45 ± 46.52 mg/dl, LDL was 82.13 ± 37.2 mg/dl, that of HDL was 37.4 ± 11.25 mg/dl and TGL was 170.41 ± 90.93 mg/dl

Table 10: Descriptive analysis of comparison of Lp(a) levels and Total Cholesterol

Total Cholesterol	Lp(a) Level	N	Mean ± SD	p-value
Normal	**High**	58	109.33 ± 65.95	0.518
	Normal	27	17.09 ± 8.34	
High	**High**	12	101.68 ± 56.73	
	Normal	7	12.39 ± 5.28	
Total		104	77.98 ± 68.22	

N- Number of patients

Among the study population subjects with normal Total cholesterol and high Lp(a) (58), the average Lp(a) value was 109.33 ± 65.95 mg/dl, and among those with normal Lp(a)(27) it was 17.09 ± 8.34 mg/dl.

Among those with high Total cholesterol and high Lp(a) (12) the average Lp(a) was 101.68 ± 56.73 mg/dl and that with normal Lp(a) was 12.39 ± 5.28 mg/dl.

Table 11: Descriptive analysis of comparison of Lp(a) levels and LDL in the study population

LDL	Lp(a) Level	N	Mean ± SD	p-value
Normal	**High**	54	110.52 ± 62.8	0.128
	Normal	21	16.94 ± 8.9	
High	**High**	16	99.61 ± 69.97	
	Normal	13	14.8 ± 6.31	
Total		104	77.98 ± 68.22	

Among the study population subjects with normal LDL and high Lp(a) (54) the average Lp(a) value was 110.52 ± 62.8mg/dl, and among those with normal Lp(a) (21) it was 16.94 ± 8.9 mg/dl

Among those with high LDL and high Lp(a) (16) the average Lp(a) was 99.61 ± 69.97 mg/dl and that with normal Lp(a) was 14.8 ± 6.31mg/dl.

Table 12: Descriptive analysis of comparison of HDL and Lp(a) values in the study population

HDL	Lp(a)	N	Mean ± SD	p-value
Normal	**High**	24	126.79 ± 73.52	0.029
	Normal	8	18.56 ± 7.52	
Low	**High**	46	98.23 ± 57.1	
	Normal	26	15.37 ± 8.1	
Total		104	77.98 ± 68.22	

Among the study population, subjects with normal HDL and high Lp(a) (24) their average Lp(a) value was 126.79 ± 73.52mg/dl, and those with normal HDL (8) and was 18.56 ± 7.52 mg/dl.

Among the subjects with low HDL and high Lp(a) (46) the average Lp(a) was 98.23 ± 57.1 mg/dl and that with normal Lp(a) (26) was 15.37 ± 8.1 mg/dl.

Table 13: Descriptive analysis of comparison of Lp(a) and TGL values in the study population

TGL	Lp(a)	N	Mean ± SD	p-value
Normal	**High**	30	127.32 ± 77.15	0.347
	Normal	19	17.36 ± 8.5	
High	**High**	40	93.55 ± 48.5	
	Normal	15	14.56 ± 7.24	
Total		104	77.98 ± 68.22	

Among the study population, subjects with normal TGL and high Lp(a) (30) their average Lp(a) value was 127.32 ± 77.15mg/dl, and those with normal HDL (19) and was 17.36 ± 8.5 mg/dl.

Among the subjects with high TGL and high Lp(a) (40) the average Lp(a) was 93.55 ± 48.5 mg/dl and that with normal Lp(a) (15) was 14.56 ± 7.24 mg/dl.

Figure 10: Bar graph depicting the LDL levels in the study population

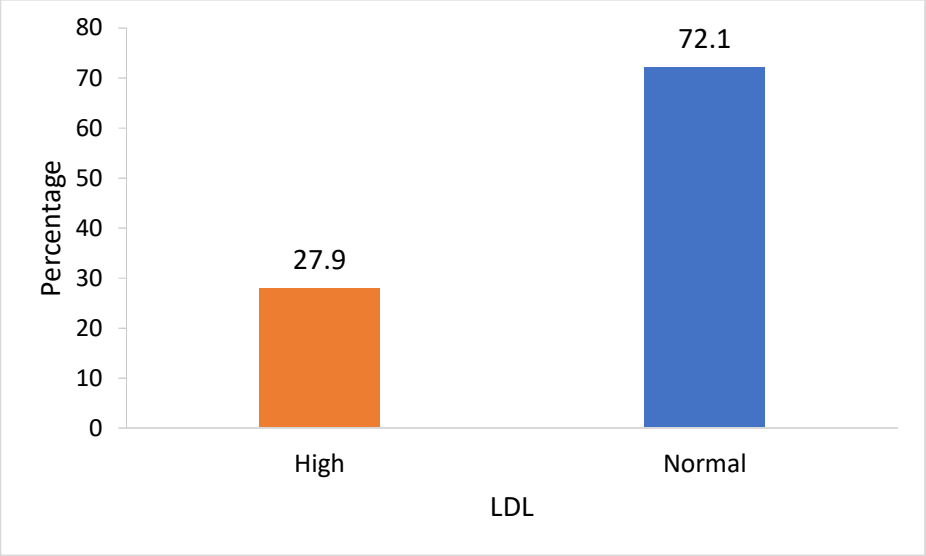


Table 14: Descriptive analysis of Lp(a) and Lipid profile in the study population

Lipid Profile		Lp(a)				Total	
		High		Normal			
		n1	%	n2	%	n	%
Total Cholesterol	**High**	12	17.1	7	20.6	19	18.3
	Normal	58	82.9	27	79.4	85	81.7
LDL	**High**	16	22.9	13	38.2	29	27.9
	Normal	54	77.1	21	61.8	75	72.1
HDL	**Low**	46	65.7	26	76.5	72	69.2
	Normal	24	34.3	8	23.5	32	30.8
TGL	**High**	40	57.1	15	44.1	55	52.9
	Normal	30	42.9	19	55.9	49	47.1

n1- number of subjects with high Lp(a)

n2- number of subjects with normal Lp(a)

The subjects with high Lp(a) and high TC were 12 (17%) and with normal TC were (58)83%.

16 (23%) subjects with high Lp(a) had high LDL, 54 (77%) had high Lp(a) and normal LDL.

46 (66%) had high Lp(a) and low HDL, 34% (24) had high Lp(a) and normal HDL.

57% (40) had high Lp(a) and high TGL and 43% (30) had high Lp(a) and normal TGL.

21% (7) had normal Lp(a) with high TC, 79% (27) had normal Lp(a) and normal TC.

38% (13) had normal Lp(a) with high LDL, 62% (21) had normal Lp(a) and normal LDL.

77% (26) had normal Lp(a) with low HDL, 24% (8) had normal Lp(a) and normal HDL.

44% (15) had normal Lp(a) with high TGL, 56% had normal Lp(a) and normal HDL

Figure 11: Bar graph depicting the comparison of Lp(a) with LDL in the study population

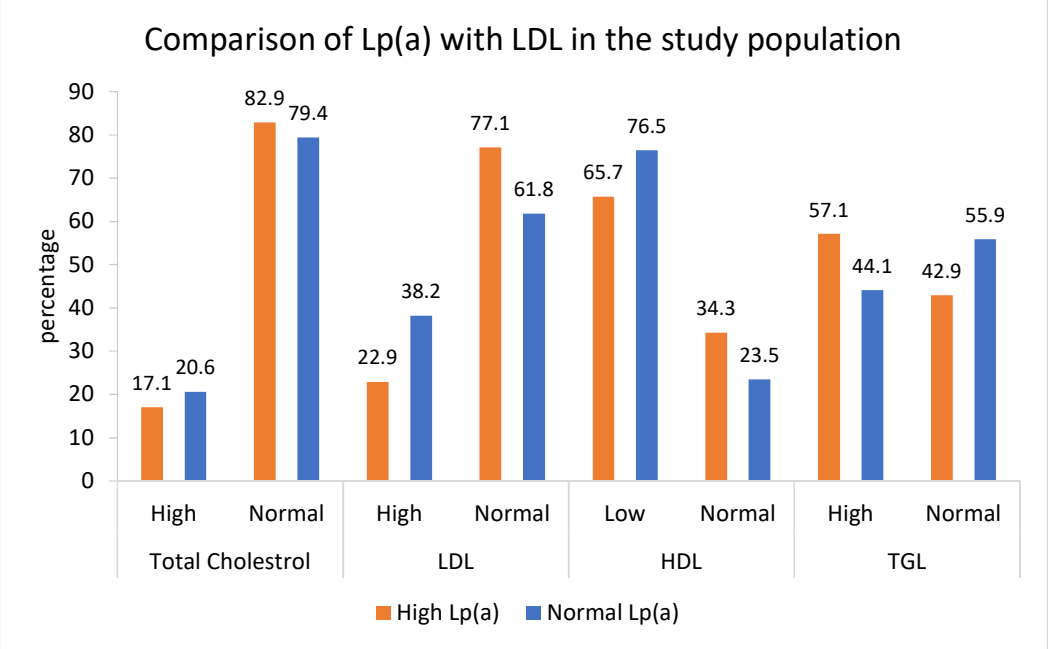


Table 15: Descriptive analysis of BMI in the study population

BMI	Frequency	Percent	Average BMI(kg/m^2)	Std. deviation
Normal	7	6.7	20.46	1.58
Overweight	14	13.5	24.08	0.54
Obese	83	79.8	29.22	2.97
Total	104		27.93	3.79

In the study population, 7% were of normal BMI, 14% were overweight, 80% were obese. Maximum of the study population belong to the obese category of BMI. The average BMI in obese subjects was found to be 29.22kg/m^2, overweight subjects was 24.08 and that of normal subjects was 20.46kg/m^2

Figure 12: BMI in the study population

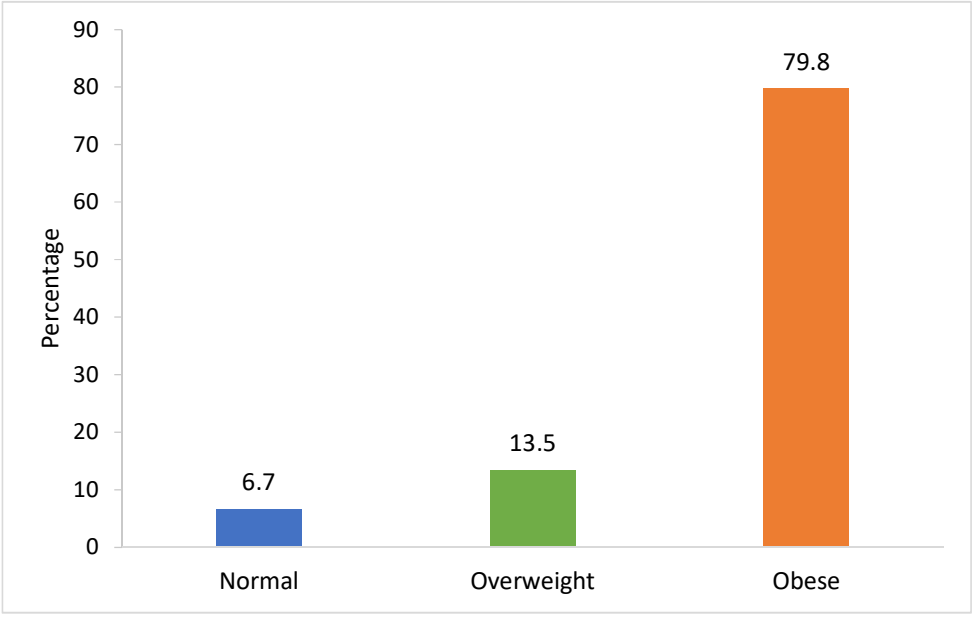


Table 16: Descriptive analysis of Lp(a) and BMI in the study population

BMI	Lp(a)			
	Mean	N	Std. Deviation	P-value
Normal	115.41	7	126.23	0.317
Overweight	79.20	14	55.72	
Obese	74.61	83	63.68	

In our study it was noted that 14 of the subjects were overweight and had an average value of Lp(a) of 79.20 ± 55.72 mg/dl. 83 of them were obese and had an average Lp(a) of 74.61 ± 63.68 mg/dl.

Figure 13: Bar graph depicting the use of tobacco and alcohol in the study population

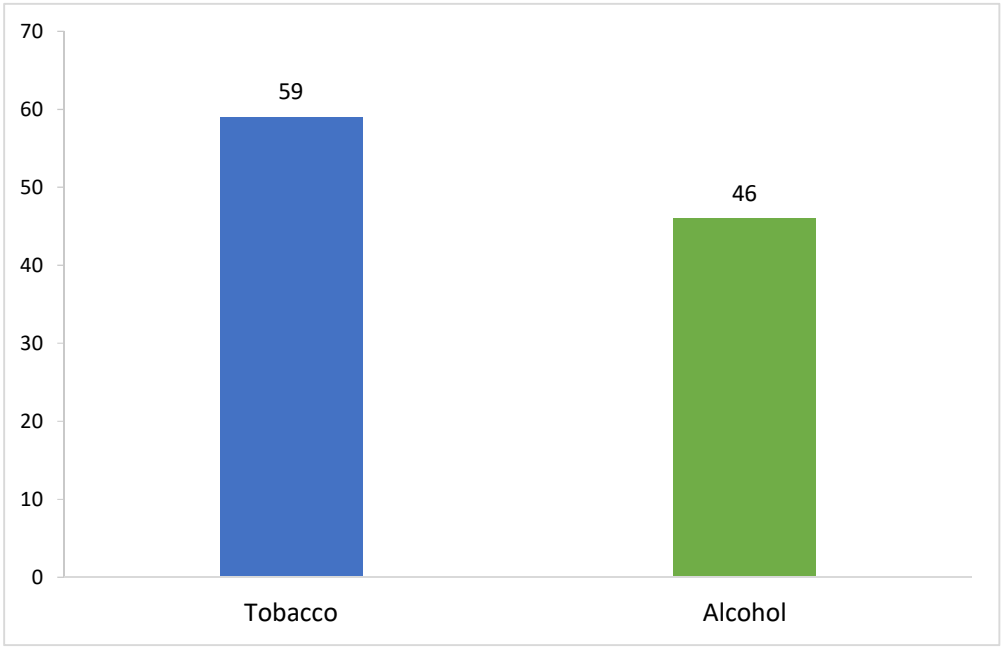


Table 17: Descriptive analysis of the average Lp(a) value in subjects who consumed tobacco.

Tobacco	High Lp(a)		Normal Lp(a)		Total	
	Mean±SD	p-value	Mean±SD	p-value	Mean±SD	p-value
Yes	95.99 ± 57.35	0.128	14.81 ± 7.58	0.112	61.59 ± 59.43	0.005
No	119.38 ± 68.82		19.75 ± 8.35		99.45 ±73.52	

In the subject who consumed tobacco, the average Lp(a) value was 61.59 ± 59.43 mg/dl and those who did not consume was 99.45±73.52 mg/dl.

Figure 14: Bar graph showing the comorbidities in the first degree relatives of the study population.

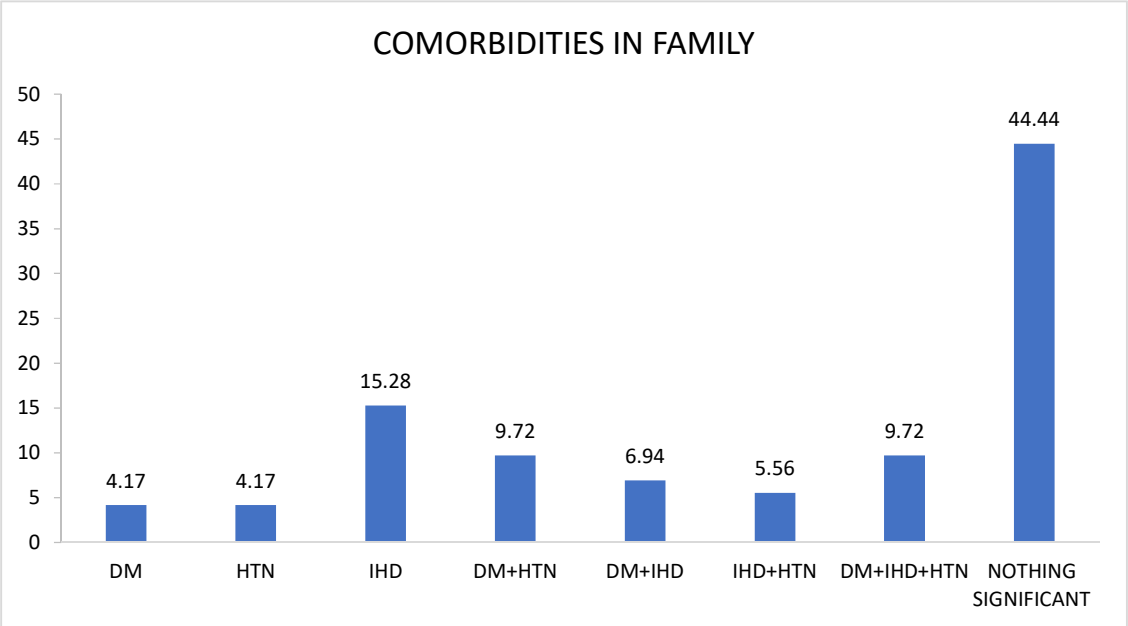


Table 18: Descriptive analysis of comorbidities in the study population

Comorbidities	N	Percent
No DM & No HTN	19	18.3
HTN and No DM	22	21.2
DM and No HTN	21	20.2
DM and HTN	42	40.4
Total	104	100

N-number of patients

Among the study population, 18% (19) had no comorbidities, 21% (22) had only HTN, 20% (21) had only DM and 40% (42) had both DM and HTN.

Figure 15: Bar graph depicting the comorbidities in the study population

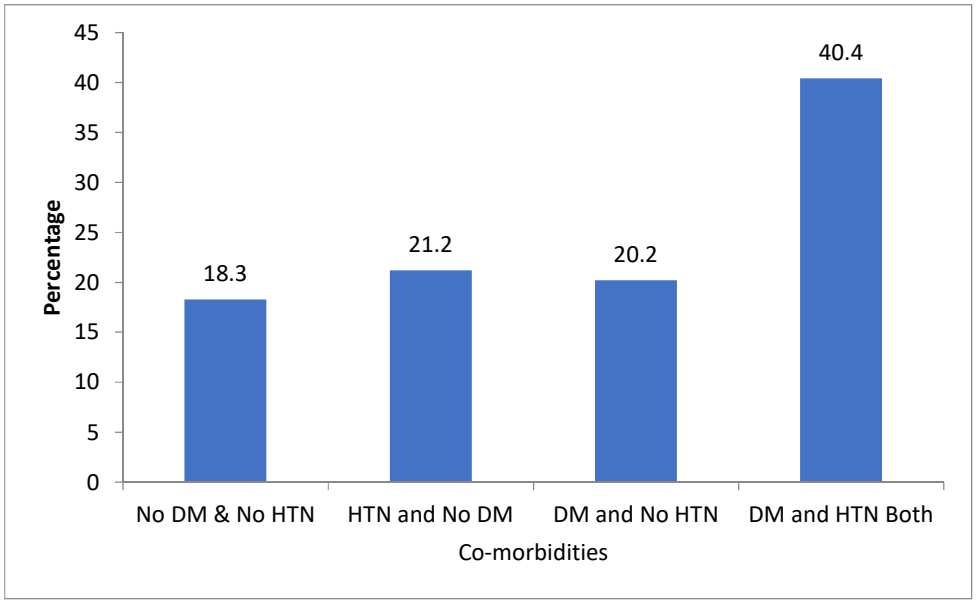


Table 19: Descriptive analysis of subjects with comorbidities in the study population

Lp(a)	Co-morbidities	N	Lp(a) level		
			Mean	Std. Deviation	Median
High	**No DM & No HTN**	16	106.24	64.80	98.35
	HTN and No DM	9	91.24	63.20	70.80
	DM and No HTN	11	82.70	28.17	83.00
	DM and HTN Both	27	116.28	74.01	97.30
	Total	63	104.29	64.24	90.20
Normal	**No DM & No HTN**	2	22.90	7.92	22.90
	HTN and No DM	11	14.69	8.68	10.60
	DM and No HTN	7	19.74	7.76	21.00
	DM and HTN Both	10	14.58	8.50	11.85
	Total	30	16.38	8.42	13.35
Total	**No DM & No HTN**	18	96.98	66.60	79.65
	HTN and No DM	20	49.14	56.99	28.20
	DM and No HTN	18	58.22	38.54	50.75
	DM and HTN Both	37	88.79	77.91	72.40
	Total	93	75.93	67.16	57.00

In the study population, subjects with high Lp(a) in subjects with no co-morbidities had an average of 98.35 mg/dl, in patients with HTN and no DM had an average value of 70.8 mg/dl, patients with DM and no HTN had an average value of 83, whereas subjects with both co-morbidities had 97.3 mg/dl.

Subjects with normal Lp(a) in subjects with no co-morbidities had an average of 22.9 mg/dl, in patients with HTN and no DM had an average value of 10.6 mg/dl, patients with DM and no HTN had an average value of 21 mg/dl, whereas subjects with both co-morbidities had 11.85 mg/dl.

In total, 79.65 mg/dl was the average in subjects with no comorbidities, 28.2 mg/dl in subjects with HTN only, 50.75 mg/dl in DM only and 72.4 mg/dl in both comorbidities.

Table 20: Descriptive analysis of Co-morbidities and Lp(a) in the study population.

Co-morbidities	Lp(a)	N	Mean ± SD	p-value
None	**High**	28	117.24 ± 72.8	0.038
	Normal	14	14.47 ± 7.22	
DM and No HTN	**High**	11	89.08 ± 58.74	
	Normal	11	14.69 ± 8.68	
No DM and HTN	**High**	14	89.4 ± 42.63	
	Normal	7	19.74 ± 7.76	
DM and HTN	**High**	17	120.44 ± 65.62	
	Normal	2	22.9 ± 7.92	
	Total	104	77.98 ± 68.22	

The average Lp(a) among the subjects with no comorbidities but high Lp(a) the average Lp(a) was 117.24 ± 72.8mg/dl, and those with normal Lp(a) was 14.47 ± 7.22 mg/dl

Among those with DM only as a comorbidity with high Lp(a) the average was 89.08 ± 58.74mg/dl, and with normal Lp(a) was 14.69 ± 8.68mg/dl

Among those with HTN only as a comorbidity with high Lp(a) the average was 89.4 ± 42.63mg/dl, and with normal Lp(a) was 19.74 ± 7.76mg/dl

And those with both DM and HTN, those with high Lp(a) the average Lp(a) was 120.44 ± 65.62mg/dl and with normal Lp(a) range it was 22.9 ± 7.92mg/dl

The overall average Lp(a) in the study population was 77.98± 68.22 mg/dl

Table 21: Descriptive analysis of subjects with Diabetes

Lp(a)	DIABETES	N	Mean	Std. Deviation	p-value
High	Yes	42	107.53	70.28	0.315
	No	28	89.91	61.45	
Normal	Yes	21	18.64	13.05	0.09
	No	13	52.66	63.58	

Table 22: Descriptive analysis depicting the levels of Lp(a) in subjects with Diabetes and Hypertension

Co-morbidities		Lp(a)			
		High		Normal	
		n1	%	n2	%
DIABETES	Yes	25	61	13	31.7
	No	38	60.3	17	27
HYPERTENSION	Yes	27	67.5	9	22.5
	No	36	56.3	21	32.8

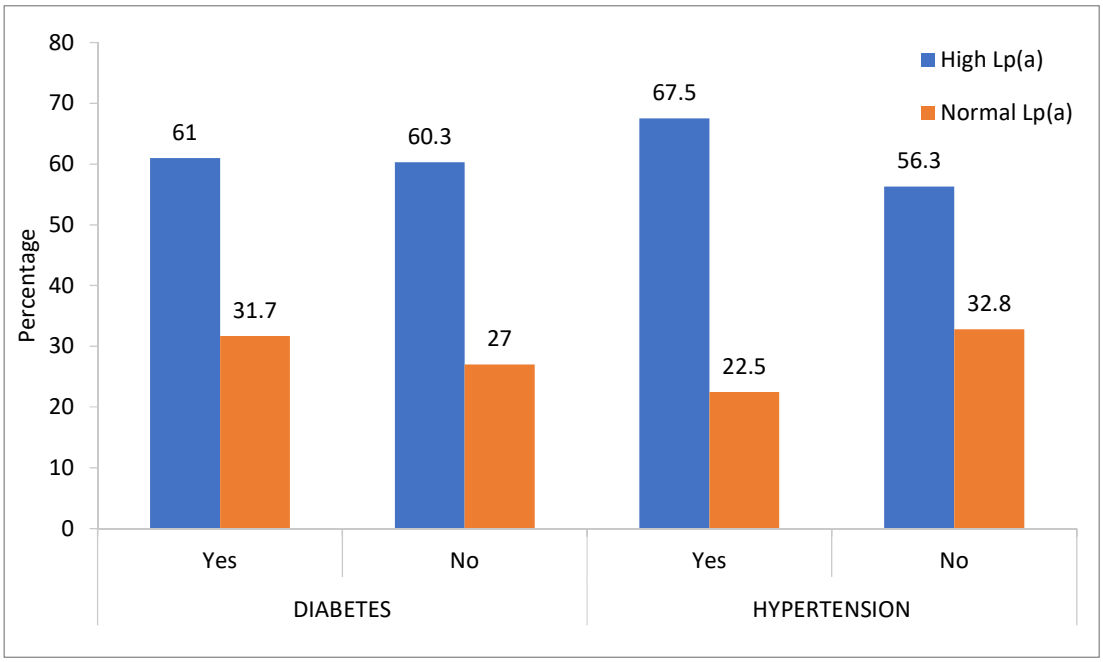
n 1: number of subjects with high Lp(a)

n2: number of subjects with normal Lp(a)

In the study population, 25 subjects with high Lp(a) and diabetes and 27 had hypertension.

Whereas, subjects with normal Lp(a) 13 had diabetes and 9 had hypertension.

Figure 16: Bar graph depicting the levels of Lp(a) in subjects with Diabetes and Hypertension



Among the subjects who had Diabetes, 61% had high Lp(a) and 32% had normal Lp(a); subjects with no Diabetes 60% had high Lp(a) and 27% had normal Lp(a).

Among the subjects who had Hypertension, 68% had high Lp(a) and 23% had normal Lp(a); subjects with no Hypertension 56% had high Lp(a) and 33% had normal Lp(a).

There was no correlation seen in the levels of Lp(a) and the comorbidities in th study population.

Table 23: Descriptive analysis of type of coronary artery disease in the study population.

Diagnosis	Lp(a)				p-value
	n	Mean	Std. Deviation	Median	
STEMI	43	76.83	59.20	72.4	0.907
NSTEMI	41	79.25	75.49	57.0	
UNSTABLE ANGINA	20	71.11	66.42	33.1	
Total	104	76.68	66.82	61.45	

n- number of patients

Among the subjects, 43 subjects presented with STEMI with an average value of Lp(a) of 72.4 mg/dl, 41 subjects presented with NSTEMI with average Lp(a) value of 57 mg/dl and 20 subjects presented with Unstable Angina with average Lp(a) of 33.1 mg/dl

Figure 17: Bar graph depicting the percentage of population with Lp(a) levels in type of CAD

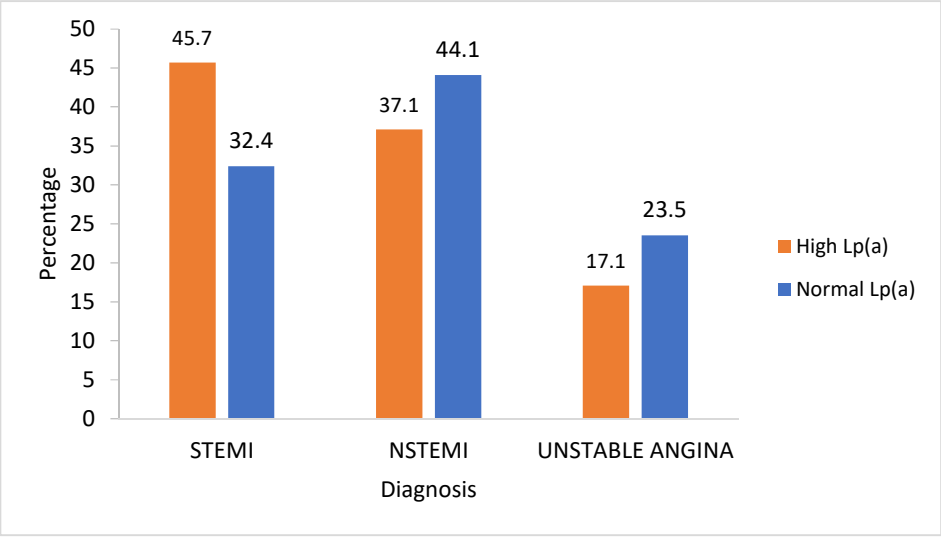


Table 24: Descriptive analysis of CAD and Lp(a) in the study population

Diagnosis	Lp(a)	N	Mean ± SD	p-value	p-value
STEMI	**High**	32	97.84 ± 53.86	0.000	0.814
	Normal	11	13.08 ± 6.89		
NSTEMI	**High**	28	112.77 ± 74.53	0.000	
	Normal	13	18.75 ± 8.26		
UNSTABLE ANGINA	**High**	10	127.33 ± 63.55	0.000	
	Normal	10	16.05 ± 8.26		
Total		104	77.98 ± 68.22		

Among the study population, subjects with STEMI with high Lp(a) were 32 with an average Lp(a) of 97.84 ± 53.86 mg/dl, those with normal Lp(a) were 11 with average Lp(a) of 13.08 ± 6.89mg/dl.

In those with NSTEMI with high Lp(a) were 28 with an average Lp(a) of 112.77 ± 74.53 mg/dl, those with normal Lp(a) were 13 with average Lp(a) of 18.75 ± 8.26 mg/dl.

In those with Unstable Angina with high Lp(a) were 10 with an average Lp(a) of 127.33 ± 63.55 mg/dl, those with normal Lp(a) were 10 with average Lp(a) of 16.05 ± 8.26 mg/dl

Table 25: Descriptive analysis Levels of Lp(a) with CAD in the study population

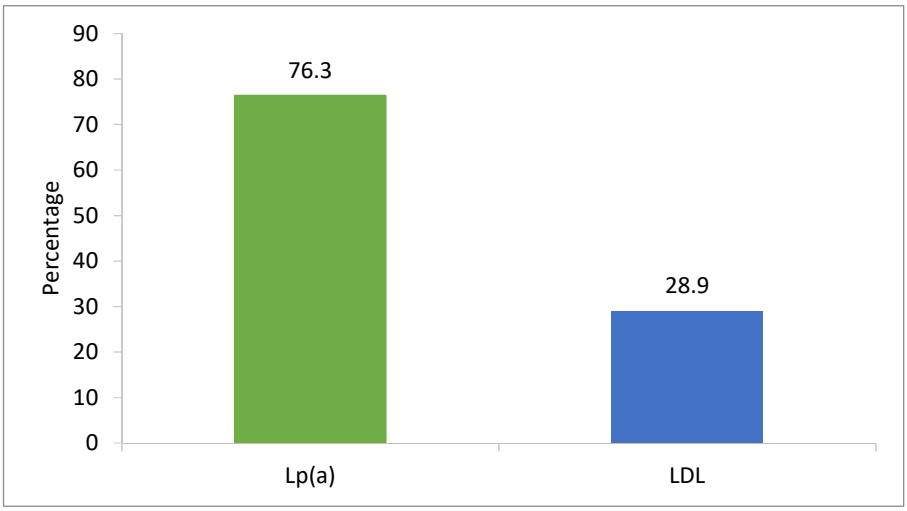
Lp(a)	Diagnosis	n	Mean	Std. Deviation	Median	p-value
High	**STEMI**	32	98.33	53.49	84.30	0.000
	NSTEMI	26	114.43	74.60	96.65	
	UNSTABLE ANGINA	12	108.78	60.91	104.50	
	Total	70	106.10	62.84	96.00	
Normal	**STEMI**	11	14.30	8.35	10.60	
	NSTEMI	15	18.27	7.66	17.30	
	UNSTABLE ANGINA	8	14.61	8.07	11.80	
	Total	34	16.12	7.98	13.35	
Total	**STEMI**	43	76.83	59.20	72.40	
	NSTEMI	41	79.25	75.49	57.00	
	UNSTABLE ANGINA	20	71.11	66.42	33.10	
	Total	104	76.68	66.82	61.45	

n- number of subjects

The above table depicts the relation of Lp(a) and the type of CAD in the subjects. Among the subjects with high Lp(a) 32 had STEMI, 26 had NSTEMI and 12 had unstable angina, with average Lp(a) of 84.3 mg/dl, 96.65 mg/dl and 104.5 mg/dl respectively.

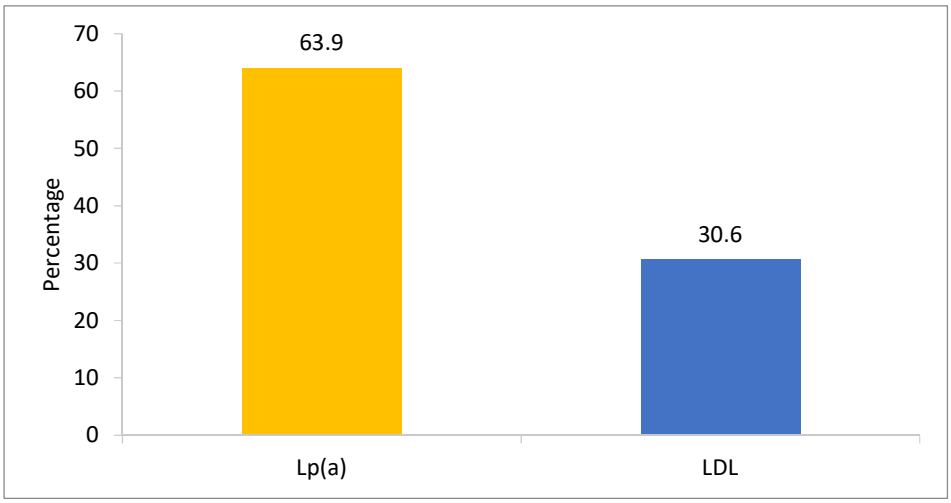
In subjects with normal Lp(a), 11 had STEMI, 15 had NSTEMI and 8 had unstable angina with average Lp(a) of 10.6 mg/dl, 17.3 mg/dl and 13.35 mg/dl respectively.

Figure 18: Bar graph depicting the comparison of Lp(a) levels with LDL levels with subjects with STEMI



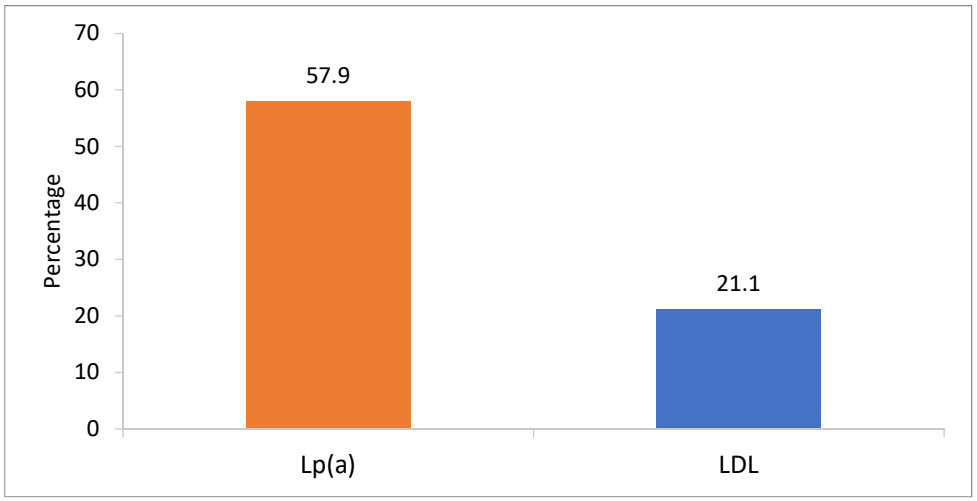
The graph depicts the higher prevalence of Lp(a) over LDL in subjects with STEMI in the study population.

Figure 19: Bar graph depicting the the comparison of Lp(a) levels with LDL levels with subjects with NSTEMI



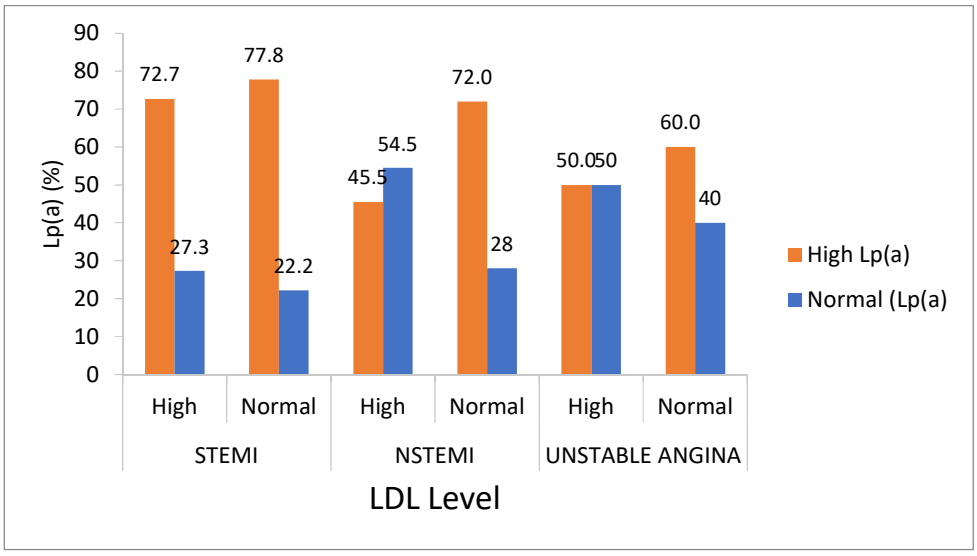
The graph depicts the higher prevalence of Lp(a) over LDL in subjects with NSTEMI in the study population

Figure 20: Bar graph depicting the comparison of Lp(a) levels with LDL levels with subjects with Unstable Angina



The graph depicts the higher prevalence of Lp(a) over LDL in subjects with Unstable Angina in the study population.

Figure 21: Bar graph depicting the comparison of Lp(a) and LDL in study population



The above graph shows the comparison of Lp(a) levels with LDL in all the subjects with CAD. It shows that the Lp(a) levels are higher as compared to LDL in all the subjects.

Table 26: Descriptive analysis depicting the Lp(a) levels and their CAG findings

ANGIOGRAPHY	Lp(a)			
	N	Mean	Std. Deviation	Median
NORMAL	6	55.233	46.612	52.200
SVD	61	75.551	72.104	52.500
DVD	33	82.073	62.655	70.800
TVD	4	81.700	50.055	99.400
Total	104	76.685	66.817	61.450

Descriptive analysis of the coronary angiography revealed that 6 had normal angiography with average Lp(a) of 52.2 mg/dl, 61 had single vessel disease (SVD) with average Lp(a) of 52.5 mg/dl, 33 had double vessel disease with average Lp(a) of 70.8 mg/dl, and 4 had triple vessel disease (TVD) with average Lp(a) of 99.4 mg/dl.

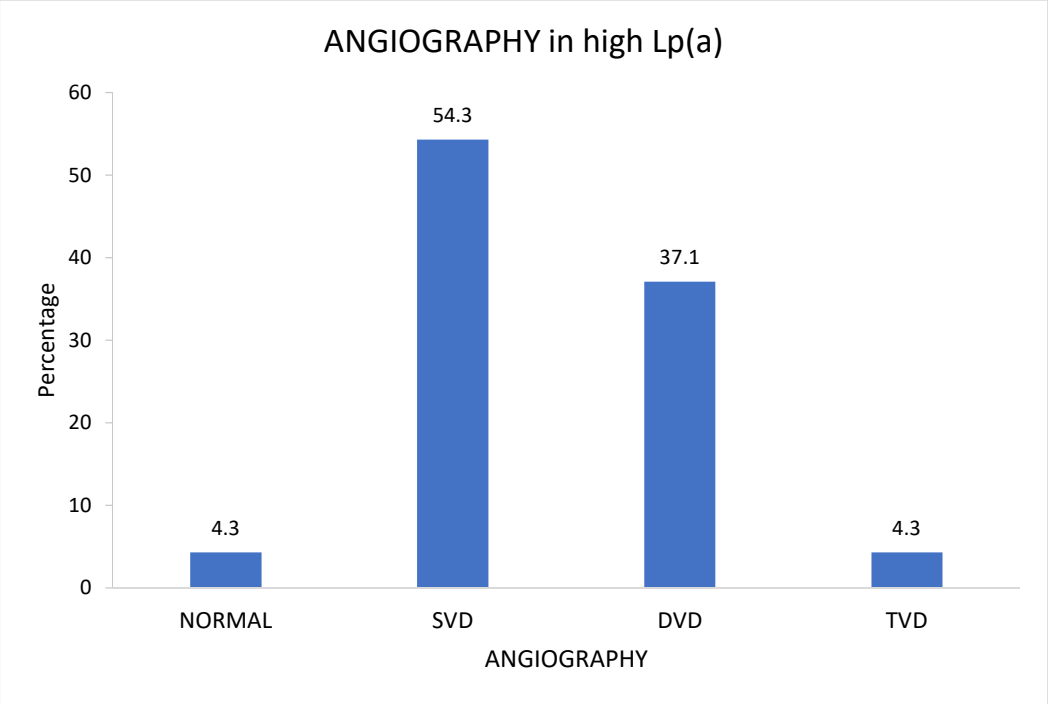
Table 27: Descritpive analysis of Lp(a) in subjects in their CAG in the study population

ANGIOGRAPHY	Lp(a)			
	High		Normal	
	n1	%	n2	%
NORMAL	3	4.3	3	8.8
SVD	38	54.3	23	67.6
DVD	26	37.1	7	20.6
TVD	3	4.3	1	2.9

n1- Number of subjects with high Lp(a),
n2 – Number of subjects with normal Lp(a)

In our study population, 3 (4.3%) subjects had normal CAG with high Lp(a), and 8.8% (3) had normal Lp(a), In subjects with SVD, 54.3 % (38) had high Lp(a) and 67.5% (23) had normal Lp(a). In subjects with DVD, 37.1% (26) had high Lp(a) and 20.6% (7) had normal Lp(a) and in subjects TVD, 4.3% (3) had high Lp(a) and 2.9% (1) had normal Lp(a).

Figure 22: Bar graph depicting the CAG in subjects with high Lp(a) in the study population



Among the study population who had high Lp(a), 4.3% had no coronary artery lesion, where as 54.3% had involvement of a single vessel and 37.1% had double vessel involvement and 4.3% had triple vessel involvement.

Table 28: Descriptive analysis of Lp(a) levels with CAG findings in the study population.

ANGIOGRAPHY	Lp(a) Level	N	Mean ± SD	p-value	p-value
Normal	**High**	3	96.7 ± 14.34	0.001	0.751
	Normal	3	13.77 ± 8.21		
SVD	**High**	38	110.93 ± 70.58	0.000	
	Normal	23	17.1 ± 8.36		
DVD	**High**	26	105.32 ± 62.57	0.001	
	Normal	7	14.96 ± 7.35		
TVD	**High**	3	106 ± 14.67	0.029	
	Normal	1	8.8		
Total		104	77.98 ± 68.22		

Among the subjects with normal CAG and high Lp(a) (3) the average Lp(a) was 96.7 ± 14.34 mg/dl, and that with normal Lp(a) (3) was 13.77 ± 8.21 mg/dl.

In the subjects with SVD in CAG with high Lp(a) (38), average Lp(a) was 110.93 ± 70.58 mg/dl and those with normal Lp(a) (23) was 17.1 ± 8.36 mg/dl.

Among the subjects with DVD on CAG and high Lp(a) (26) the average Lp(a) was 105.32 ± 62.57mg/dl, and that with normal Lp(a) (7) was 14.96 ± 7.35 mg/dl

Among the subjects with TVD on CAG and high Lp(a) (3) the average Lp(a) was 106 ± 14.67 mg/dl, and that with normal Lp(a) (1) was 8.8 mg/dl.

Table 29: Descriptive analysis depicting the comparison of Lp(a) and LDL with CAG findings in the study population

		ANGIOGRAPHY					
		Normal		Abnormal		Total	
		n1	%	n2	%	n	%
LDL	**High**	1	16.7	28	28.6	29	27.9
	Normal	5	83.3	70	71.4	75	72.1
Lp(a)	**High**	3	50.0	67	68.4	70	67.3
	Normal	3	50.0	31	31.6	34	32.7

n1- Number of subjects with normal angiography
n2- number of subjects with abnormal angiography
n- total number of subjects

Among the subjects who had high LDL 17% (1) had normal CAG, 29% (28) had abnormal CAG.

Whereas, 83% (5) had normal LDL with normal CAG and 71% (70) had abnormal CAG.

Among the subjects with high Lp(a), 50% (3) had normal CAG, 68% (67) had abnormal CAG findings.

Whereas, 50% (3) had normal LDL with normal CAG and 32% (31) had abnormal CAG.

Table 30: Descriptive analysis depicting the comparison of Lp(a) and LDL and their findings in CAG in the study population

ANGIOGRAPHY		Lp(a)	LDL
Normal	N	6	6
	Mean	55.23	81.00
	Std. Deviation	46.61	17.65
	Median	52.20	79.50
Abnormal	N	98	98
	Mean	78.00	82.20
	Std. Deviation	67.81	38.12
	Median	61.45	78.50
Total	N	104	104
	Mean	76.68	82.13
	Std. Deviation	66.82	37.20
	Median	61.45	78.50

Among the subjects with normal CAG (6), the average Lp(a) was 52.2 mg/dl and LDL was 79.5 mg/dl.

And those with abnormal CAG (98) average Lp(a) was 61.45 mg/dl and LDL 78.5 mg/dl.

Figure 23: Bar graph showing the levels of Lp(a) and LDL in subjects with normal and abnormal angiography respectively

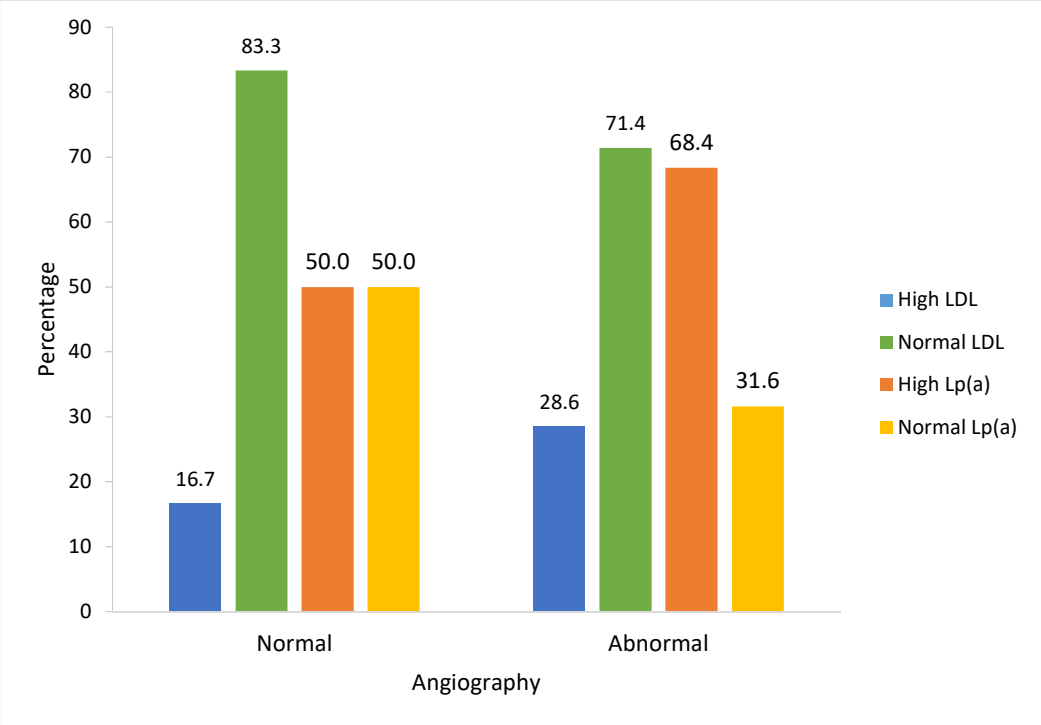


Table 31: Descriptive analysis of comparison of Lp(a) and LDL with angiographic findings

ANGIOGRAPHY				Lp(a)		Total
				High	Normal	
NORMAL	LDL	High	n1	1	0	1
			% within LDL group	100.0	0.0	
		Normal	n2	2	3	5
			% within LDL group	40.0	60.0	
SVD	LDL	High	n3	8	8	16
			% within LDL group	50.0	50.0	
		Normal	n4	30	15	45
			% within LDL group	66.7	33.3	
DVD	LDL	High	n5	7	4	11
			% within LDL group	63.6	36.4	
		Normal	n6	19	3	22
			% within LDL group	86.4	13.6	
TVD	LDL	High	n7	0	1	1
			% within LDL group	0.0	100.0	
		Normal	n8	3	0	3
			% within LDL group	100.0	0.0	
Total	LDL	High	n9	16	13	29
			% within LDL group	55.2	44.8	
		Normal	n10	54	21	75
			% within LDL group	72.0	28.0	

Subjects having normal angiography were 6, among then subjects with high Lp(a) and high LDL was 1, but with normal Lp(a) and high LDL were none.

Similarly, subjects with high Lp(a) and normal LDL were 2 and with both normal Lp(a) and LDL were 3.

Subjects with SVD on CAG were 61, among them subjects with high Lp(a) and high LDL were 8(50%), but with normal Lp(a) and high LDL were 8 (50%) as well. Similarly, subjects with high Lp(a) and normal LDL were 30 (67%) and with both normal Lp(a) and LDL were 15 (33%)

Subjects with DVD on CAG were 33, among them subjects with high Lp(a) and high LDL were 7(64%), but with normal Lp(a) and high LDL were 4 (36%). Similarly, subjects with high Lp(a) and normal LDL were 19 (86%) and with both normal Lp(a) and LDL were 3(14%)

Subjects with TVD on CAG were 4, among them subjects with high Lp(a) and high LDL were 0, but with normal Lp(a) and high LDL was 1 (100%). Similarly, subjects with high Lp(a) and normal LDL were 3 (100%) and with both normal Lp(a) and LDL were 0.

In total, among 104 subjects, 16 (55%) subjects had high Lp(a) and high LDL, 13 (45%) had normal Lp(a) and high LDL; 54(72%) had high Lp(a) but normal LDL and 21 (28%) had normal Lp(a) and normal LDL.

Figure 24: Bar graph depicting the CAG findings with comparison of LDL and Lp(a) in the study population

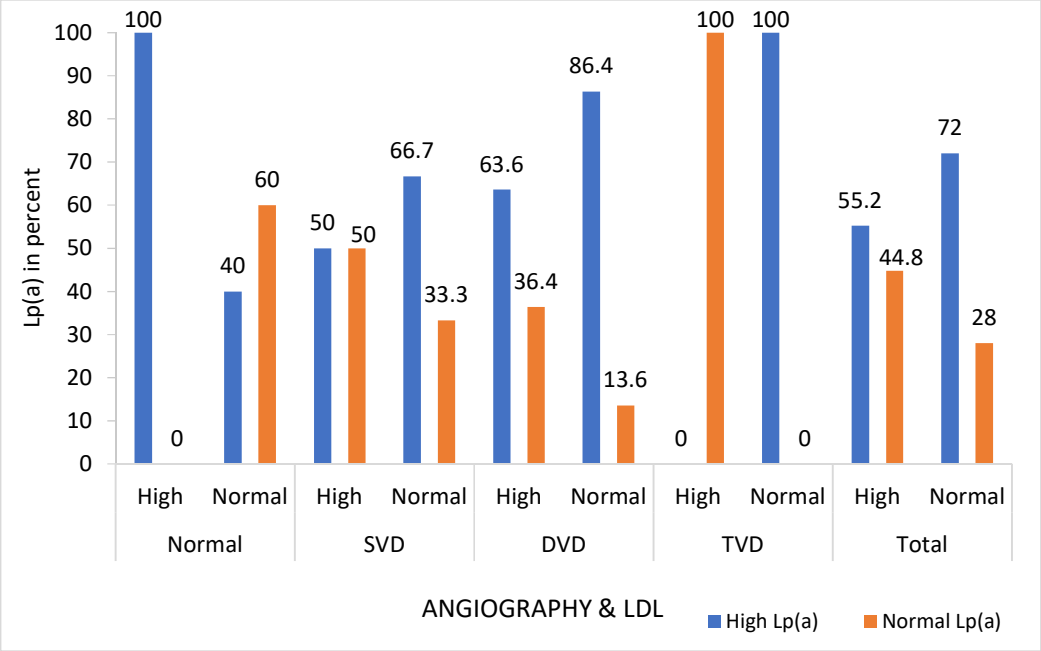


Figure 25: Bar graph depicting the culprit vessel in the CAG of study population

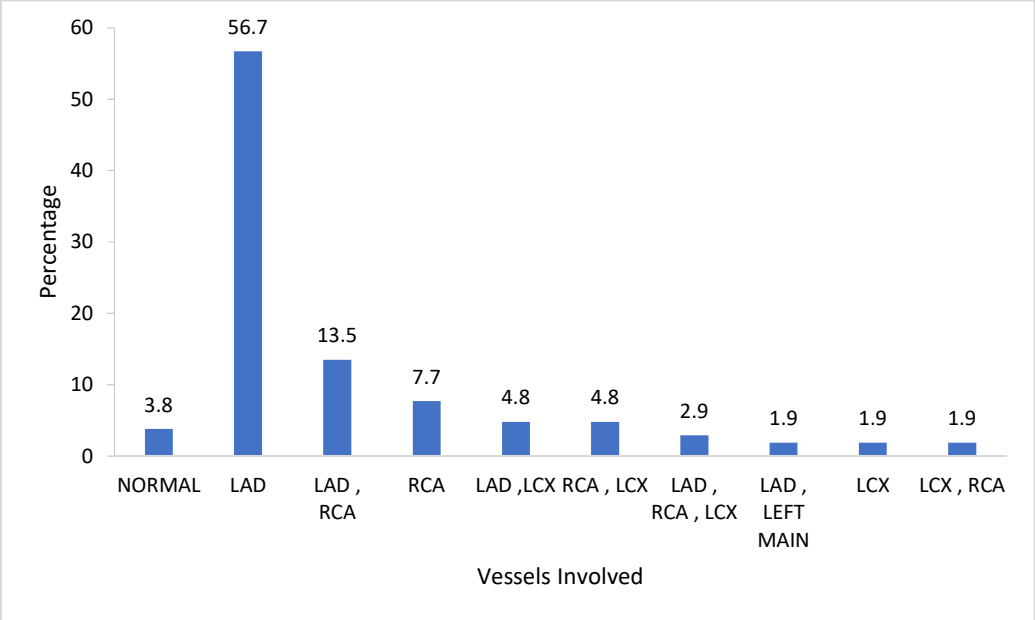
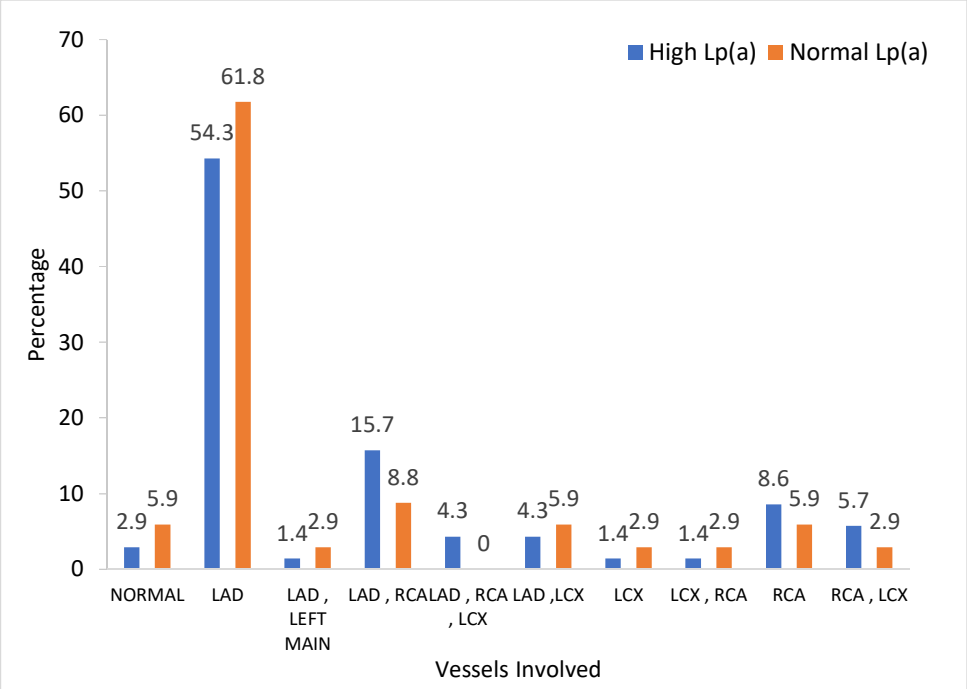


Table 32: Descriptive analysis of Lp(a) levels and culprit vessels on CAG

Vessels Involved	Lp(a)				Total	
	High		Normal			
	n1	%	n2	%	n	%
NORMAL	2	2.9	2	5.9	4	3.8
LAD	38	54.3	21	61.8	59	56.7
LAD, LEFT MAIN	1	1.4	1	2.9	2	1.9
LAD, RCA	11	15.7	3	8.8	14	13.5
LAD, RCA, LCX	3	4.3	0	0	3	2.9
LAD, LCX	3	4.3	2	5.9	5	4.8
LCX	1	1.4	1	2.9	2	1.9
LCX, RCA	1	1.4	1	2.9	2	1.9
RCA	6	8.6	2	5.9	8	7.7
RCA, LCX	4	5.7	1	2.9	5	4.8

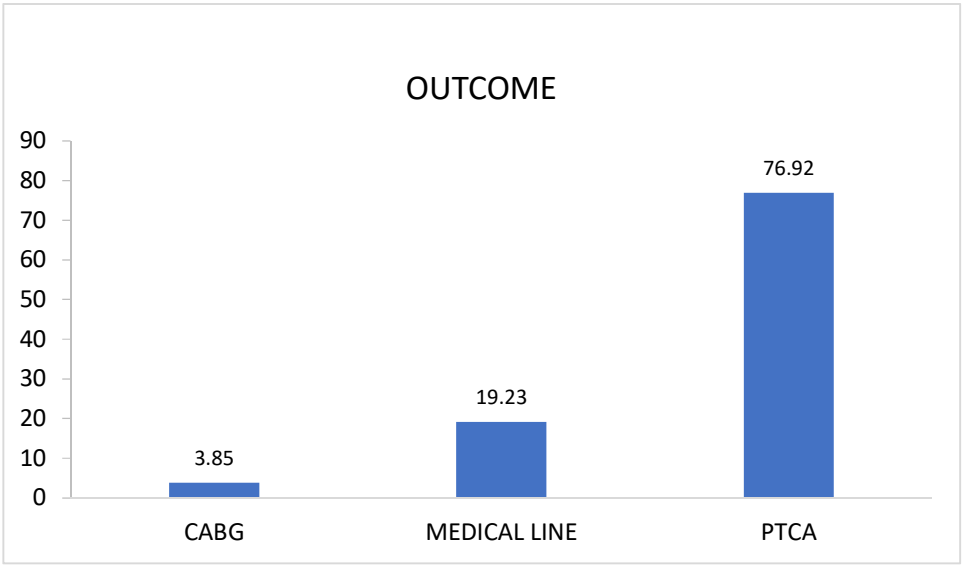
Most of the subjects 54% (38) with high Lp(a) had LAD occlusion followed by LAD and RCA combination. With normal Lp(a) 21 subjects 62% (21) had LAD occlusion.

Figure 26: Bar graph depicting the culprit vessel in subjects with high and normal Lp(a) in the study population



Most common vessel involved was the Left Anterior Descending.

Figure 27: Bar graph depicting the outcome in the study population



DISCUSSION

Coronary Artery Disease is a major epidemic affecting India and is currently one of the most common causes of mortality and morbidity in developed and developing countries as well. The annual number of deaths from CAD in India was projected to rise from 2.26 million (1990) to 4.77 million (2020). The higher rate of CAD in South Asians especially Indians, as compared to people of other ethnic origin, may indicate a possible genetic susceptibility and have prompted the need for better risk indicators and management strategies.

More attention needs to be paid to this younger population as the rate of CAD in them has been increasing in the recent past. The present study was undertaken in young patients of ischemic heart disease to assess clinical, biochemical and angiographic profile, conventional and newer risk factors, and correlation of risk factors with significant and nonsignificant CAD.

Primary goal of the study was to find the association of Lp(a) in young age group. Lp(a) level in an individual is 80-90% genetically determined in an autosomal codominant inheritance pattern with full expression by 1-2 years of age and adult-like levels achieved by approximately 5 years of age. Lp(a) levels remain stable through an individual's lifetime regardless of lifestyle.

A hospital based cross-sectional study was conducted in the tertiary care centre. Patients between the age of 18 to 49 were the focus of the investigation. Patients with ischemic ECG changes, RWMA (regional wall motion abnormalities) in 2D Echocardiography and/or had symptoms like chest discomfort, dyspnoea or diaphoresis were taken up for the study.

Demographic characteristics

In terms of risk factors for CAD, age, gender, and family history are all regarded to be non-modifiable. Men and women over the age of 45 and over the age of 55 are at an increased risk of CAD, according to a study by Hajar et al[33]. The subjects were classified based on their age into 18-30, 31-40 and 41-49. According to these data, the mean age of the current study subjects with CAD was 41.1 years, and approximately 62% were aged 41-49 years, 30 (29%) to 31-40 years, and 10 (10%) belonged to the 18-30 years group. More men (81.73 percent) than women contributed in the current study (18.27 percent). Males were shown to have a higher chance of developing coronary artery disease than females.

Among the young adults of age 35 to 54 years, the incidence of acute myocardial infarction (AMI) has increased. CAD occurring in young is particularly alarming, termed “premature” and defined as onset of CAD <45 years.[124]

The study LOLIPOPS showed the high prevalence of CAD in south Asians and especially Indians are more vulnerable to have CAD in young age group with a prevalence of 5% to 10%. As many as 4% to 10% of AMI events occur in this age group and carries a poor long-term prognosis in young adults. It is paramount to better understand conventional and unique risk factors to prevent ASCVD events in this population as it has a negative impact on the country's economic growth and productivity.

Conventional risk factors such as smoking, diabetes, hypertension, obesity and family history seem to be contributing in older subjects with CAD. These risk factors seems to vary in younger individuals. New biomarkers such as lipoprotein(a) seem to associated with CAD in young and have been assessed in the present study.

Lipoprotein(a)

Lp(a) consists of a low-density lipoprotein LDL–like particle containing a specific highly polymorphic glycoprotein, apolipoprotein(a) [apo(a)] that is covalently bound via a disulfide bond to the apoB100 of the particle. The apo(a) component of Lp(a) acts as proatherogenic and prothrombotic.

Recent studies have shown that Lp(a) inhibits the generation of transforming growth factor-β (TGF-β) leading to migration and proliferation of smooth muscle cells into the intima, thus further enhancing the formation of atheroma plaque. It's still unclear exactly how Lp(a) causes atherosclerosis, but recent evidence suggests that it suppresses plasmin production and promotes cholesterol accumulation in atherosclerosis.

Plasminogen Activator Inhibitor-1 (PAI-1) and tissue Plasminogen Activator (t-PA) inhibition, as well as the binding of Lp(a) to extracellular matrix components like proteoglycans or glycosaminoglycans, all contribute to the atherothrombogenic mechanisms of Lp(a). Lp(a) and LDL are also susceptible to exudation and phagocytosis, which results in the accumulation of Lp(a) in the vascular wall.

A study done by Byambaa et al,[125] found a striking differences in Lp(a) levels and its distribution in several groups. Blacks have a mean Lp(a) concentration twice as high of that in Whites. Many other studies based on the ethnic differences have also confirmed that Lp(a) levels in subjects of African descent have about twice as high levels as Caucasians, Asian populations, while intermediate levels were reported in South Asians. This interesting, but to date unresolved, variability might provide a possibility to evaluate a potential evolutionary advantage associated with Lp(a); however, the observed interethnic alteration could also be due to the apo(a) allele distribution in the subclass of the population.

Approximately 50% of first heart attacks occur before 55 years and 25% occur before 40 years of age. Analysis of INTERHEART data in South Asians revealed a prevalence of AMI of 11.7 in Indian aged below 40 years [102]. The prevalence of ischemic heart disease in 1960 in urban India was 2% and increased 7-fold to ≈14% by 2013 [100, 105, 106]. Similarly, it more than quadrupled in rural areas, from 1.7% to 7.4% between 1970 and 2013 [107].

A recent large-scale meta-analysis conducted by Erqou et al, [94] included 36 prospective studies with 126,634 individuals and confirmed the modest but strong risk for CVD caused by elevated Lp(a), especially with a level of Lp(a) ≥ 240 mg/L

Pineda et al,[112] found that young CAD patients had significantly higher Lp(a), TGLs, fibrinogen, fibrin D-dimer, and von Willebrand factor (vWf) but lower HDL-C.

The current study aims to assess the association of Lipoprotein(a) levels in proven CAD in young patients less than 49 years of age. The overall average Lp(a) in the study population was 77.98± 68.22 mg/dl. To assess the prevalence of Lp(a)in the age groups, it was found that in the age groups 18-30 years of age was 75.67 ± 18.86 mg/dl, 31-40 years of age was 106.87 ± 54.91 mg/dl and that of age group 41-49 was 113.1 ± 71.45mg/dl. It wound be fair to conclude that the levels of Lp(a) in the youngest group such as 18-30 years of age also had high Lp(a) indicating a possible genetic predisposition.

It is a well-established fact that the male sex is at a higher risk of developing CAD as compared to females. Middle-aged men have a 2–5 times higher risk than women. Kumar et al,[113]showed the prevalence of coronary heart disease among males in the villages was 1.7% and among females 1.5%. However, risk ratio differs between populations. There was a clear male preponderance (84.3%) in our study,

which was in agreement with previous studies, suggesting that CAD is predominantly a disease of men.[114, 115] Female represented only 15.7% of our study population. This was a much higher frequency compared with data from India (5%).

The best-known study, and a model for many others, The Framingham Study, was launched in the early 1950s. Several thousand men and women in Framingham, Massachusetts, were examined for certain personal factors, and subsequently shown through many years of follow-up, to be authoritative and consistent indicators of increased risk of coronary heart disease. The concept of risk factors was born. The most consistent and powerful of these in explaining CAD, the typical risk factors, were cigarette smoking, blood pressure and cholesterol. Others were less common (diabetes mellitus), less consistent (obesity and exercise) or less readily measured (diet, alcohol and psychosocial factors). In a multivariate analysis by Ghambir et al,[123]smoking, Lp(a), HDL and TGL were assessed to be the independent predictors of CAD risk. In the present study, Lipoprotein(a) was found to be the independent predictor.

After The Framingham Study, The Seven Countries Study sought to explain the large variation in death rates from coronary heart disease in different countries. It showed that obesity and physical exercise accounted for little, as did cigarette smoking, but the main role went to cholesterol.

A study by Collet et al,[124] describes a unique cohort of 880 adults who experienced symptomatic CAD at a young age (via <45 years), who were followed for up to 20 subsequent years. The average age was 40 years upon first presentation. Of these 87% were male and 13% were females and majority of the subjects were mainly smokers with a family history of CAD or hyperlipidemia. Despite initial prescription of aspirin (98%) and statins (93%), one third of these patients had a recurrent Major

Adverse Cardiovascular Events (MACE) and of these, 36% had at least a second recurrence of AMI. Smoking increases the risk of CAD by 3–5 times. Another study in India by Mishra V et al,[130] concluded that smoking was one of the major risk factor associated with CAD in their patients who were below 40 years of age. Yet another study by Aggarwal et al,[111] concluded that in young smokers, presence of hypertension, central obesity, diabetes mellitus and metabolic syndrome identifies a subset at increased risk for future acute CAD requiring more rigorous follow up and treatment. Pais et al,[33] has shown smoking to be the most dominant risk factor in Indian population studies.

In our study, smoking was a major modifiable risk factor that was present in 59% of patients with an average Lp(a) value of 61.59 ± 59.43 with a p value of 0.005, similar to observations in INTERHEART study. Few also had habits of both the form of tobacco intake, further increasing the risk of CAD. Few females could have also been a target for passive smoking, increasing their risk of developing CAD. In line with previous studies, our study demonstrates that cigarette smoking was the dominant risk factor predisposing to an earlier onset of CAD,

The association of obesity and CHD was fist noted by Kannel et al,[36]in Framingham 50 years ago. Obesity is a well-established risk factor for CAD [40]. When the younger and older patients with CAD were compared on the basis of obesity, there was no much difference in the prevalence of obesity in the groups.

A study conducted by Marcial et al[109]in young individuals between 21 to 35 years of age were studied and it was noted that obese patients had 5.94 times the possibility of having occlusion of the coronary vessel than normal weight patients. Obesity was the most important treatable predictor of premature obstructive CAD in our young adult population. A recent study by Poirier P et al,[122] reported that higher

body mass index (BMI) during childhood is associated with an increased risk of CHD in adulthood. The prevention and control of overweight and obesity in adults and children has become a key element for the prevention of cardiovascular diseases. Study by Qi Q et al,[110] found that there is an association between hereditary predisposition to obesity and higher consumption of sweetened beverages. This is most often seen in younger individuals in urban settings which can lead to a sedentary lifestyle and therefore increasing the risk of CAD in the patient.

Similarly, in our study we had 83 (79.8%) patients who were obese and this made the maximum bulk of the study population on the basis of BMI. 14 (13.5%) patients were overweight and only 7 (6.7%) were of ideal BMI. The average Lp(a) in subjects who were obese was 74.61 ± 63.68mg/dl and that in overweight was found to be 79.2 ± 55.72 mg/dl.

Diabetes is associated with a 2 to 4-fold increased mortality risk from heart disease. Furthermore, in patients with DM there is an increased mortality after MI, and worse overall prognosis with CAD. The American Heart association considers diabetes to be one of the seven major controllable risk factors for CVD.

A study by Rainwater et al,[129] reported significantly lower Lp(a) levels in Diabetic patients compared with matched nondiabetic controls in the San Antonio Heart Study. In a recent report from the ERIC-Norfolk study,[140] a strong inverse association between the Lp(a) level and new-onset DM was observed.

In our study, we noted that 20% (21) had only Diabetes and 40% (42) had both HTN and DM with average Lp(a) value of 89.08 ± 58.74 mg/dl and 120.44 ± 65.62 mg/dl respectively. However, subjects with no comorbidities had an average of 117.24 ± 72.8 mg/dl. Various studies such as that of Aggarwal et al,[139] have

demonstrated a recent increase in the prevalence of HTN [8.86% (2001-2002) to 27.7% (2009-2010)] in young CAD.

In our study we found that 21.2% were hypertensive which goes hand in hand with the previous notion that risk of CAD increase with HTN. The average Lp(a) in our study was 89.4 ± 42.63 mg/dl in those with Hypertension and 120.44 ± 65.62 mg/dl in subjects with both HTN and DM.

Dyslipidemia is an important risk factor for CAD. There seems to be a little difference in incidence of lipid abnormalities in younger and older patients. Another study by Hatmi et al,[137] compared the LDL and Total cholesterol in persons with CAD of age more than 55 and less 55 years, demonstrated a significantly increased level of LDL and total cholesterol in the latter group. On the contrary, in another study by Sinha et al,[138] there was a high prevalence of lipid abnormalities in young CAD when compared to older CAD group. These differences in lipid parameters could be attributed to the effect of dietary, genetic and environmental factors on lipid metabolism. Insulin resistance and hypertriglyceridemia may contribute together to the increased risk of CAD in hypertension. Triglyceride concentrations had a positive predictive value of 70% to identify individuals at risk for CAD in HTN.

In our study the average of Total cholesterol was found to be 156.45±46.52 mg/dl, that of HDL was 37.4 ± 11.25 mg/dl and that of TGL was 170.41 ± 90.93 mg/dl. A comparison of LDL and Lp(a) was studied, and we found that the average value of LDL was 82.13±37.2 mg/dl and that of Lp(a) was 77.97 ± 68.21 mg/dl. We also noted that majority of study population, 67.31% had higher level of Lp(a) and 32.69% had normal Lp(a) level, in contrast to this, 72.1% had normal LDL and only 28% had high LDL.

This approves with the studies that claim that Lp(a) is much more specific than LDL is of CAD. We found that all the lipid profile components were within normal limits and yet the subjects had CAD. This could to the fact Lp(a) is much more specific than LDL is of CAD.

In a study by Juonala et al,[116] to detect the risk factors in young adults, it was found that young subjects with a positive family history of CAD had greater sub-clinical atherosclerosis as compared to those with no such family history. A study by Michos et al,[117]observed that in the presence of family history and metabolic risk factors the subjects had higher prevalence of coronary artery calcium than those without any family history. This stresses the role of genes in the aetiology of CAD in young population. Studies by Otaki et al,[118] have shown that severe coronary atherosclerosis is seen in person with a positive family history of premature CAD and is a very strong predictor of future acute cardiac event. In a study by Goel et al,[84] found that the family history of premature CAD became the second most vital risk factor in young Indians with CAD. Thus, analysis of genetic factors that plays a role in atherogenesis, thrombogenesis, thrombolysis, lipid metabolism, and other metabolic factors is needed.

In our study it was noted that 55.56% had positive family history and this proves the association of positive family history with CAD in young. Most of them had CAD and DM as their main comorbidities, alone or in combination.

A study conducted by Ashfaq et al,[84] showed that the Lp(a) levels correlated positively with severity of atherosclerosis. The most relevant finding was that Lp(a) levels 21 mg/dL were not only associated with the presence of coronary disease but also with the severity of the coronary atherosclerosis. A trend towards an association

between higher Lp(a) levels and severe patterns of coronary atherosclerosis has been reported.

Similar to this, in our study, we tried to associate the levels of Lp(a) with the severity of the CAD. We noted that the subjects with STEMI had a comparatively higher level of Lp(a) when compared to NSTEMI and Unstable Angina. There was a significant difference in the value of Lp(a) in these groups.

Our study had 32 subjects with STEMI, 28 with NSTEMI and 10 with Unstable Angina with an average Lp(a) of 72.4 mg/dl, 57 mg/dl and 33 mg/dl respectively.

In another study consisting of 76 patients with ACS who underwent intravascular ultrasound (IVUS) at baseline and at 10-month follow-up after PCI, the results showed that patients with serum levels of Lp(a) >20 mg/dl had slight plaque progression despite of statin therapy, suggesting Lp(a) levels could be an alternative predictor of further plaque progression. Similarly, Hartmann et al,[119] by serial IVUS examination reported that Lp(a) was positively related to plaque progression in the left main coronary artery. Chetan et al,[135] also demonstrated that Lp(a) was positively and independently correlated with increased percent atheroma volume by using IVUS examination.

In a study by Shui,[120] it was demonstrated that elevated baseline serum Lp(a) was positively associated with angiographic progression of coronary artery disease. Another study by Schwartzman et al,[121] results indicate that elevated plasma Lp(a) is an independent risk factor for angiographic CAD in chronic stable angina and may have particular significance in women.

In the study done by Fauzia Ashfaq et al,[136] in 134 North Indian patients, Lp(a) concentration with normal coronaries was found to be 18.9 mg/dL, patients with single vessel involvement had Lp(a) of 39.2 mg/dL, double vessel was 58 mg/dL and triple vessel was 69.2 mg/dL. Younger populations have higher rate of SVD than the older population.

Similar to these, in our study, most of the patients that is, 61 had SVD with average Lp(a) of 52.5 mg/dl, DVD was seen in 33 patients with average Lp(a) of 70.8 mg/dl and 4 had TVD with average Lp(a) of 99.4 mg/dl and 6 had normal coronaries. It was noted that the most common artery to be involved was LAD in majority of the subjects. It is noted that Lp(a) levels were higher in patients with multiple vessel involvement as compared to the ones with single or double vessel involvement. This clearly shows that elevated Lp(a) adds to the severity of atherosclerotic changes and involves more than vessels.

Among the study population, 77% had to undergo PTCA, 19.23% underwent medical line of treatment and 4% had to undergo CABG.

CONCLUSION

In both developed and developing countries, coronary artery disease (CAD) is one of the leading causes of morbidity and mortality. The young suffering from CAD is increasing at an alarming rate in India and this needs appropriate intervention to minimise the CAD in our country.

1. A cross-sectional hospital-based study was done in 104 subjects aged 18-49 years with CAD in a tertiary care institute and the Lp(a) in them was assessed.
2. The overall average Lp(a) value was 77.98 mg/dl in the study population.
3. 82% (85) males and 18% (19) females contributed to the study, showing male preponderance.
4. The subjects were categorized into 3 groups depending upon the age: 18-30, 31-40, 41-49 years of age, with 10, 30 and 64 subjects in each category respectively. The average Lp(a) in these groups were 75.67 ± 18.86 mg/dl, 106.87 ± 54.91 mg/dl, 113.1 ± 71.45 mg/dl respectively.
5. The overall average value of Total cholesterol was normal in 81.7% with an average value of 156.45 ± 46.52 mg/dl which was within normal limits.
6. LDL was high in only 27.9% of the study population and the average value was 82.13 ± 37.2 mg/dl which was also within the normal limits.
7. HDL was low in 69.2% with an average value of 37.4 ± 11.25 mg/dl in the study population.
8. Triglycerides was high in 47.1% with an average value of 170.41 ± 90.93 mg/dl in the study population.
9. Lp(a) and LDL levels were compared and were found to have an average of 77.98 ± 68.22 mg/dl and 82.13 ± 37.2 mg/dl respectively.

10. Majority of the individuals had normal LDL values but high Lp(a) values.
11. Lp(a) was significantly high in subjects who had Lipid profile within the normal ranges.
12. Majority of the individuals belonged to the obese category with an average BMI of 29.22 ± 2.97kg/m^2 and the average Lp(a) in these subjects was 74.61 ± 63.68 mg/dl.
13. 14% subjects (14) were overweight with an average BMI of 24.08 ± 0.54 kg/m^2 with an average Lp(a) of 79.2 ± 55.72 mg/dl.
14. Among the study population, 56.73% (59) had habits such as smoking with an average Lp(a) of 61.59 ± 59.43 mg/dl.
15. Among the study population, 18% (19) had no comorbidities, 21.2% (22) had HTN and 20.2% (20) had DM only and 40.4% (42) had both DM and HTN.
16. Among the patients with DM, the average Lp(a) value was 107.53 ± 70.28 mg/dl.
17. In the study population with elevated Lp(a), 32 had STEMI, 28 had NSTEMI and 10 has Unstable Angina with an average Lp(a) of 72.4 mg/dl, 57 mg/dl and 33 mg/dl respectively. There was a significant difference in the value of Lp(a) in these 3 groups of CAD, making the Lp(a) value a very important marker for ACS. (p=0.000)
18. All 104 patients underwent CAG, out of which 6 had normal angiography with average Lp(a) of 52.2 mg/dl, 61, had single vessel disease (SVD) with average Lp(a) of 52.5 mg/dl. 33 had double vessel disease with average Lp(a) of 70.8 mg/dl, and 4 had triple vessel disease (TVD) with average Lp(a) of 99.4 mg/dl.

Strengths

The present study holds significant relevance, as very few Indian studies have evaluated the role of Lp(a) as a risk predictor of CAD in young. The present study adds to the literature evidence advocating the use of Lp(a) as a more specific biomarker of CAD.

Limitations

The lack of a control group was one of the primary drawbacks of the research. The isoform size of Lp(a), which directly correlates with CVD risk, has not been identified. Due to the COVID-19 pandemic, the study's sample size could not be extended enough to conduct a more in-depth investigation.

Recommendation

A screening of Lp(a) in younger population will help in early detection of CAD and drugs that specifically target Lp(a) should come up, so that therapeutic measures can be implemented to prevent CAD in young individuals.

SUMMARY

Coronary artery disease (CAD) is the principal cause of mortality and morbidity in developing and developed countries. There is an alarming increase in the prevalence of coronary artery disease in young population of India. There is an urgent need for newer risk predictors and management protocols because of the worrisome rise in CAD prevalence, according to recent literature studies. Premature CAD has a significant impact on a country's growth and economy because it is more prevalent in younger people.

A cross-sectional hospital-based study was conducted on 104 patients between the ages of 18 and 49 who had a coronary artery lesion. For each participant, medical history and clinical examination were conducted. Investigations such as a complete hemogram, fasting lipid profile (FLP), Lipoprotein(a), ECG, ECHO, X-RAY, and angiography were performed and the results of coronary angiography were noted, T-test, chi-square and one-way ANOVA were used to evaluate continuous data, and Excel 2007 was utilised for categorical data. By utilising a bar graph and pie charts, the distribution of the variables were depicted. Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. P value <0.05 was considered statistically significant. The data were analysed by using SPSS software. Various CAD factors were also examined for correlations with Lp(a).

The study had majority of males and the overall average age was 41.1 years. Most of the CAD subjects exhibited high Lp(a), therefore underscoring the importance of the lipoprotein measurement in the risk assessment process for CAD

patients. The overall average Lp(a) value was found to be 77.98 ± 68.22 mg/dl. The average total cholesterol levels were low (156.45 ± 46.52 mg/dl) in the study population. Average HDL was also found to be low (37.4 ± 11.25 mg/dL) among participants, which is consistent with previous studies that have linked a lower HDL level to an increased risk of developing CAD". Average LDL was found to be of normal range (82.13 mg/dl) and average TGL was high (170.41 ± 90.93 mg/dl) in the study population. On comparing Lp(a) and LDL levels, it was noted that majority of the population had normal LDL levels and high Lp(a) levels.

Among the study population, 14 of the subjects were overweight and had an average value of Lp(a) of 79.20 ± 55.72 mg/dl and 83 of them were obese and had an average Lp(a) of 74.61 ± 63.8 mg/dl. It was also observed that the most of the subjects had a history of CAD in their parents.

A significant finding was seen in the Lp(a) levels of patients with STEMI, NSTEMI and Unstable Angina with average Lp(a) of 72.4 mg/dl, 57 mg/dl and 33 mg/dl respectively. There also was a strong link between Lp(a) and angiographically seen SVD, DVD and TVD with average Lp(a) values of 52.5 mg/dl, 70.8 mg/dl and 99.4 mg/dl respectively. Most of the subjects had SVD and the most common vessel involved was LAD.

The present study was one of a kind as there are no such studies in the study region to estimate the possible cause of CAD in young individuals. Accurate and cost-effective screening tools, like Lp(a), may be used to identify patients at risk of developing CAD.

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ANNEXURE I – ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 286 **Date: 24/12/2019**

To,

Dr. Aishwarya Anil Patted
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"ASSOCIATION OF LIPOPROTEIN (A) IN CORONARY ARTERY DISEASE IN YOUNG INDIVIDUALS",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II

CONSENT FORM - ENGLISH

Title Of Research Study: "ASSOCIATION ON LIPOPROTEIN(a) IN CORONARY ARTERY DISEASE IN YOUNG INDIVIDUALS"

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me and has been explained to me in my vernacular language and all my questions have been answered. I will be given a copy of this consent form.

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :……...

Signature / Left thumb impression :……...

of the participant

Name of the legally authorized :……...

representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

Investigator's name and signature :……...

Date:

Place:

ANNEXURE III – PROFORMA

Title of Research Study: Association of Lipoprotein(a) in Coronary Artery Disease in young individuals.

Case number –		**Age/ Sex-**	
Name-			
Occupation-		**Religion-**	
Address-			
IP Number-		**Socioeconomic status-**	
Complaints at presentation-			
Admission Diagnosis-			
Risk Factors- **1) Diabetes-**			
2) Hypertension-			
3) Smoking -			
4) Dyslipidemia-			
5) Obesity-			
6) Drug history-			
7) Familial history –			

Physical Examination: Pulse:	Height :	BMI:
BP:	Weight:	
CVS: **a)Inspection**		
b)Palpation		
c)Percussion		
d)Auscultation		
RS:		
PA:		
CNS:		
Complete hemogram-		
Hb		
TLC		
PCV		
Platelet		
Reticulocyte count		
PS		
HbA1c		
Trop I		
Lipid profile-		
Total cholesterol		
Low- density lipoprotein (LDL):		
High- density lipoprotein (HDL):		
Triglycerides:		
Lp(a):		

ECG findings:	
Coronary angiography report-	
2D Echo report-	

ANNEXURE IV – MASTER CHART

1. Lp(a)- Lipoprotein(a)
2. DOE- Dyspnea on exertion
3. LDL- Low Density Lipoprotein
4. HDL- High Density Lipoprotein
5. TGL- Triglycerides
6. TC- Total Count
7. Hb- Hemoglobin
8. RBC- Red Blood Cell
9. OHA- Oral Hypoglycemic Drugs
10. HTN- Hypertension
11. DM- Diabetes Mellitus
12. BMI- Body Mass Index
13. BP- Blood Pressure
14. ECG- Electrocardiogram
15. 2D Echo- 2D Echocardiography
16. NSR- Normal Sinus Rhythm
17. AWMI- Anterior Wall Myocardial Infarction
18. IWMI- Inferior Wall Myocardial Infarction
19. LVH-Left Ventricular Hypertrophy
20. EF- Ejection Fraction
21. SVD- Single Vessel Disease
22. DVD- Double Vessel Disease
23. TVD-Triple Vessel Disease
24. PTCA- Percutaneous Transluminal Coronary Angioplasty
25. CABG- Coronary Artery Bypass Grafting.

Age	Sex	Religion	Occupation	Chief Complaints	Admission diagnosis	Risk Factors	DIABETES	HYPERTENSION	SMOKING	TOBACCO	ALCOHOL	DRUG HISTORY	FAMILY HISTORY	Height (cm)	Weight(kg)	BMI (kg/m2)	Pulse (bpm)	BP (mmHg)	INVESTIGATIONS	Hb	RBC	TC	Platelets	Total Cholestrol	LDL	HDL	TGL	Lp(a)	ECG	2D ECHO	ANGIOGRAPHY	Treatment
29	Male	Hindu	Business	Chest pain x 1 day	IHD - AWMI , DM		YES	YES	YES	YES	YES	OHA FOR DM	FATHER- IHD, DM + HTN	160	70	27.34	88	110/70		19.5	6.59	28.7	309	249	171	47	153	79.1	Acute AWMI	IHD WITH AWMI WITH RBBB WITH EF 40% , CHB WITH S/P TPI	SVD	PTCA
32	Male	Hindu	Police	Chest pain , Sweating , DOE x 2 days	IHD with AWMI		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	168	85	30.12	82	120/80		9.9	4.83	11.5	466	99	44	36	93	148	Evolved AWMI	IHD WITH AWMI WITH EF 40	SVD	PTCA
45	Male	Hindu	Priest	Chest discomfort, Profuse sweating - 3 days	IHD with IWMI		NO	NO	NO	YES	NO	NONE	FATHER - IHD	157	62	25.15	78	130/90		14.3	4.31	7.9	271	190	123	37	100	8.8	IWMI	IHD WITH IWMI WITH EF 50%	SVD	PTCA
29	Male	Muslim	Farmer	Chest pain x 3 days	ACS WITH NSTEMI		NO	NO	NO	YES	YES	NONE	NOTHING SIGNIFICANT	170	76	26.30	82	130/80		11.1	5.23	10	294	115	61	30	119	7	NSR	NRWA FF-60%	NORMAL	PTCA
49	Male	Muslim	Govt. officer	Chest pain x 15 days	IHD with IWMI 2) DM 3) OLD IHD		YES	NO	NO	YES	NO	IHD , HTN TABLETS	FATHER - IHD	170	78	26.99	66	120/90		12	3.82	10.3	305	294	202	34	288	8.8	Evolved AWMI	S/P PTCA, IWMI WITH EF- 50%	TVD	CABG
47	Male	Hindu	Teacher	Chest pain x 10 days	IHD with AWMI		NO	YES	NO	NO	NO	T. TELMIKIND 40 OD	NOTHING SIGNIFICANT	168	73	25.86	88	120/90		12.5	4.3	9.3	242	150	97	33	101	83	Acute AWMI	IHD WITH AWMI WITH EF 45%	SVD	PTCA
43	Male	Muslim	Army Officer	Chest pain x 1 day	IHD with AWMI 2) DM		YES	NO	YES	NO	NO	OHA	NOTHING SIGNIFICANT	168	88	31.18	72	150/90		16.2	4.43	12.3	228	119	54	37	142	10.6	Acute AWMI	IHD WITH AWMI WITH EF 45%	SVD	PTCA
46	Male	Hindu	Army Officer	Chest pain x 2 days	IHD with TMT +ve , s/p PTCA		YES	YES	NO	NO	NO	T. TELMIKIND 40 OD	MOTHER-IHD	160	78	30.47	78	130/90		16.6	5.6	8.9	256	150	96	19	188	147	Acute AWMI	S/P PTCA, IWMI WITH EF- 60%	SVD	PTCA
35	Male	Hindu	Govt. service	Chest pain , profuse sweating x 1 day	IHD with AWMI , SVD		NO	NO	YES	NO	YES	NONE	MOTHER- IHD, DM, HTN	168	70	24.80	76	110/90		16.7	5.2	16.8	368	209	142	39	141	13.7	Acute AWMI	HYPOKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APEX EF 50%	SVD	PTCA
46	Male	Muslim	Business	Chest pain x 3 days	IHD- NSTEMI, SVD, DM, HTN		YES	YES	NO	NO	YES	OHA, ANTI-HTN	MOTHER, FATHER- IHD	160	75	29.30	80	120/90		13.1	4.87	14.5	301	199	53	37	100	33.1	Old IWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX, EF- 45%	SVD	MEDICAL LINE
35	Male	Hindu	Business	Chest pain x 1 day	ACS with AWMI		YES	NO	NO	NO	YES	OHA	NOTHING SIGNIFICANT	165	75	27.55	78	110/90		16.2	5.2	15.6	451	205	127	36	212	110.4	Acute AWMI	IHD- AKINESIA OF ANT WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF-40%	SVD	PTCA
40	Male	Hindu	Agriculturist	Chest pain x 1 day	IHD WITH AWMI, DVD		YES	YES	NO	YES	YES	OHA, ANTI HTN	NOTHING SIGNIFICANT	159	60	23.73	80	110/80		10.9	4.4	6.9	263	200	120	30	199	31.2	Acute AWMI	AKINESIA OF ANT WALL, SEPTUM, APEX EF45%	DVD	PTCA
46	Female	Hindu	Business	DOE, Chest pain x 1 day	ACS- AWMI		NO	NO	NO	NO	NO	NONE	FATHER - IHD	154	75	31.62	88	120/80		11.7	4.6	7.9	235	132	58	51	116	22.4	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APEX WITH HYPOKINESIA OF INFERIOR WALL, SEPTUM EF-35	SVD	MEDICAL LINE
33	Male	Hindu	Business	Chest pain x 1 day	IHD- IWMI, DM		YES	NO	NO	YES	YES	OHA	MOTHER- DM, HTN ,FATHER- HTN	169	80	28.01	78	130/90		14.1	4.97	15.6	301	195	123	31	206	18.6	Evolved AWMI	AKINESIA OF INF WALL, INFEROSEPTUM WALL, HYPOKINESIA OF INFEROLATERAL SEPTUM, EF-45%	DVD	PTCA
48	Male	Hindu	Agriculturist	Chest pain x 1 day	IHD - ASMI, DVD, HTN, DM		YES	YES	NO	NO	NO	OHA, ANTI HTN	FATHER - IHD	170	80	27.68	76	120/90		14.6	5.1	12.3	396	110	60	41	47	212.6	ASMI	AKINESIA OF APICAL SEPTUM, APEX, HYPOKINESIA OF ANT WALL, ANTEROSEPTUM, EF 45%	DVD	PTCA
48	Male	Hindu	Farmer	Chest pain x 2 days	IHD- AWMI, HTN , DVD		NO	YES	YES	YES	YES	ANTI HTN	NOTHING SIGNIFICANT	170	80	27.68	80	140/90		12.1	4.56	7.9	359	133	67	39	135	37.6	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APEX WITH HYPOKINESIA OF INFERIOR WALL, SEPTUM EF-35	DVD	PTCA
45	Male	Hindu	Farmer	DOE , profuse sweating x 1 day	IHD- AWMI, DM, DVD		YES	NO	NO	YES	YES	OHA	MOTHER- IHD, HTN	175	75	24.49	82	130/80		9	3.26	10	217	88	42	23	180	70.8	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APEX WITH HYPOKINESIA OF INFERIOR WALL, SEPTUM EF-45	DVD	PTCA
45	Female	Hindu	Cleaner	DOE , profuse sweating x 1 day	IHD with AWMI with LBBB 2) DM 3) HTN 4) s/p PTCA to LAD		YES	YES	NO	NO	NO	ANTI-HTN DETAILS NOT KNOWN	NOTHING SIGNIFICANT	157	60	24.34	79	130/90		11.3	4.39	10.2	204	148	75	40	163	138.1	Evolved AWMI	AKINESIA OF ANTERIOR WALL, SEPTUM, APEX EF 45%	SVD	PTCA
27	Male	Hindu	Farmer	Chest discomfort x 1 day	IHD with AWMI s/p lyses on 22/6/20		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	160	68.9	26.91	96	110/80		17.3	5.3	12	357	298	200	33	199	53	Acute AWMI	IHD AKINESIA OF ANTEROSEPTUM, APICAL AEPTUM, APEX EF45%	SVD	PTCA
44	Male	Hindu	Farmer	Chest painx 1 day	IHD with AWMI with LBBB , DVD		YES	YES	YES	NO	NO	T. TENEDIA 20 OD , T. AMLODAC 5 OD , T. TAZLOC CT 40 OD, T. GLYCOMET GP1 OD	NOTHING SIGNIFICANT	153	63	26.91	81	100/70		18.4	6.21	12.1	318	90	42	35	90	195.2	NSR	IHD WITH INF, INFEROLATERAL, ANTERIOR, ANTEROLATERAL WALL WITH EF 40%	DVD	PTCA
42	Male	Hindu	Business	Chest pain x 1 day	IHD Acute AWMI		NO	NO	NO	YES	YES	NONE	FATHER - IHD	158	70	28.04	90	120/80		15.7	5.2	10.2	302	180	54	32	69	28.8	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX, EF- 45%	SVD	PTCA
48	Male	Hindu	Business	Chest pain x 2 days	IHD- IWMI, HTN, SVD		NO	YES	NO	YES	YES	ANTI HTN	MOTHER- DM , HTN	160	70	27.34	69	120/80		13.6	3.9	11.1	238	137	92	39	91	27.1	ACUTE IWMI	AKINESIA OF INFEROLATERAL WALL, EF- 45%	SVD	PTCA
42	Male	Hindu	Business	Chest pain , DOE x 2 days	IHD- OLD IWMI, DVD, HTN		NO	YES	NO	NO	NO	ANTI HTN	NOTHING SIGNIFICANT	169	67	23.46	80	130/80		15.1	4.25	15.4	241	109	49	37	114	63.9	Old IWMI	HYPOKINESIA OF INFERIOR WALL, INFEROSEPTUM, INFEROLATERAL WALL EF- 50%	DVD	PTCA
39	Female	Hindu	Housewife	DOE , profuse sweating x 1 day	IHD with TMT +ve for inducible ischemia		NO	YES	NO	NO	NO	NONE	NOTHING SIGNIFICANT	150	57	25.33	96	140/100		12	5.53	7.4	274	148	65	37	231	110	NSR	NORMAL, EF- 60%	NORMAL	MEDICAL LINE
39	Male	Hindu	Doctor	Profuse sweating x 2 days	ACS WITH NSTEMI , SVD		YES	NO	NO	YES	NO	OHA	NOTHING SIGNIFICANT	160	80	31.25	70	150/80	inv	11.4	4.55	10	306	154	76	31	234	13	IWMI	IHD- HYPOKINESIA OF ANF WALL, INFEROSEPTUM, EF50%	SVD	PTCA
45	Female	Muslim	Housewife	Profuse sweating x 1 day	IHD WITH AWMI		YES	YES	NO	NO	NO	T. TELMA 20. OHA	FATHER - IHD	155	86	35.80	80	120/80		9.9	4	11.9	660	151	96	42	63	28.5	Acute AWMI	IHD- AKINESIA OF ANT WALL, ANTEROSEPTAL, APICAL, APEX EF45%	SVD	PTCA
32	male	hindu	business	chest pain x 4 days	IHD- AWMI , SVD		NO	NO	NO	NO	YES	NONE	NOTHING SIGNIFICANT	170	80	27.68	80	130/80		10.3	3.7	17.5	220	176	70	39	243	85.6	Acute AWMI	IHD- ANT WALL MI, ANTEROSEPTAL WALL, APICAL, APEX EF- 50%	SVD	MEDICAL LINE
49	Male	Hindu	Teacher	Chest pain x 1 day	IHD- Evolved AWMI , DVD, HTN, DM		YES	YES	NO	NO	NO	DETAILS NOT KNOWN	FATHER- IHD, DM	160	70	27.34	86	120/60		13.6	4.62	8.3	183	80	31	28	103	165.7	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF-45%	DVD	PTCA
49	Male	Muslim	Business	Chest pain x 3 days, DOE x 3 days	IHD WITH AWMI, HTN		NO	YES	YES	YES	YES	T. ECOSPRIN 75 0-0-1	FATHER - IHD	175	102	88.00	109	140/90		14.4	5.17	9.3	222	140	80	38	109	90.2	Acute AWMI	NORMAL , EF 60%	TVD	PTCA
39	Male	Hindu	Business	Chest pain , profuse sweating X 3 DAYS	ACS-OLD IWMI WITH ACUTE AWMI lysed with Reteplase, CKD, post PTCA		YES	NO	NO	YES	YES	OHA	NOTHING SIGNIFICANT	176	80	25.83	100	90/60		15.4	5.39	10.5	388	154	65	45	220	72.1	Old IWMI	IHD- AKINESIA OF ANT WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX, INF WALL, INFEROSEPTUM, EF30%	DVD	CABG
42	male	hindu	agriculture	DOE, chest pain 3 days	IHD- NSTEMI		NO	NO	NO	NO	NO	NONE	FATHER- IHD, MOTHER IHD HTN	169	78	27.31	72	130/80		13.2	4.6	12.2	347	156	68	92	209	149.6	Acute AWMI	HYPOKINESIA OF APICAL SEPTUM, APEX EF 50	SVD	MEDICAL LINE
47	male	hindu	agriculturist	DOE, chest tightness 3 days	ACS- IWMI, RVMI, PWMI lysed with streptokinase		NO	NO	NO	YES	YES	NONE	FATHER - IHD, DM, HTN	172	76	25.69	80	110/60		12.3	3.8	12.8	542	94	33	48	64	98.2	NSR	AKINESIA OF INFERIOR WALL, INFEROLATERAL , ANTEROLATERAL EF 45%	SVD	MEDICAL LINE
49	Male	Hindu	Farmer	DOE x 7 days	IHD- AWMI, DM,HTN, DVD		YES	YES	NO	YES	NO	OHA	NOTHING SIGNIFICANT	165	76	27.92	98	120/90		10.4	4.21	20.85	231	134	80	13	205	44.7	Acute AWMI	HYPOKINESIA OF APICAL SEPTUM, APEX EF 50	DVD	PTCA
39	Male	Hindu	Farmer	Chest painx 3 days	IHA- AWMI, S/P ASD closure , DVD		NO	NO	NO	NO	NO	T. ASPIRIN 150	MOTHER - DM	175	78	25.47	98	130/90		16.1	5.52	13.9	351	183	108	25	250	75.4	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF-45%	DVD	PTCA
42	Male	Hindu	Pvt company Service	chest pain x 3 years	IHD- EVOLVED AWMI with LV apical clot		YES	NO	NO	YES	NO	OHA	MOTHER - DM	180	80	24.69	82	120/90		14.7	4.92	7.9	244	132	80	26	126	52.5	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTERO SEPTUM, APEX EF-40%	SVD	PTCA
47	Female	Hindu	housewife	Profuse sweating , Chest pain x 1 day	IHD - EVOLVED ASWI , SVD		YES	NO	NO	NO	NO	DETAILS NOT KNOWN	MOTHER-IHD	160	58	22.66	76	130/90		12	3.83	8.4	292	115	58	32	124	24.8	Evolved AWMI	AKINESIA OF APICAL SEPTUM, APEX EF 50%	SVD	PTCA
46	Male	Hindu	Farmer	Chest pain x 1 day, sweating	IHD- AWMI, DVD		NO	NO	NO	NO	NO	NO	MOTHER - HTN	167	65	23.31	84	130/90		12.6	4.32	12.2	309	180	39	45	180	212.6	Evolved AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF40%	DVD	PTCA
42	Male	Hindu	Business	profuse sweating x 4 days, DOE	IHD- IWMI, HTN, SVD		NO	YES	YES	NO	NO	ANTI HTN	NOTHING SIGNIFICANT	165	81	29.75	80	120/90		11.5	4.98	10.1	398	146	79	51	82	35.4	IWMI	HYPOKINESIA OF INFERIOR WALL, INFEROSEPTUM, INFEROLATERAL WALL EF- 50%	SVD	PTCA
43	Male	Hindu	Business	Chest pain x 4 days	ACS - IWMI lysed with streptokinase		YES	NO	NO	NO	NO	OHA	FATHER - IHD	152	89	38.52	85	130/90		16.4	5.29	9.5	219	115	47	30	192	7	IWMI	HYPOKINESIA OF INFEROLATERAL WALL, EF- 50%	DVD	PTCA
49	Male	Hindu	Business	Profuse sweating x 4 days , DOE	IHD WITH OLD AWMI, S/P PTCA, HTN , DM		YES	YES	NO	NO	NO	NOT KNOWN	NOTHING SIGNIFICANT	160	79	30.86	88	130/80		11.1	4.42	14.6	246	222	112	28	411	170.5	Old IWMI	S/P PTCA, HYPOKINESIA OF BASAL INF WALL, INFEROSEPTUM, EF-50	SVD	PTCA
44	Female	Hindu	Housewife	Profuse sweating x 3 days	IHD- UNSTABLE ANGINA		NO	YES	NO	NO	NO	ANTI HTN	MOTHER- DM , HTN	150	80	35.56	86	120/90		10.5	4.28	9.5	281	250	120	30	120	99	NSR	NO REGIONAL WALL ABNORMALITY, EF 60%	SVD	MEDICAL LINE
28	male	hindu	student	chest pain x 4 days	IHD- Evolved AWMI		NO	NO	NO	NO	NO	NONE	FATHER- HTN	168	75	26.57	78	130/80		11.4	5.4	7.8	273	156	67	29	301	57	Evolved AWMI	NO REGIONAL WALL ABNORMALITY, EF 60%	SVD	MEDICAL LINE
44	Male	Hindu	business	DOE , profuse sweating x 1 day	IHD- NSTEMI		YES	YES	YES	YES	NO	HTN , DM TABLETS	NOTHING SIGNIFICANT	160	75	29.30	96	160/100		11.3	3.39	15	341	200	147	36	184	17.3	T wave inversion in avL,V2-5	AKINESIA OF INF WALL, INFEROLATERAL, ANTEROLATERAL , LATERAL EF45%	DVD	PTCA
44	Female	hindu	shopkeeper	chest pain- 4 days	IHD- SVD, DM		YES	NO	NO	NO	NO	OHA	MOTHER- IHD, DM	158	65	26.04	110	130/80		12.7	5.25	10.8	214	152	47	48	72	235.4	NSR	NO REGIONAL WALL ABNORMALITY, EF 60%	SVD	PTCA
43	Male	Hindu	Business	DOE x 4 days	IHD- IWMI, DVD		NO	NO	NO	NO	YES	NONE	MOTHER, FATHER- IHD	167	89	31.91	79	120/80		12	4.8	11	298	157	92	33	158	67.9	IWMI	IHD- AKINESIA OF INFERIOR WALL, INFEROSEPTUM. INFEROLATERAL WALL, EF 45%	DVD	PTCA
38	Male	Hindu	Business	Profuse sweating x 5 days	IHD WITH EVOLVED AWMI		NO	NO	NO	YES	NO	NONE	NOTHING SIGNIFICANT	160	75	29.30	80	130/80		13.1	4.22	6.5	284	137	83	33	104	280.7	Evolved AWMI	AKINESIA OF ANTERIOR WALL, APICAL SEPTUM, APEX EF-45%	SVD	PTCA
42	Male	Hindu	Business	Chest pain x 6 days	IHD- NON Q AWMI, DVD		YES	NO	YES	YES	YES	OHA	FATHER- HTN , DM	165	85	31.22	98	120/90		10.9	5.22	6.7	237	152	95	34	117	8.1	T WAVE INVERSION IN V2-6	HYPOKINESIA OF MID ANTEROSEPTUM, APICAL SEPTUM, APEX EF-50%	DVD	PTCA
40	Male	Hindu	Farmer	DOE , profuse sweating x 5 days	IHD - IWMI, LYSED WITH STREPTOKINASE, RECANALISED RCA		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	162	80	30.48	98	100/70		11.4	3.26	10.3	192	210	119	48	178	12.3	IWMI	IHD - AKINESIA OF INF WALL, INFEROSEOTUM, INF LATERAL WALL, EF- 45%	SVD	MEDICAL LINE
48	Female	Hindu	Farmer	Chest pain x 8 days	IHD -AWMI WITH MODERATE LV DYSFUNCTION		YES	YES	NO	NO	NO	OHA	NOTHING SIGNIFICANT	152	56	24.24	100	110/80		14	5.76	11.1	28	115	47	30	190	116.5	Evolved AWMI	AKINESIA OF ANTERIOR WALL, APICAL SEPTUM, APEX EF-35 %	DVD	PTCA
45	Male	Hindu	Farmer	Chest pain x 10 days on off	IHD- UNSTABLE ANGINA		NO	YES	YES	YES	NO	ANTI HTN	NOTHING SIGNIFICANT	165	86	31.59	81	110/70		14.2	4.86	5.8	271	138	76	35	134	22.9	NSR	NORMAL EF- 55%	NORMAL	MEDICAL LINE
38	Male	Hindu	Business	DOE, profuse sweating x 4 days	IHD- Unstable angina		NO	YES	YES	YES	YES	ANTI HTN	FATHER - IHD	160	60	23.44	86	130/80		12.9	4.8	11.2	186	143	76	50	80	12.2	ST depression in ant leads	NORMAL EF- 50%	DVD	PTCA
30	Female	muslim	shopkeeper	chest pain x 2 days	IHD- AWMI		NO	NO	NO	NO	NO	NONE	MOTHER-IHD	154	80	33.73	80	160/90		12.7	4.42	9.6	350	171	84	41	229	71.6	QS COMPLEX IN V2-3	AKINESIA OF MID ANTEROSEPTUM, APICAL SEPTUM, APEX EF- 45%	DVD	CABG
40	Male	Hindu	Business	Chest pain x 6 days	ACS- evolved Lateral wall MI , HTN		NO	YES	YES	YES	YES	ANTI HTN	NOTHING SIGNIFICANT	159	60	23.73	120	170/100		14.5	6.99	10	298	129	67	28	171	120	Lateral wall MI	AKINESIA OF INF WALL, INF LATERAL, APEX, APICOLATERAL,LATERAL WALL, EF45	DVD	PTCA
46	Male	Muslim	Business	Chest pain x 8 days	IHD-IWMI, DVD		NO	NO	NO	YES	NO	IHD TABLETS	FATHER - IHD	155	49	20.40	89	130/90		13.9	4.36	10.8	316	146	78	51	89	196.7	IWMI	AKINESIA OF INFERIOR WALL, INFEROSEPTUM, INFEROLATERAL WALL, EF- 45%	DVD	PTCA
47	Female	Hindu	Teacher	Profuse sweating, DOE x 3 days	IHD-AWMI		NO	YES	NO	NO	NO	ANTI HTN	NOTHING SIGNIFICANT	152	57	24.67	90	180/100		13.8	4.8	8.8	297	163	93	51	95	81.5	Acute AWMI	NORMAL EF-60%	NORMAL	MEDICAL LINE
41	Male	Hindu	Farmer	Chest pain x 3 days	IHD - Evolved IWMI with unstable angina		NO	YES	YES	YES	YES	ANTI HTN	FATHER - IHD, DM, HTN	160	80	31.25	72	120/80		14.5	5.3	10.6	274	209	178	62	100	111.4	Evolve IWMI	NORMAL EF-60%	DVD	PTCA
36	Male	Hindu	Agriculturist	DOE , profuse sweating x 7 days	IHD- NSTEMI		NO	NO	YES	NO	NO	NONE	NOTHING SIGNIFICANT	160	70	27.34	78	120/80		13.7	4.26	15.4	315	130	53	34	216	7	NSR	NORMAL EF-60%	SVD	PTCA
43	Female	Hindu	housewife/farmer	DOE, Chest pain x 4 days	IHD USA, HTN, DM		YES	YES	NO	NO	NO	OHA	NOTHING SIGNIFICANT	150	65	28.89	80	120/90		13	4.65	12.1	547	110	48	34	92	33.4	Sinus Tachycardia	NORMAL, EF-50%	SVD	PTCA
40	Female	Muslim	Housewife	DOE x 4 days	IHD WITH OLD IWMI		YES	NO	NO	NO	YES	OHA	NOTHING SIGNIFICANT	165	70	25.71	80	130/80		11.2	4.66	9.95	240	112	36	33	214	52.1	Old IWMI	AKINESIA OF INF WALL, INFEROSEPTUM, INFEROLATERAL, EF 40%	DVD	PTCA

46	Female	Hindu	Daily wager	Chest pain, DOE x 2 days	IHD- NSTEMI		NO	NO	NO	NO	YES	NONE	MOTHER-IHD	152	84	36.36	68	130/90		12.1	5.18	10.5	301	100	24	49	180	36.2	Old IWMI	AKINESIA OF ANT WALL, APEX, EF40%	SVD	PTCA
47	male	hindu	agriculturist	Chest pain, DOE x 7 days	IHD Acute IWMI + RVMI thrombolysed Reteplase		YES	NO	NO	YES	YES	OHA	MOTHER - IHD. HTN , FATHER IHD	180	85	26.23	50	90/60		10.2	4.78	12.6	213	212	107	29	382	7	Biphasic T waves in V1-V5	NORMAL EF60%	SVD	PTCA
46	Male	Hindu	Business	DOE , Chest pain x 2 days	IHD unstable angina		NO	NO	NO	YES	NO	NONE	NOTHING SIGNIFICANT	160	80	31.25	70	110/70		12.8	3.95	7.7	324	137	83	33	104	32.8	NSR	NORMAL EF 60%	DVD	PTCA
41	Male	Muslim	Business	Chest pain x 6 days	ACS -AWMI , HTN , DM		YES	YES	YES	YES	YES	OHA , ANTIHTN	FATHER - IHD	170	98	33.91	96	160/100		12.3	4.59	15.1	507	159	72	30	284	66.2	Acute AWMI	AKINESIA OF ANT WALL, APEX, EF40%	SVD	PTCA
36	male	hindu	business	chest pain x 4 days	IHD- AWMI , SVD		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	178	80	25.25	94	120/70		14.6	5.07	7.5	518	125	56	31	192	43.1	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTERO SEPTUM, APEX EF-40%	SVD	MEDICAL LINE
42	Male	Christian	Business	Profuse sweating, chest pain x 3 days	NORMAL		NO	NO	NO	YES	NO	NONE	NOTHING SIGNIFICANT	160	80	31.25	85	130/80		15.9	5.9	9.8	285	137	83	33	104	11.4	NSR	NORMAL	NORMAL	MEDICAL LINE
26	Male	Hindu	Business	Chest pain x 4 days	IHD- EVOLVED AWMI, HTN, SVD		NO	YES	YES	YES	YES	ANTI HTN	FATHER - IHD	178	89	28.09	80	140/90		17.8	5.77	10.7	238	208	144	42	108	21	Evolved AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF- 40%	SVD	PTCA
47	Male	Hindu	Business	chest pain x 2 days	IHD- AWMI, SVD		NO	NO	NO	NO	NO	NONE	MOTHER- DM , HTN	169	89	31.16	84	140/90		15.7	5.5	10.4	310	190	139	**29**	382	34.2	Acute AWMI	HYPOKINESIA OF ANTEROSEPTUM, ANTERIOR WALL, APEX EF- 45%	SVD	PTCA
43	Male	Muslim	Business	chest pain x 4 days	IHD with USA, SVD		NO	YES	YES	YES	YES	NONE	MOTHER- IHD, DM , HTN, FATHER- IHD	173	73	24.39	68	120/80		15.8	5.16	12.2	277	167	101	32	171	19.5	NSR	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF-45%	SVD	PTCA
30	Male	Hindu	Business	DOE,chest pain x 1 day	IHD with USA		NO	NO	NO	YES	NO	NONE	NOTHING SIGNIFICANT	175	75	24.49	80	130/80		13.7	3.66	11.6	258	212	108	30	380	98.6	LVH strain	NORMAL EF 60%	NORMAL	MEDICAL LINE
45	Male	Hindu	Farmer	DOE x 10 days	IHD- Evolved AWMI, HTN		NO	YES	YES	YES	YES	ANTI HTN	FATHER, MOTHER- DM, HTN, IHD	160	60	23.44	86	120/80		18.3	5.28	11	323	145	57	26	308	77.7	Evolved AWMI	HYPOKINESIA OF DISTAL IVS, APEX EF-45%	SVD	PTCA
49	Male	Hindu	Farmer	Chest pain x 4 days	ACS - IWMI lysed with streptokinase		NO	NO	YES	YES	NO	NONE	FATHER, MOTHER- DM, HTN, IHD	165	70	25.71	86	120/90		13.2	5.2	8.78	287	140	65	25	249	108.6	Acute awmi	AKINESIA OF INF, INFEROSEPTUM, INFEROLATERAL WALL EF-45%	TVD	PTCA
43	Male	Hindu	Business	DOE x 10 days	IHD- AWMI lysed streptokinase		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	160	78	30.47	68	130/90		16.5	5.52	19.7	387	218	98	46	160	241	Acute AWMI	HYPOKINESIA OF ANTERIOR WALL, EF 55%	SVD	PTCA
18	Male	Hindu	student	Profuse sweating x 2 days	IHD- EVOLVED AWMI with LV apical clot		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	180	60	18.52	83	130/80		13.1	5.74	10.45	422	100	53	24	116	27.4	Evolved AWMI	AKINEISA OF ANT WALL EF- 40%	SVD	PTCA
35	Male	Hindu	Business	DOE, profuse sweating x 4 days	IHD- Evolved AWMI, s/P ptca , SVD		NO	NO	NO	YES	NO	NONE	NOTHING SIGNIFICANT	158	80	32.05	86	120/90		13.9	5.08	12.7	301	153	91	34	141	97.3	IWMI	AKINESIA OF INFERIOR WALL, INFEROSEPTUM, INFEROLATERAL WALL, EF- 40%	SVD	PTCA
46	Male	Hindu	Business	chest pain x 3 days	IHD- AWMI, SVD		NO	YES	NO	YES	YES	OHA	FATHER, MOTHER- DM, HTN, IHD	180	68	20.99	78	130/80		15.1	4.9	11.1	511	219	108	39	160	7	Acute AWMI	AKINESIA OF ANTERIOR WALL, SEPTUM, APEX EF 45%	SVD	PTCA
37	Male	Hindu	Business	chest pain 2 days	IHD- Unstable angina	Risk Factor	YES	NO	NO	NO	YES	OHA	NOTHING SIGNIFICANT	165	70	25.71	83	120/80	INVESTIGATIONS	14.3	5.23	8.18	152	125	56	31	197	139.7	NSR	NORMAL, EF-60%	SVD	PTCA
33	male	hindu	business	DOE, chest pain 1 day	IHD- AWMI,		YES	YES	YES	YES	YES	OHA, ANTI HTN	FATHER - IHD, DM, HTN	155	88	36.63	84	130/80		14.4	4.61	13.1	360	234	119	54	304	80.2	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX, EF 50%	DVD	PTCA
40	Male	Hindu	service	DOEx 2 days	IHD WITH NSTEMI		YES	YES	NO	YES	YES	OHA, ANTI HTN	NOTHING SIGNIFICANT	160	65	25.39	86	130/90		15	5.56	15	370	165	109	35	107	163.5	Old IWMI	IHD, HYPOKINESIA OF ANTERIOR WALL, ANTEROSEPTAL WALL, APICAL SEPTUM, APEX	DVD	PTCA
41	Male	Hindu	BUSINESS	Chest pain x 1 day	IHA- Acute AWMI		YES	NO	NO	YES	YES	OHA	MOTHER-IHD	160	70	27.34	86	120/90		15.2	5.71	8.6	300	152	51	27	370	54	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF40%	SVD	PTCA
40	Male	Hindu	Business	chest pain x 2 days	IHD-OLD AWMI , SVD		YES	NO	YES	YES	YES	OHA	NOTHING SIGNIFICANT	174	85	28.08	68	120/90		13.6	4.71	6.5	226	105	27	41	186	28.6	Old IWMI	NORMAL , EF 60%	SVD	MEDICAL LINE
41	Male	Christian	Business	DOE, profuse sweating x 4 days	IHD WITH AWMI		NO	NO	YES	YES	YES	NONE	FATHER, MOTHER-IHD, HTN	160	75	29.30	75	140/90		13.5	3.91	6.1	281	98	55	30	65	7	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF-45%	SVD	PTCA
43	Male	Hindu	Farmer	DOE ,chest pain x 4 days	IHD WITH UNSTABLE ANGINA		YES	YES	NO	NO	NO	OHA	FATHER - IHD	158	48	19.23	71	130/90		13	4.8	9.3	400	61	18	30	67	22.9	NSR	NORMAL, EF- 60%	DVD	PTCA
29	Male	Hindu	farmer	chest pain x1 day	IHD with Evolved AWMI		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	158	70	28.04	80	120/90		17.4	5.39	9.5	207	76	38	26	62	94.7	Evolved AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF 45%	SVD	PTCA
43	Male	Hindu	Agriculturist	DOE ,chest pain, profuse sweating x 3 days	IHD with AWMI, DVD		NO	NO	NO	YES	YES	NONE	NOTHING SIGNIFICANT	158	80	32.05	84	120/90		12	5.2	8.4	180	228	107	58	313	97.3	Acute AWMI	IHD WITH AWMI, EF- 40%	DVD	MEDICAL LINE
48	male	hindi	farmer	Chest pain x 3 days	IHD - Acute Inferolateral MI		NO	NO	YES	YES	YES	NONE	FATHER , MOTHER - HTN, IHD	160	80	31.25	110	130/80		16	4	6.5	302	120	59	44	84	116.9	Acute Inferolateral wall MI	AKINESIA OF INF WALL, INFEROLATERAL SEGMENT WITH HYPOKINESIA OF ANTEROLATERAL SEGMENT EF 50%	SVD	PTCA
38	Male	Hindu	farmer	DOE, profuse sweating x 4 days	IHD- AWMI, SVD		NO	NO	YES	YES	YES	NONE	MOTHER- IHD , DM FATHER-HTN	167	87	31.20	100	120/90		12.3	5.7	5.9	239	126	57	23	160	72.4	Acute AWMI	AKINESIA OF ANT WALL, APICAL SEPTUM, APEX, EF- 45%	SVD	MEDICAL LINE
49	Female	Hindu	housewife	Chest pain x 3 days	IHD WITH EVOLVED AWMI		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	150	50	22.22	86	120/80		14	5.9	11.9	189	189	105	65	95	310.2	Evolved AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF 45%	SVD	PTCA
41	Male	Hindu	Service	Chest pain x 4 days	IHD WITH AWMI		YES	NO	YES	YES	YES	OHA	FATHER- DM	160	80	31.25	78	120/90		13.8	4.3	8.9	291	211	147	32	159	34.2	Acute AWMI	AKINESIA OF ANT WALL, SEPTUM, APEX EF- 45%	SVD	PTCA
37	Male	Hindu	Business	DOE, profuse sweating x 4 days	IHD-Acute LWMI, TVD		NO	NO	NO	YES	NO	NONE	FATHER- HTN	167	87	31.20	100	120/90		9.7	3.3	10.1	265	181	99	34	240	119.2	LWMI	HYPOKINESIA OF APICAL SEPTUM, APEX EF-50%	TVD	CABG
41	Female	Hindu	Housewife	DOE x 7 days	IHD- AWMI		NO	NO	NO	NO	NO	NONE	NOTHING SIGNIFICANT	160	68	26.56	72	110/70		8.7	4.3	11.3	324	100	91	50	290	140.7	Acute AWMI	AKINESIA OF ANT WALL, SEPTUM, APEX, EF60%	SVD	MEDICAL LINE
49	Female	HINDU	Housewife	chest pain, doe x 5 days	ACS - PWMI		NO	YES	NO	NO	NO	OHA	NOTHING SIGNIFICANT	158	65	26.04	110	130/80		7.9	4.5	7.2	234	199	118	39	186	28.5	NSR	HYPOKINESIA OF ANTEROLATERAL, LATERAL EF 50%	DVD	PTCA
43	Male	Hindu	Business	Chest pain , DOE 4 days	IHD - USA		YES	NO	YES	YES	NO	OHA	NOTHING SIGNIFICANT	160	80	31.25	80	100/70		10.3	5.1	10	90	90	39	27	122	7.3	NSR	NORMAL EF60%	SVD	PTCA
48	Male	Hindu	Business	Profuse sweating , DOE, chest pain- 4 days	IHD- AWMI, SVD, DM		YES	NO	YES	YES	YES	OHA	MOTHER- IHD, DM	174	85	28.08	68	120/90		11.9	4.9	7	143	62	19	13	89	27.8	Acute AWMI	AKINESIA OF APEX, HYPOKINESIA OF APICAL SEPTUM, EF-45%	SVD	PTCA
31	male	hindu	business	Chest pain, DOE x 3 days	IHD- AWMI, SVD		NO	NO	NO	NO	YES	NONE	FATHER-IHD, HTN	160	75	29.30	75	140/90		12.4	4.2	5.7	264	186	84	43	296	10.5	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, APICAL SEPTUM, APEX EF- 60%	SVD	MEDICAL LINE
49	Male	hindu	business	profuse sweating , DOEx 3 days	IHD- ACUTE IWMI WITH RVMI		NO	NO	YES	NO	YES	NONE	MOTHER- DM , HTN	158	48	19.23	71	130/90		12.9	3.72	5.1	90	174	113	42	94	13	ACUTE AWMI	HYPOKINESIA OF BASAL INFERIOR WALL, EF-50%	DVD	PTCA
40	Male	muslim	Shopkeeper	chest pain- 4 days	IHD- ACUTE AWMI, DM, DVD		YES	NO	NO	NO	YES	OHA	FATHER, MOTHER- DM, HTN, IHD	158	70	28.04	80	120/90		8.2	3.89	6.3	294	142	92	38	61	113.1	Acute AWMI	AKINESIA OF APEX, HYPOKINESIA OF APICAL SEPTUM, EF-45%	DVD	PTCA
45	Male	hindu	Business	chest pain x 3 days	IHD - Acute AWMI, DVD		NO	YES	YES	NO	NO	ANTI HTN	MOTHER- DM , HTN	158	80	32.05	84	120/90		16.4	5.78	13.8	242	149	79	62	68	59	Acute AWMI	NO REGIONAL WALL ABNORMALITY, EF 60%	DVD	PTCA
41	Female	muslim	housewife	DOE x 3 days	IHD- EVOLVED AWMI, SVD		NO	YES	YES	NO	YES	ANTI HTN	FATHER-DM, IHD	160	80	31.25	110	130/80		12.5	4.66	10.3	441	180	80	51	200	74.9	Evolved AWMI	AKINESIA OF APEX, HYPOKINESIA OF APICAL SEPTUM, EF-40%	SVD	PTCA
49	Male	hindu	shopkeeper	chest pain x 3 days	IHD- IWMI, SVD		NO	YES	YES	NO	NO	ANTI HTN	MOTHER- DM , HTN	167	87	31.20	100	120/90		14.9	4.81	12.2	302	118	70	30	91	208	IWMI	AKINESIA OF INFERIOR WALL, INFEROSEPTUM, INFEROLATERAL WALL, EF- 40%	SVD	PTCA
28	male	hindu	Pvt company Service	DOE x 4 days	IHD - nonQ AWMI, SVD		NO	NO	YES	NO	YES	NONE	FATHER-IHD, HTN	160	68	26.56	72	110/70		17.1	5.6	6.4	294	210	111	36	314	16.9	Non Q AWMI	HYPOKINESIA OF APICAL SEPTUM, APEX EF 55%	SVD	MEDICAL LINE
40	Femlale	Muslim	housewife	Profuse sweating x 2 days	IHD - old IWMI		NO	NO	NO	NO	NO	NONE	MOTHER- DM, HTN,IHD FATHER - DM	158	65	26.04	110	130/80		12.1	4.9	9.6	266	193	49	38	529	16.4	Old IWMI	AKINESIA OF INFEROSEPTUM, INFEROLATERAL , INFERIOR WALL, EF 45%	SVD	PTCA
40	Female	hindu	Daily wager	chest painx 4 days	IHD- NON Q AWMI, SVD		NO	NO	NO	NO	NO	NONE	FATHER- DM, MOTHER- HTN, IHD	160	80	31.25	110	130/80		8.6	4.37	10.4	440	150	48	48	180	143.1	T WAVE INVERSION IN V2-5	HYPOKINESIA OF APICAL SEPTUM, APEX, EF- 50%	SVD	PTCA
36	Male	hindu	shopkeeper	Chest pain x2 days	IHD- AWMI , SVD		YES	YES	YES	NO	YES	ANTI HTV, OHA	MOTHER- DM , HTN	167	87	31.20	100	120/90		14.4	4.82	10.3	246	154	47	48	69	141.7	Evolved AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM,APICAL SEPTUM, APEX, EF- 45%, LA CLOT +	SVD	PTCA
39	male	hindu	Business	chest pain, sweating, DOE x 3 days	IHD- Acute AWMI , DM, DVD		YES	NO	YES	NO	YES	OHA	FATHER- DM, IHD	160	68	26.56	72	110/70		11.3	4.47	13.2	232	212	121	57	168	116.9	Acute AWMI	AKINESIA OF ANTERIOR WALL, ANTEROSEPTUM, EF- 35%	DVD	PTCA