DOI: 10.22270/jmpas.V10I6.2018 ISSN NO. 2320–7418

International peer reviewed open access journal

Journal of Medical Pharmaceutical and Allied Sciences

Journal homepage: www.jmpas.com CODEN: JMPACO

Research articles

Study on lipoprotein (a) and apo-lipoprotein ratio associated with low grade inflammation in coronary artery disease

Shivasekar Meera*, Thirunavukkarasu Jaishankar, Vinodhini V M

SRM Medical College Hospital & Research Centre, SRMIST, Kattankulathur, Kancheepuram, Tamil Nadu, India

ABSTRACT

Coronary artery disease is a major cause of mortality and morbidity in India. Lipoprotein (a) is a heterologous type of plasma lipoprotein particle with a neutral lipid core and a protein component composed of Apolipoprotein B subunits that are covalently bound to a unique protein known as Apolipoprotein (a). Lipoprotein (a) acts as a pro-inflammatory mediator and may increase the formation of plaque lesions in atherosclerosis. Elevated serum Lipoprotein (a) levels are associated with an increased risk of coronary artery disease. This study aims to assess the level of lipoprotein (a) and Apo-lipoprotein ratio along with highly sensitive C-reactive protein in Coronary artery disease and Coronary artery disease subjects with diabetes patients. This cross-sectional study was conducted in SRM Medical College Hospital and Research Centre on Patients appearing the Department of Cardiology and Medicine. The study was conducted on 300 Patients in age group of ≤55years. After an overnight fasting, blood samples were taken for lipid profile and analyzed for Lipoprotein (a), Apo-lipoprotein B, Apo-lipoprotein A_1, highly sensitive C-reactive protein and Glycated hemoglobin. For Statistical analysis, Analysis of variance and Pearson's correlation was used. Mean levels of Lipoprotein (a), Apo-lipoprotein B, and hs-CRP showed a significant increase in Coronary artery disease and Coronary artery disease subjects with diabetes compared to the control ($p < 0.001$). A significant positive correlation was observed between Lipoprotein (a) and highly sensitive C- reactive protein. In addition to traditional parameters, assessment of Lipoprotein (a), apo-lipoprotein ratio, and highly sensitive C- reactive protein can prove to be a valuable tool in population risk assessment and Coronary artery disease management.

Keywords: Apo lipoprotein, Coronary artery disease, Low-Grade Inflammation, Inflammatory marker

Received – 23/09/2021, Reviewed - 02/11/2021, Revised/ Accepted- 03/12/2021

Correspondence: Meera Shivasekar * ✉ meeras@srmist.edu.in

SRM Medical College Hospital & Research Centre, Kattankulathur, Kancheepuram, Tamil Nadu, India

INTRODUCTION

Coronary artery disease cause 7 million fatalities among adults (21.9 percent of total deaths), expected to climb 26.3 percent by 2030 globally per year, with the majority of these deaths occurring in emerging nations [1]. Lp(a) levels are primarily determined by variations in the Lp(a) and LPA gene loci, which encode Apo (a)[2]. Lipoprotein (a) is similar to the LDL moiety except that it has Apo protein (a) linked to it, which inhibits fibrinolysis. The presence of Apo (a) in atherosclerotic lesions with an Apo B / Apo-A_1 ratio first revealed the role of Lp(a) in the etiology of atherosclerosis [3]. Apo-lipoprotein (a) is a highly polymorphic glycoprotein found in humans. Apo-A1 is the main protein found in HDL. It stimulates the enzyme lecithin-cholesterol acyltransferase (LCAT). Apo (a) of Lp (a) is structurally similar to plasminogen, which is necessary for the capture of cholesterol produced by the foam cell. Apo-lipoprotein B-100 (Apo-B) is a protein that is present in both Lp(a) and LDL B-100[4]. The Apo-B/A_1 ratio could be a better predictor of atherosclerosis than the LDL/HDL ratio.

High sensitivity C-reactive protein (hs-CRP) is an acute inflammatory protein that functions as an indicator of atherosclerosis and it predicts a person's cardiovascular risk[5]. As a result, our study suggested that, rather than being a mediator of vascular disease, Lp(a) and hs-CRP appear to be markers and are redirected to one of the potential risk characteristics of atherosclerosis. CAD patients were studied to examine if elevated blood glucose levels were interrelated with higher incidence of cardiovascular disease. Despite the fact that diabetes is a known cause for CAD. In the absence of diabetes, the incidence of increased blood glucose levels is unclear [6].

The amount of hemoglobin A1c in CAD people may be associated with the severity of coronary artery disease. Glycated hemoglobin levels indicate 2 to 3 months of average increased glucose consumption also postprandial blood sugar level rises, and indicate minimal intra-individual variance [7]. The use of hemoglobin

A1c in the research of type-2 diabetes is a unique clinical technique supported by the Association of American Diabetes, which exposes the correlation among Glycated hemoglobin and microvascular illness. Increased hemoglobin A1c levels are recognized to be an individual risk factor for cardiovascular disease in patients both with and without diabetes. Hemoglobin A1c values of fewer than 7% are thought to be in the range for decreasing the risk of vascular problems [8]. The therapeutic efficacy of routine Lp(a) testing is impossible to distinguish; however, in patients with elevated values of Lp(a), more severe care of those other lipoprotein variables may be necessary.

MATERIAL AND METHOD

This cross-sectional study was done from October 2020 to April 2021.The age group of ≤55 years was comprised of 300 patients on patients visiting cardiology and General Medicine. This study adheres to the Helsinki Declaration and was approved by the SRM Medical College Hospital and Research Centre's institutional ethical council (ECN: 1513/ICE/2018). All participants provided written informed consent.

Inclusion Criteria

This research only included individuals who had just been confirmed with CAD. The eligibility guidelines were used to diagnose a patient of CAD:

a) A documented incidence of a myocardial infarction

b) A clear history of angina pectoris with recorded electrocardiogram (ECG) abnormalities

Group – A (Healthy Controls)

The control group consisted of persons without clinical symptoms of coronary artery disease, diabetes, hypertension, or dyslipidemia.

Group – B (CAD)

Serum glucose value are in the normal level CAD patients

Group – C (CAD+DM)

Previously known diabetic presenting with CAD.

Exclusion Criteria

pregnancy and Patients with corticosteroids, arthritis, and rheumatoid arthritis were excluded

Baseline Measurement

At the time of enrollment, medical and demographic data were obtained, and records were identified before examination. During the clinical session, a questionnaire was used to obtain basic information such as age, gender, and a history of diabetes, medication usage. Before transferring the data to a database, an expert questioner checked the questionnaires for lost data and completeness. The arterial blood pressure was taken in triplicate using conventional techniques, and the averaged values were utilized in the study. For each individual, laboratory report information was recorded.

As part of the physical assessment, a 12-lead resting ECG was conducted. For determining obesity and body composition, following anthropometric measurements were taken.

Fasting blood samples from participants and controls were taken from antecubital and intravenous sites in the morning using a fully aseptic procedure. To separate serum, blood was centrifuged at 2500 rpm for 15 minutes. Glucose estimation and regular lipid profile measurements included plasma TC, TG, LDL-C, HDL-C, TC/HDL-C ratio, LDL-C/HDL-C ratio. The enzymatic end-point glycerol phosphate oxidase-peroxidase technique was used to calculate total cholesterol and triglycerides (Beckmann Coulter AU480 Analyzer). The levels of hemoglobin A1C (HbA1C) were measured by whole blood using the Bio-Rad.

Measurement of Lp(a)

Lipoprotein (a) is a Simple Step Bioassay technique method to monitor humans Lipoprotein (a) proteins in serum samples. TMB substrate is added, which is catalyzed by HRP during incubation, resulting in blue coloring. The intensity of the signal at 450 nm is evaluated in proportion to the amount of bound analyte.

Measurement of hs-CRP

The direct sandwich method was used to calculate hs-CRP using an ELISA kit. Every microplate well contains a biotinylated detection antibody which is specific to hs-CRP as well as an Avidin-Horseradish Peroxidase (HRP) mixture. The optical density is measured using a spectrophotometer at 450 nm (OD).

Measurement of Apo-lipoprotein- B 100

The Human ApoB ELISA Kit is an enzyme immunoassay designed for the detection and quantification of human ApoB (ApoB-100) in blood samples. Using 450 nm as the principal wave length, measure absorbance with a spectrophotometer.

Measurement of Apo-lipoprotein- A_1

Pre-coated and blocked 96-well plates with an Apo-lipoprotein A_1 specific antibody. TMB is catalyzed by Streptavidin-Peroxidase to generate a blue compound that turns yellowish once an acidic stopping solution will be added. Using a micro plate reader, absorbance is measured at 440-460 nm.

Statistical analysis

The data was examined using the Statistical Package for the Social Sciences (SPSS 21.0). The results from the study were given as mean and standard deviation. The difference was statistically significant if the p-value was less than 0.05. The statistical significance of the study and control groups was determined using ANOVA. Pearson correlation coefficient was used to calculate the correlation between different variables

RESULT AND DISCUSSION

A total of 300 Patients were enrolled in this study. Total 78(78%) were male and 22(22%) were female CAD Patients. And 81(81%) were male and 19(19%) were female CAD+DM Patients. And 87(87%) were male and 13(13%) were female Patients were

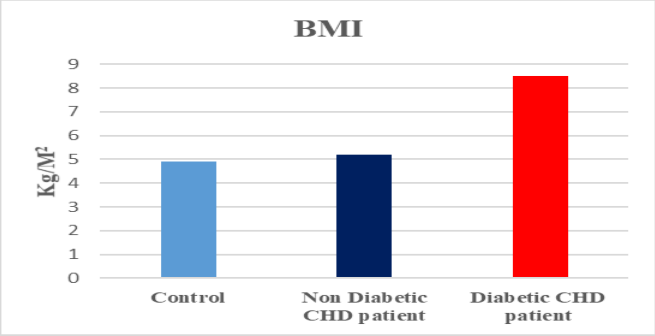
selected as control. There were 42 CAD patients with a family history of CAD and 53 CAD+DM patients with a family history of CAD in the Patients group. Alcohol intake and the sort of diet consumed are examples of lifestyle habits. In the present study the level of BMI, Waist Circumference, Waist Hip Ratio systolic blood pressure were significantly elevated in both CAD+DM and CAD patients compared to controls as depicted in (Table 1)

Table 1: Demographics and baseline characteristics between CAD+DM and CAD Patients and Healthy Controls

Parameters		Controls (n=100)	CAD Patients (n=100)	CAD+DM Patients (n=100)	p-value
Mean age		41.8 ± 9.7	42.3 ± 10.5	49.6 ± 6.4	0.418
Male Sex (%)		87(87.0%)	78(78.0%)	81(81.0%)	-
Female Sex (%)		13(13.0%)	22(22%)	19(19.0%)	-
BMI(kg/m^2)		20.83±0.21	24.51±0.29	25.03±0.19	<0.001
WC (cm)		91.3 ± 9.4	97.4 ± 8.9	98.8 ± 4.3	<0.001
WHR		0.91±0.04	1.08±0.02	1.09±0.03	<0.001
SBP (mm Hg)		111.23 ±17.46	120.16±15.85	122.26±13.95	<0.001
DBP (mm Hg)		74.45 ±7.81	86.21 ±12.13	82.16 ±16.47	<0.001
Diet	Non-vegetarian	64(64.0%)	74(74.0%)	79(79.0%)	-
	vegetarian	36(36.0%)	26(26.0%)	21(21.0%)	-
Alcohol drinking	Drinkers	13(13.0%)	56(56.0%)	65(65.0%)	-
	Non-Drinkers	87(87.0%)	44(44.0%)	35(35.0%)	-
Family history of CAD	Yes	0	42(42.0%)	53(53.0%)	-
	No	100(100.0%)	58(58.0%)	47(47.0%)	-

Values are expressed as mean ± SD*p-value <0.05 is considered Significant

Figure 1 Mean levels of Body mass index between three Groups

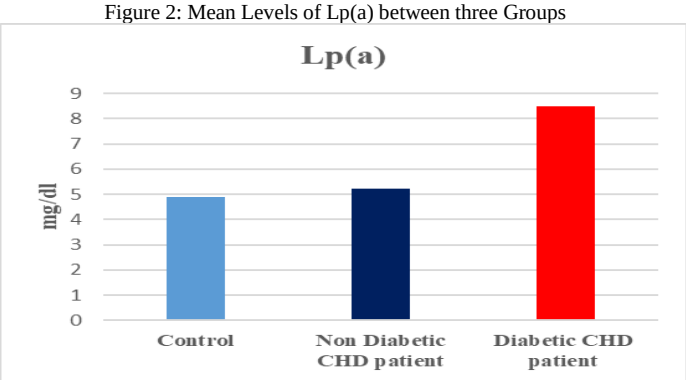


(Figure 1). FPG, Total Cholesterol, Triglyceride, LDL, VLDL, LDL/HDL ratio, TC/HDL ratio, and HbA1c levels are significantly elevated in CAD+DM and CAD when compared to controls, according to the research. The mean HDL-C levels in CAD+DM and CAD patients did not differ significantly.

Assessment of Serum Lipoprotein (a)

We looked at the concentration of Lipoprotein (a) in each of the three groups and found a statistically significant difference. When compared to Group A – Healthy Controls, serum Lipoprotein (a) levels were significantly greater in Group B – CAD Patients and Group C – CAD+DM. Serum Lipoprotein (a) levels differed significantly between Group B and Group C (p< 0.001) (Figure 2).

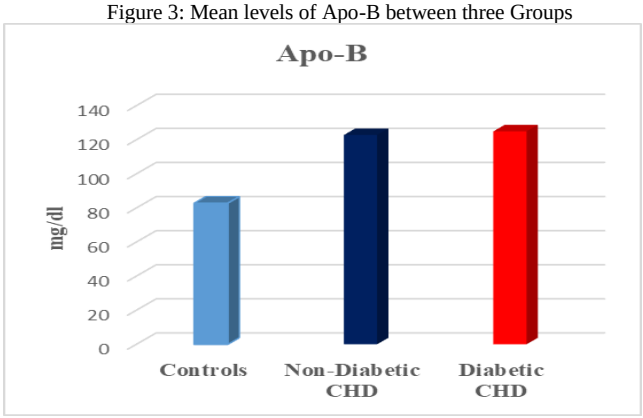
Figure 2: Mean Levels of Lp(a) between three Groups



Assessment of serum Apo-lipoprotein

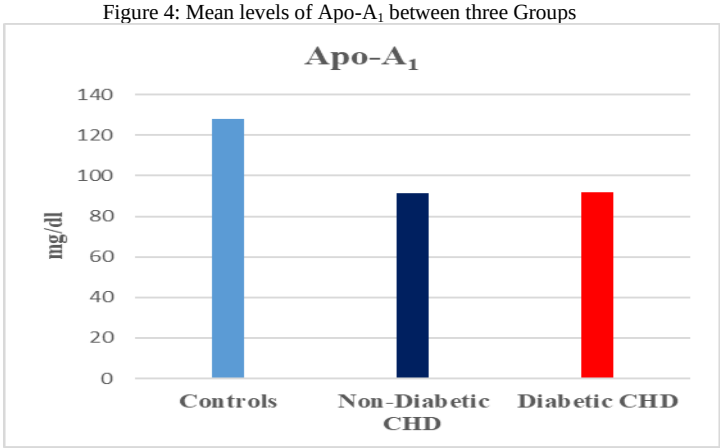
We examined Apo-lipoprotein levels in all three groups and observed a significant difference. Serum Apo-B levels were considerably higher in Group B – CAD patients and Group C – CAD +DM patients compared to Group A – Healthy Controls (Figure 3).

Figure 3: Mean levels of Apo-B between three Groups



Serum Apo-A_1 levels were considerably lower in Group B – CAD Patients and Group C – CAD+DM as compared to Group A – Healthy Controls. Serum Apo-lipoprotein levels differed significantly between Group B and Group C (p<0.001) (Figure 4).

Figure 4: Mean levels of Apo-A_1 between three Groups



When compared to controls, there was a significant rise in the Apo-B 100/Apo-A_1 Ratio in CAD patients.

Assessment of hs-CRP

We evaluated the amount of hs-CRP in all three groups and identified a statistical difference. The serum hs-CRP levels in Group B – CAD Patients and Group C – CAD+DM were substantially higher ($p<0.001$) than in Group A – Healthy Controls.

Assessment of HbA1c

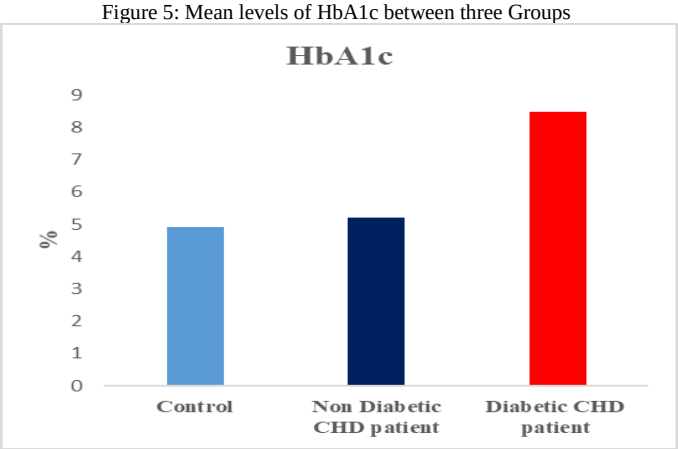
We found a statistically significant change in HbA1c levels across all three groups. Serum HbA1c levels were considerably higher in Group B – CAD Patients (5.21±0.28), $p< 0.001$, and Group C – CAD+DM (8.49±2.32), p 0.001 as compared to Group A – Healthy Controls (4.9±0.17). There was also a statistically significant difference in HbA1c readings between Groups B and C ($p <0.001$) (Figure 5) (Table 2).

Table 2 Comparison of Biochemical Parameter between CAD+DM and CAD Patients and Healthy Controls

Parameters	Controls (n=100)	CAD Patients (n=100)	CAD+DM Patients (n=100)	p- value
FPG (mg/dl)	85.12±3.8	99.11±5.42	169.98±66.28	<0.001
TC (mg/dl)	159.2±15.7	205.46±41.42	236.55±40.33	<0.001
TG (mg/dl)	87.4±19.4	159.7±69	178.86±90.08	<0.001
HDL (mg/dl)	48.4±8.25	34±7	37.83±4.25	<0.001
LDL (mg/dl)	106.4±12.59	143.4±15.23	164.64±27.32	<0.001
VLDL (mg/dl)	15.16±7.64	24.19±9.27	34.08±14.29	<0.001
TC/HDL-C Ratio	3.48±0.49	6.75±1.01	6.50±1.36	<0.001
LDL-C/HDL-C Ratio	2.35±0.53	4.22±0.75	4.41±0.90	<0.001
Lp(a) (mg/dl)	17.77±3.56	35.80±4.36	36.38±4.96	<0.001
Apo-B 100 (mg/dl)	82.77±12	122.79±15.09	124.93±17.37	<0.001
Apo-A_1 (mg/dl)	127.82±3.56	91.29±4.34	91.95±4.93	<0.001
Apo-B/Apo-A_1 Ratio	0.64±0.07	1.33±0.10	1.36±0.09	<0.001
hs-CRP (mg/L)	1.76±0.35	6.33±0.91	7.93±0.54	<0.001
HbA1c (%)	4.9±0.17	5.21±0.28	8.49±2.32	<0.001

Values are expressed as mean ± SD *p value < 0.05 is considered significant

Figure 5: Mean levels of HbA1c between three Groups



Lp(a) is associated with BMI, Waist Circumference, Waist Hip Ratio, Triglyceride, LDL-C, VLDL-C, TC/HDL ratio, and LDL/HDL Ratio, Apo-B, Apo-A_1 hs-CRP in CAD patients. Furthermore, Lp(a) is inversely associated to FPG, TC, HDL-C, and HbA1c. Lp(a) is positively associated with BMI, FPG, Waist Hip Ratio, TC, Triglyceride, HDL-C, LDL-C, VLDL-C, TC/HDL ratio, and LDL/HDL Ratio, Apo-B, Apo-A_1, hs-CRP, and HbA1c in CAD+DM patients. Lp(a) is also adversely associated to waist circumference and HDL-C (Table 3).

Table 3 The Pearson correlations between CAD and CAD+DM patients

Variables	Lp(a)			
	CAD Patients		CAD+DM patients	
	r-value	p- value	r-value	p- value
BMI	0.113 a	<0.001	0.038 a	<0.001
WC (cm)	0.028 a	<0.001	-0.055 b	<0.001
WHR	0.302 a	<0.001	0.069 a	<0.001
FPG (mg/dl)	-0.109 b	<0.001	0.200 a	<0.001
TC (mg/dl)	0.920 a	<0.001	0.011 a	<0.001
TG (mg/dl)	0.155 a	<0.001	0.017 a	<0.001
HDL (mg/dl)	-0.144 b	<0.001	-0.085 b	<0.001
LDL (mg/dl)	0.994 a	<0.001	0.994 a	<0.001
VLDL (mg/dl)	0.153 a	<0.001	0.108 a	<0.001
TC/HDL-C Ratio	0.632 a	<0.001	0.642 a	<0.001
LDL-C/HDL-C Ratio	0.740 a	<0.001	0.800 a	<0.001
Apo-B	0.990 a	<0.001	0.992 a	<0.001
Apo-A_1	0.998 a	<0.001	0.998 a	<0.001
hs-CRP	0.856 a	<0.001	0.279 a	<0.001
HbA1c	-0.037 b	<0.001	0.234 a	<0.001

These findings reveal an increase in TG and a reduction in HDL, which is strongly linked to CAD. In our investigation, we found that both CAD+DM and CAD patients had higher Lp(a) levels. [9] Our result shows the mean level of Fasting Plasma Glucose and HbA1c are significantly increased when compared to control and is strongly associated with CAD. In CAD Patients, Lp (a) is negatively correlated with Fasting Plasma Glucose (r = -0.109) and HbA1c (r = -0.037). Our study, for the first time, suggested that Patients with high Lp(a) and CAD patients had high risk for CAD in south Indian population. Lp (a) positively correlated with BMI, WC, WHR(r = 0.302) (Table 3). Lp(a) levels were linked to LDL cholesterol in a research by Pedreno et al[10,11]. Like LDL, Lp(a) undergoes oxidative alteration to become a substrate for macrophage absorption, therefore fueling the development of foam cells[12].

The pathogenic mechanisms of Lp[a] surplus involve elevated thrombogenesis as well as impaired fibrinolysis by trying to compete with plasminogen, inhibition of transforming growth factor, plaque destabilization, elevated migration and smooth muscle cell proliferation, occlusive thrombus formation, impaired formation of collateral vessels, elevated oxidation absorption, management of LDL-C, and up regulation in expression of plasminogen activator inhibitor (PAI-I)[13].

In this study Lp(a) is positively correlated with Apo-lipoproteins and its ratios between both CAD Patients and CAD+DM patients. The combined detection of Lp(a) and the Apo-lipoproteins and its ratios proved very effective in pinpointing Patients with coronary artery disease. In the AMORIS research, Walldius et al. discovered that high Apo-B and low ApoA_1 are stronger predictors of CAD death than standard cholesterol measurement [14].

The Apo-B/ Apo-A_1 ratio reflects a balance of pro-atherogenic and anti-atherogenic components [15].According to Sniderman et al., higher Apo-B levels are associated with an increased risk of CAD [16]. Lp(a) concentrations were shown to be highly linked with hs-CRP levels in CAD Patients, indicating that

Lp(a) may also serve as an acute-phase reactant[17].An increase in plasma CRP levels results in atherogenic plaque. Which procoagulant and infective substances trigger procoagulant and inflammatory responses [18].Lp(a) is significantly correlated with HbA1c (0.234) in diabetes Patients and negatively correlated in CAD Patients in our study (-0.037).Glycated hemoglobin (HbA1c) was shown to be more accurate in predicting the severity of diabetes than fasting glucose levels[19].Elevated HbA1c levels are associated with significant coronary artery disease and are a major predictor of mortality in people who are not diabetic [20,21].HbA1c is the most important risk factor determining the severity of coronary artery disease, according to Saleem and Yao Liu.

CONCLUSION

The development of CAD at a young age is a complex disease with significant morbidity and death. Lp(a), Apo-A_1 and Apo-B, as well as hs-CRP, are emerging biochemical risk indicators for predicting young people with early cardiovascular risk. This study shows that adding Lp(a), Apo-lipoproteins, hs-CRP to the conventional lipid profile, which might be beneficial for predicting early development of CAD. Lp(a) is not routinely tested as part of a regular lipid profile since measuring methods are technically difficult.

Financial support & sponsorship None

Conflicts of Interest None

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How to cite this article

Shivasekar Meera, Thirunavukkarasu Jaishankar, Vinodhini V M, 2021. Study on lipoprotein (a) and apo-lipoprotein ratio associated with low grade inflammation in coronary artery disease. Jour. of Med. P'ceutical & Allied. Sci. V 10 - I 6, 2018, P- 3981 - 3985. doi: 10.22270/jmpas.V10I6.2018