International Journal of Research in Medical Sciences
Hossan MS et al. Int J Res Med Sci. 2023 Sep;11(9):3168-3174
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20232508

Original Research Article

Correlation of lipoprotein (a) level with severity of coronary lesion in coronary heart disease patients

M. Sawkat Hossan[1]*, Prabir Kumar Das[2], Saurav Das[1], Partha Bhattacharyya[1], Khondaker M. Ismail[1], Iqbal Mahmud[1], Syed Miskatun Nur[1], M. Najim Uddin[2], Abu Sayem Mohammad Omar Faroque[1], M. Emran Hossain[2]

[1]OSD (DGHS), Deputed to Chittagong Medical College, Chittagong Medical College, Chattogram, Bangladesh
[2]Department of Cardiology, Chittagong Medical College, Chattogram, Bangladesh

Received: 07 June 2023
Revised: 13 August 2023
Accepted: 14 August 2023

***Correspondence:**
Dr. M. Sawkat Hossan,
E-mail: sawkat1@gmail.com

ABSTRACT

Background: Cardiovascular diseases (CVDs) are the leading cause of death in developing nations, especially in low and middle-income countries (LMICs). Bangladesh has been undergoing an epidemiological transition from communicable to non-communicable diseases. This study aimed to investigate the association of risk factors with coronary heart disease (CHD) in patients from Bangladesh.
Methods: This cross-sectional observational study was conducted in the department of cardiology, Chattogram Medical College and Hospital, Chattogram, Bangladesh from July 2018 to June 2019. A total of 100 patients were enrolled.
Results: This cross-sectional study enrolled 100 CHD patients with a mean age of 53.21±10.29 years. The majority were obese (64, 64.0%), and hypertension was the most prevalent risk factor (77, 77.0%), followed by smoking (65, 65.0%) and dyslipidemia (58, 58.0%). Most patients had triple vessel disease (53, 53.0%), and significant CHD (81, 81.0%). Patients with severe CHD had higher levels of blood LDL-C, triglycerides, and Lp(a). Lp(a) levels, history of dyslipidemia, and LDL-C were independently associated with a Gensini score ≥20. These findings emphasize the independent association between Lp(a) and CHD severity, warranting greater attention to patients with elevated Lp(a) levels.
Conclusions: This study suggested that Lp(a) is an independent risk factor for CHD in patients from Bangladesh. More attention should be paid to such patients with elevated Lp(a) level.

Keywords: CHD, CVD, Lipoprotein, Risk factors

INTRODUCTION

Cardiovascular diseases (CVDs) have been the foremost reason for death in developing nations, mostly in low and middle-income countries (LMICs) of the last century.[1] World Health Organization (WHO) stated that CVDs were the causes of 17.5 million deaths around the globe in 2012, of which 80% occurred in LMICs (WHO 2015) and 85% of all universal disabilities ascended from CVDs.[2,3] Bangladesh has been undergoing an epidemiological change from communicable disease to non-communicable disease. The overall mortality rate has lessened considerably over the last couple of decades.[4] Chronic diseases, especially the 'fatal four' i.e. CVD, cancer, chronic respiratory disease, and diabetes are increasing at an alarming rate and causing deaths.[5] CHD is a vital medical and public health matter since it is the commonest reason for death all over the world.[6] The occurrence of CHD varies considerably by population and may be up to 10-fold.[7] The exact prevalence of CHD

in Bangladesh is not known and only a limited number of small-scale epidemiological studies are accessible. Even though marked disparity in values, there seems to be a rising prevalence of CHD in Bangladesh.[8] Many aspects are accountable for causing CHD, but, notably, 5 to 10 percent of CHD patients have none of the known risk factors.[9] Risk factor alteration is an essential part of the management of patients who have or are at high risk for CVD.[10] As well as established cardiovascular risk factors, clinical research has known more than 100 other conditions that may be related to a growing threat of CVD.[11] As a result of declines in morbidity and mortality attributable to hypertension, smoking, and dyslipidemia, the relative contribution of new threats to the total load of CVD is probable to increase.[12] Lipoprotein (a) [Lp(a)] is a subclass of low-density lipoprotein (LDL) that has newly increased biomarker importance due to its association with CVD. It is a circulating lipoprotein in which the basic apolipoprotein B-100 (apoB100) on an LDL particle is altered by the covalent addition of another protein, namely, apolipoprotein (a).[13] The relationship between Lp(a) levels and the severity of coronary atherosclerosis in patients with unsteady angina or acute MI has been examined in numerous studies with controversial results.[14] The potential value of small apo(a) isoforms in predicting severe angiographically demonstrable atherosclerosis remains unclear. Elevated serum Lp(a) is an independent interpreter of CHD and myocardial infarction (MI).[15] Studies have rarely attempted to document the correlation between the level of Lp(a) and the complexity of coronary lesions in CHD patients, in Bangladesh. In this context, this study aimed to find the correlation between Lp(a) and the severity of coronary lesions in CHD patients at the cardiology department of Chattogram Medical College Hospital, Chattogram, Bangladesh.

General objective

To analyze the distribution of different risk factors of coronary artery disease in CHD patients.

Specific objectives

To find out the association among Lp(a) with LDL-C and HDL-C, cholesterol, triglyceride, and fasting blood sugar.

To find out the relationship among Lp(a) with other demographic variables (Age, sex, history of smoking and family history of CHD, BMI, waist height ratio, HTN.

METHODS

This cross-sectional observational study was carried out in the department of cardiology, Chattogram Medical College and Hospital, Chattogram, Bangladesh from July 2018 to June 2019. Due to resource constraints, 100 patients (N=100) were enrolled in the study.

A consecutive sampling technique was followed. Informed written consent was taken from all study subjects. History was taken and clinical examinations were performed following the standard procedure of clinical methods. Demographic profiles of the patients which included age, major risk factors like diabetes, hypertension, dyslipidemia, smoking, prior CHD and family history of CHD were recorded. The body height was measured in the standing position without shoes. Weight was measured similarly without shoes and heavy dresses. Waist circumference (WC) was measured at the mid-point between the distal border of the ribs and the top of the iliac crest with subjects standing at the end of a normal expiration. Hip circumference was measured at the largest circumference of the buttocks. With all aseptic precautions 5 ml of fasting blood sample was drawn with the subject in a sitting position, with limited use of a tourniquet. Fasting lipid profile, serum Lp(a), and fasting plasma glucose was determined on the day of blood collection. If not, the sample after centrifugation was stored at 4°C for up to 4 days. Coronary angiography was performed by percutaneous femoral or radial approach. Coronary angiograms were obtained for each coronary vessel in ≥2 projections. Analysis of the coronary angiograms was performed visually by experienced interventional cardiologists working in the CMCH catheterization laboratory. The severity of the CHD was assessed by the Gensini score. Ethical clearance was taken from the hospital.

Data analysis

The statistical analysis was carried out by using Statistical Package for Social Sciences (SPSS-23). Continuous variables were presented as mean ± standard deviations (SDs) (normal distribution data) or median with 25^{th} and 75^{th} percentile (abnormal distribution data, Lp(a); variables were compared by Student's t-test (normal distribution data), Mann-Whitney U-test or Kruskal-Wallis test (abnormal distribution data). Categorical variables were summarized as frequencies with percentages and compared with the chi-square test. To identify the factors which were independently associated with high Gensini scores, binary univariate, and multivariate logistic regression analyses were performed.

Inclusion Criteria

Acute coronary syndrome patients who had undergone coronary angiography within one month of the index event.

Exclusion criteria

Patients who refused to give consent. Patients with some co-morbid conditions (stroke, renal failure, liver failure, acute infection, malignant diseases). Patients with congenital or valvular heart disease who suffered MI. Pregnancy and lactation.

RESULTS

Among the study population (N=100), the mean age of the patients was 53.21±10.29 years and most of the study patients (68, 68%) were in the 40 to 60 years of age group. Around three-fifths of the participants (70, 70.0%) were male. The majority of the patients (64, 64.0%) were obese, and ninety-one patients (91, 91.0% had central obesity by waist height ratio (Table 1).

Table 1: Distribution of the study population based on demographic characteristics (N=100).

Characteristics	(N, %)
Age in years	
<40	7, 7.0
40-60	68, 68.0
Mean±SD	53.21±10.29
>60	25, 25.0
Gender	
Male	70, 70.0
Female	30, 30.0
BMI, in kg/m^2	
Normal (<23)	21, 21.0
Overweight (≥23-24.99)	15, 15.0
Obese (≥23)	64, 64.0
Mean±SD	26.05±3.44
Waist height ratio	
Normal (<0.5)	9, 9.0
Central obese (≥0.5)	91, 91.0
Mean±SD	0.55±0.04

Based on risk factors, hypertension was the most prevalent risk factor in the study population (77, 77.0%) followed by smoking (65, 65.0%), dyslipidemia (58, 58.0%), and diabetes mellitus (35, 35.0%) and thirty- two patients (32, 32.0%) reported to have a family history of CHD. Based on, the lipid profile status and fasting blood glucose status of the patients, fifty-seven (57, 57.0%), twenty-one (21, 21.0%), ninety-one (91, 91.0%), ninety-three (93, 93.0%) and twenty (20, 20.0%) patients had high cholesterol, LDL-C, HDL-C, triglyceride and fasting blood sugar (FBS) level respectively. Only nineteen patients (19, 19.0%) had normal Lp(a) levels and more than half of them (53, 53.0%) had Lp(a) level >30 mg/dl regarded as a high-risk level. According to the extent and severity of the CHD, most of the patients (53, 53.0%) had triple vessel disease, most had (81, 81.0%) significant CHD, and around half of them (46, 46.0%) had genuine score >40 (Table 2). The association between Lp(a) levels of the patients with their angiographic severity and extent was described in Table 6. It shows that blood Lp(a) levels had a significant association with the extent and severity of CHD as assessed by the Gensini score. No significant difference in Lp(a) levels among different groups was found in the present study. Likewise, there were no significant differences in Lp(a) levels concerning the gender and smoking status of the patients. No significant difference in median Lp(a) levels among different risk groups was found in the present study (Table 3).

Table 2: Distribution of the study population based on several risk factors (N=100).

Risk factors	(N, %)
Family history of CHD	32, 32.0
Hypertension	77, 77.0
Diabetes mellitus	35, 35.0
Dyslipidaemia	58, 58.0
Smoking (current and ex)	65, 65.0
Total cholesterol	
Normal	43, 43.0
High	57, 57.0
LDL cholesterol	
Normal	79, 79.0
High	21, 21.0
HDL cholesterol	
Normal	9, 9.0
High	91, 91.0
Triglyceride	
Normal	7, 7.0
High	93, 93.0
Fasting blood glucose	
Normal	80, 80.0
High	20, 20.0
Lp(a) category	
Normal risk (<14 mg/dl)	19, 19.0
Borderline risk (14-30 mg/dl)	28, 28.0
High risk (>30 mg/dl)	53, 53.0
Number of vessels involved	
Single vessel	19, 19.0
Double vessel	28, 28.0
Triple vessel	53, 53.0
Significant CHD	
No	19, 19.0
Yes	81, 81.0
Gensini score	
<20	24, 24.0
20-40	30, 30.0
>40	46, 46.0

There was a positive but weak statistically significant correlation observed between Lp(a) levels and total cholesterol and LDL-C levels. In contrast, no significant correlation was found between Lp(a) levels and FBS, HDL-C, and triglyceride levels (Table 4). The study patients were divided into two groups (Gensini score <20 and Gensini score ≥20) according to coronary artery disease severity. Based on the association between different demographic and risk factors related variables of the patients with the severity of CHD, there were significant associations between gender, dyslipidemia, and smoking. Male patients, patients with dyslipidemia, and smokers were more likely to have severe CHD in comparison to their counterparts.

Table 3: Association between Lp(a) with CAG findings, demographic behavioral, and risk factors of CAD.

Parameters	Median (IQR) Lp(a), mg/dl	P value
Number of vessels involved		
Single vessel	27 (16.1-41.9)	<0.001*
Double vessel	26.8 (12.8-44.2)	
Triple vessel	44.5 (30.2-66.3)	
Significant CHD		
No	22.9 (12.9-33.6)	0.042#
Yes	31.9 (19.4-50.7)	
Gensini score		
<20	19.5 (11.5-53.3)	0.001*
20-40	25.7 (17.4-61.3)	
>40	41.9 (29.5-53.3)	
Age in years		
<40	31.4 (20.2-48.6)	0.168*
40-60	29.3 (17.2-48.9)	
>60	34.3 (32.0-61.0)	
Gender		
Male	31.3 (18.3-48.7)	0.585#
Female	30.9 (18.9-51.2)	
Smoking		
Non-smoker	33.6 (17.0-59.1)	0.342#
Current or ex-smoker	30.7 (18.9-46.7)	
Hypertension		
No	31.4 (17.0-41.9)	0.499#
Yes	31.2 (18.9-51.5)	
Diabetes		
No	31.9 (17.8-3.4)	0.580#
Yes	31.2 (19.1-44.2)	
Dyslipidemia		
No	32.9 (16.8-51.3)	0.598#
Yes	30.3 (18.9-46.6)	
Family history of CAD		
No	31.9 (20.1-51.0)	0.358#
Yes	30.5 (14.7-47.5)	

Table 4: Correlation between Lp(a) and other lipid profile and FBS.

Parameters	Spearman correlation coefficient	P value
Fasting blood glucose, gm/dl	0.047	0.462^{ns}
Total cholesterol, mg/dl	0.125	0.045^{s}
LDL-C, mg/dl	0.275	0.006^{s}
HDL-C, mg/dl	-0.194	0.053^{ns}
Triglyceride, mg/dl	0.127	0.209^{ns}

s: Statistically significant; ns: Statistically not significant

On the other hand age, HTN, DM, family history of CHD, BMI, and waist-to-height ratio had no significant association with the severity of CHD. Based on the association between different biochemical parameters of the patients with the severity of CHD, blood LDL-C, triglyceride, and Lp(a) levels were significantly higher among the patients with severe CHD in comparison to their counterparts (Table 5). Variables that were found to have a significant association in univariate analysis were entered into the model. Besides Lp(a) levels, a history of dyslipidemia and LDL-C levels were also independently associated with a Gensini score ≥20 (Table 6).

Table 5: Univariate association between demographic and risk factor variables and severity of CHD.

Variables	Severity of CHD		P value
	Gensini score <20 (n=24)	Gensini score ≥20 (n=76)	
Age, in years	53.6±10.1	53.1±10.4	0.841#
Male gender	11 (45.8%)	59 (77.6%)	0.003*
Presence of HTN	18 (75.0%)	59 (77.6%)	0.789*
Presence of DM	6 (25.0%)	29 (38.2%)	0.239*
Presence of dyslipidemia	8 (33.5%)	50 (65.8%)	0.005*
Smoker	11 (45.8%)	54 (71.1%)	0.024*
F/H of CHD	10 (41.7%)	22 (28.9%)	0.244*
BMI, kg/m^2	25.5±3.1	26.2±3.5	0.368#
Waist to height ratio	0.54±0.03	0.55±0.04	0.625#
Fasting blood sugar, mg/dl	108.8±17.7	114.1±26.4	0.369*
Total cholesterol, mg/dl	191.2±45.5	192.7±49.7	0.890*
LDL-C, mg/dl	104.1±15.3	123.3±28.7	0.002*
HDL-C, mg/dl	36.8±4.8	34.8±6.7	0.186*
Triglyceride, mg/dl	219.6±71.1	278.0±89.5	0.004*
Lipoprotein(a), mg/dl	19.5 (-11.5-35.5)	32.1 (26.4-50.8)	0.014#

Table 6: Independent predictive factors of having severe CHD by CAG.

Variables	Adjusted OR	95% CI		P value
		Lower	Upper	
Male gender	4.46	0.82	24.41	0.085^{ns}
Dyslipidaemia	3.43	1.07	10.92	0.037^{s}
Smoking	1.23	1.00	1.06	0.756^{ns}
LDL-C	1.03	1.00	1.06	0.036^{s}
Triglyceride	1.00	0.99	1.01	0.970^{ns}
Lipoprotein(a)	2.03	1.01	4.06	0.018^{s}

CI=Confidence interval, OR=Odds ratio.

DISCUSSION

The concept of "risk factors" in CHD was initially proposed by the Framingham heart study (FHS) which demonstrated the epidemiologic relations of smoking, blood pressure, and cholesterol levels to the occurrence

of coronary artery disease (CHD).[16] The findings were truly revolutionary as they helped bring about changes in medical science. This study aimed to analyze the distribution of different risk factors of coronary artery disease in CHD patients.

In our analysis, the mean age of the patients was 53.21±10.29 years and most of the study patients (68, 68%) were in the 40 to 60 years of age group. A related study found that patients with 75 to 84 years were at high risk of CVD.[17] From a review article, patients with a mean age of 68 years were at high threat of CVD.[18] Another similar journal found that the mean age of the participant was 59.6 years.[19] The majority of the participants (70, 70.0%) were male found in this study. Several studies have identified males were at high risk of CVD.[20,21] A contradictory study found that most of the women were female and were at high risk of CVD.[17] In this current study, the majority of the patients (64, 64.0%) were obese. An article published in "current problem in cardiology" found that the threat of CHD rose in patients with growing severity of overweight or obesity.[22] Another article showed that most of the CHD patients were overweight or obese (BMI>25 kg/m^2) and 58.2% were centrally obese.[23] In our study, hypertension was the most common risk factor in the study population (77, 77.0%) followed by smoking (65, 65.0%), dyslipidemia (58, 58.0%), and diabetes mellitus. In contrast to our study, among the studied patients, the highest percentages had a history of smoking (86%) followed by a family history of premature CHD (74%), chewing tobacco (44%), hypertension (36%), history of angina (32%), diabetes mellitus (28%) and dyslipidemia (26%) in non-severe CHD group.[24] Hypertension was the commonest risk factor, followed by smoking and DM found in another study.[25] In this present analysis, lipid profile status and fasting blood glucose status of the patients, fifty-seven (57, 57.0%), twenty-one (21, 21.0%), and twenty (20, 20.0%) patients had high cholesterol, LDL-C, HDL-C, and fasting blood sugar (FBS) level respectively. While Lp(a) was an independent risk factor for severe CHD, hypertension, and DM, could not be identified as such. This is probably related to the high prevalence of hypertension and DM in our patients (77%) and the reduced effect of hypertension on coronary atherosclerosis compared to LDL-C, which has already been described in prior studies.[26] Another study found in their study that the prevalence of DM, HTN, hyperlipidaemia, family history of CHD and cigarette smoking was 27.3%, 29.5%, 39.1%, 5.8% and 26.3% respectively among the patients with CHD.[27] In the present study, a positive but weak correlation was observed between Lp(a) levels and LDL-C and total cholesterol levels. There was much controversy regarding the correlation between Lp(a) and other lipid profile. Lima et al observed no correlation between Lp(a) and other variables of the lipid profile in the subjects studied. Another article depicted that there was no any significant correlation in between Lp(a) and LDL.

However, concomitant elevations of Lp(a) and LDL cholesterol have been reported to have synergy in elevating risk in both men and women for CAD.[28]

In our study, most of the patients (53, 53.0%) had triple vessel disease, most of them had (81, 81.0%) significant CHD and around half of them (46, 46.0%) had Gensini score >4. Another study revealed that vessel score I, and II were found in 14%, and 22% of patients. A linear increase of genuine scores with increasing Lp(a) serum levels, was consistent with the findings of the other study describing a stepwise increase of CHD risk with some increased levels of Lp(a).[29] In this current study, based on the association between different demographic and risk factors related variables of the patients with the severity of CHD, there were significant associations between gender, dyslipidemia, and smoking. A similar article depicted that multivariate analysis showed a significant relationship between age, diabetes, hyperlipidemia, gender, and severity of CHD.[28] A dissimilar study suggested that normal angiograms in (29.3%) versus single vessel (14.3%), and triple vessel disease (21.3%) were non-significant.[30]

Clinical implications of the present study were that in the patients who had conventional cardiovascular risk factors with high Lp(a) levels, more severe CHD may be found, and the global cardiovascular risk may be increased. Thus, we can suggest that when assessing this risk in a CHD patient, Lp(a) levels should be considered.

The present study was conducted with very small sample size and in one specific hospital. So, the findings of the study might not represent the complete scenario of the demographic.

CONCLUSION

In summary, this study proved that there was a distinct link between Lp(a) and CHD. Higher Lp(a) levels were found in the patients who had more severe coronary stenosis, and Lp(a) was independently correlated with the existence and severity of CHD in patients who had multiple vascular diseases as measured by the Gensini Score. The new study's therapeutic relevance suggests that patients with elevated Lp(a) levels need to receive extra attention.

Recommendations

Lp(a) can be recommended as an alternative noninvasive diagnostic tool to predict the complexity of the blockage of the coronary arteries that can be performed before other invasive procedures. Thus far the relationship of lipoprotein Lp(a) to atherosclerotic disease has been demonstrated solely by cross-sectional statistical associations. Prospective studies and eventually studies of prophylactic intervention will be necessary to clarify the atherogenic role of this lipoprotein.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

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Cite this article as: Hossan MS, Das PK, Das S, Bhattacharyya P, Ismail KM, Mahmud I, et al. Correlation of lipoprotein (a) level with severity of coronary lesion in coronary heart disease patients. Int J Res Med Sci 2023;11:3168-74.