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FREQUENCY OF RAISED LIPOPROTEINS Lp(a) IN YOUNG INDIVIDUALS WITH FAMILY HISTORY OF ISCHEMIC HEART DISEASE

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ABSTRACT

Background: Elevated lipoprotein (a) [Lp(a)], a genetically determined independent risk factor, is implicated in early atherosclerotic cardiovascular disease. Its distribution among young individuals with a family history of ischemic heart disease (IHD) warrants evaluation for timely primary prevention, since these individuals may carry this latent risk prior to the emergence of conventional lipid abnormalities.

Objective: To determine the frequency of raised lipoprotein Lp(a) in young patients with a family history of ischemic heart disease.

Methods: This cross-sectional study was conducted at the Department of Cardiology, Pakistan Institute of Medical Sciences (PIMS), Islamabad, from February 7, 2026 to June 6, 2026, following IRB approval. A total of 245 young individuals aged 20–40 years who were first-degree relatives of patients with ischemic heart disease were enrolled through non-probability consecutive sampling. Serum Lp(a) was measured by ELISA; levels exceeding 125 nmol/L were defined as raised. Data were analyzed using SPSS v.26.

Results: Mean age was 31.2 ± 5.4 years; 133 (54.3%) participants were aged 31–40 years. Raised Lp(a) was observed in 98 (40.0%) individuals. Stratified analysis revealed significantly higher frequency of raised Lp(a) in those aged 31–40 years (45.1% vs. 33.9%; p=0.042), smokers (48.8% vs. 35.6%; p=0.038), and participants with any comorbidity (44.9% vs. 32.7%; p=0.036).

Conclusion: Elevated Lp(a) is prevalent among young individuals with a family history of ischemic heart disease. Raised levels are significantly associated with older age within the study range, smoking, and presence of comorbidities. Early Lp(a) screening is recommended in this high-risk group.

KEYWORDS: Ischemic Heart Disease, Young Individuals, Family History, Lipoprotein Lp(a), Cross-Sectional Study

INTRODUCTION

Cardiovascular disease remains the foremost cause of morbidity and mortality worldwide. Ischemic heart disease (IHD), characterized by partial or complete occlusion of major coronary arteries, encompasses conditions including acute myocardial infarction (MI) and angina pectoris.(1) Most patients respond adequately to standard medical therapy and revascularization strategies such as percutaneous coronary intervention and coronary artery bypass grafting; however, a subset with diffuse or refractory disease presents significant management challenges.(2)

Although IHD has historically been regarded as a condition of middle-aged and elderly individuals, its occurrence in younger populations is increasingly recognized. In developing countries, ischemic events appear to manifest approximately a decade earlier than in industrialized nations.(3) Among modifiable risk factors, dyslipidemia, tobacco use, and physical inactivity are well established. Non-modifiable determinants including family history, age, and ethnic background confer additional and unalterable risk.(4)

Lipoprotein (a) [Lp(a)] was first characterized in 1963. Its plasma levels are predominantly genetically determined, with heritability estimated at 70–90%, rendering Lp(a) a constitutive rather than acquired risk factor.(11) The Lp(a) phenotype is fully expressed by the second or third year of life, and plasma concentrations demonstrate strong tracking across repeated measurements throughout childhood and adult life.(8) Prospective and Mendelian randomization studies have established Lp(a) as an independent, causative contributor to atherosclerotic cardiovascular disease, coronary artery disease, and stroke.(7,12)

Despite growing recognition of Lp(a)'s clinical relevance, it is frequently absent from routine lipid panels. Its levels respond minimally to lifestyle modification and statin therapy, distinguishing it from LDL cholesterol and limiting conventional pharmacological management.(8) Identification at an early age is therefore particularly valuable, as it creates an opportunity for heightened surveillance, aggressive modification of co-existing risk factors, and emerging Lp(a)-lowering therapies where indicated.

Young individuals with a first-degree family history of IHD represent a genetically enriched cohort, in whom atherogenic lipid abnormalities including elevated Lp(a) may be present decades before clinical events. While existing literature predominantly addresses Lp(a) in older or general populations, evidence from young high-risk individuals particularly in South Asian settings remains limited. The present study was therefore undertaken to determine the frequency of raised Lp(a) in this clinically important group and to examine its distribution across relevant demographic and clinical subgroups.

MATERIALS AND METHODS

Study Setting and Design

This cross-sectional study was conducted at the Department of Cardiology, Pakistan Institute of Medical Sciences (PIMS), Islamabad, from 7th February 2026 to 6th June 2026, following approval from the Institutional Review Board vide No. F-5-2/2024(ERRC)/PIMS dated 17th July 2025. Written informed consent was obtained from all participants prior to enrolment.

Sample Size

Male and female individuals aged 20–40 years who were first-degree relatives (parents, siblings, or offspring) of patients admitted with a confirmed diagnosis of IHD were eligible for inclusion. IHD was defined as: (a) Myocardial infarction history of resting chest pain (VAS >4) lasting more than 30 minutes, not relieved by sublingual nitrates, with ECG evidence of ST-segment elevation or depression; or (b) Angina pectoris history of exertional chest pain (VAS >4) lasting less than 30 minutes and relieved by sublingual nitrates.

Includion/Exclusion Criteria and Sample Size Calculations

Participants with a personal history of coronary artery disease, cocaine use, blood dyscrasias including thrombophilia, familial hypercholesterolemia, or other lipid metabolism disorders, and underlying prothrombotic conditions were excluded. Sampling was performed by non-probability consecutive method. Sample size was calculated as 245, using the WHO sample size formula based on an anticipated frequency of raised Lp(a) with family history of IHD of 6.1% (Mehta et al.),(9) a margin of error of 3%, and a confidence level of 95%.

Demographic and clinical data including age, gender, BMI, residential status, educational level, occupation, socioeconomic status, relation to the index patient, and smoking history were recorded by structured history-taking and review of previous records. Comorbidities were ascertained through the following standardized criteria: diabetes mellitus was defined as HbA1c >6.5% or history of antidiabetic medication use for more than six months; hypertension was defined as blood pressure exceeding 140/90 mmHg on two separate occasions or current use of antihypertensive medication for more than six months. These were confirmed from patient history and available prior medical records. A 5 mL venous blood sample was collected from the antecubital fossa of the non-dominant arm under aseptic technique. Samples were dispatched to the hospital laboratory for estimation of serum Lp(a) by enzyme-linked immunosorbent assay (ELISA), performed by an experienced pathologist blinded to participants' clinical data, as part of the laboratory's routine internal and external quality control protocols. Serum Lp(a) levels exceeding 125 nmol/L were classified as raised, in accordance with the European Atherosclerosis Society consensus thresholds.(12) The laboratory investigation was funded as part of routine clinical care at PIMS; no additional study-specific funding was obtained and no treatment was initiated by the study team for identified elevated Lp(a) levels, with results communicated to the treating physician for clinical management as appropriate.

Data were entered and analyzed using SPSS version 26.0. Continuous variables (age, BMI, Lp[a]) were assessed for normality using the Shapiro-Wilk test, and reported as mean ± SD or median (IQR) accordingly. Categorical variables were expressed as frequencies and percentages. Raised Lp(a) was stratified against age, gender, BMI, smoking history, residential status, education, occupation, socioeconomic status, relation to patient, and comorbidities to identify potential effect modifiers. Post-stratification chi-square or Fisher's exact test was applied at a 5% level of significance.

RESULTS

The mean age of participants was 31.2 ± 5.4 years and mean BMI was 26.4 ± 3.1 kg/m². Serum Lp(a) demonstrated non-normal distribution (Shapiro-Wilk p <0.001), with a median value of 79.2 nmol/L (IQR: 48.2–90.1). Age and BMI followed normal distributions (Table 1).

Table 1: Descriptive Statistics of Continuous Variables (n=245)

Variable	Mean ± SD	Median (IQR)	Shapiro-Wilk p
Age (years)	31.2 ± 5.4	—	0.082
BMI (kg/m²)	26.4 ± 3.1	—	0.214
Lipoprotein (a) (nmol/L)	—	79.2 (48.2–90.1)	<0.001*

**Statistically significant non-normal distribution*

Among the 245 participants, 133 (54.3%) were aged 31–40 years and 112 (45.7%) aged 20–30 years. Gender distribution was near-equal: 122 (49.8%) male and 123 (50.2%) female. Overweight or obesity (BMI ≥25 kg/m²) was present in 147 (60.0%) participants. Urban residence was noted in 147 (60.0%). Smoking was reported by 82 (33.5%) individuals. Hypertension was the most prevalent comorbidity (75, 30.6%), followed by diabetes mellitus (48, 19.6%) and both together (24, 9.8%); 98 (40.0%) had no comorbidity. The relation to the index IHD patient was distributed

as: brother 58 (23.7%), sister 61 (24.9%), son 64 (26.1%), and daughter 62 (25.3%). Overall, raised Lp(a) (>125 nmol/L) was recorded in 98 (40.0%) participants (Table 2).

Table 2: Baseline Demographic and Clinical Characteristics with Raised Lipoprotein (a) among Study Participants (n=245)

Characteristic	Category	n (%)
Age (years)	20–30	112 (45.7)
	31–40	133 (54.3)
Gender	Male	122 (49.8)
	Female	123 (50.2)
BMI (kg/m²)	<25	98 (40.0)
	≥25	147 (60.0)
Residence	Urban	147 (60.0)
	Rural	98 (40.0)
Education	Matric or below	82 (33.5)
	Intermediate	81 (33.0)
	Graduate or above	82 (33.5)
Profession	Unemployed	73 (29.8)
	Employed	172 (70.2)
Socioeconomic Status	Low	110 (44.9)
	Middle	105 (42.9)
	High	30 (12.2)
Relation to Patient	Brother	58 (23.7)
	Sister	61 (24.9)
	Son	64 (26.1)
	Daughter	62 (25.3)
Smoking History	Yes	82 (33.5)
	No	163 (66.5)
Comorbidities	Nil	98 (40.0)
	Diabetes Mellitus	48 (19.6)
	Hypertension	75 (30.6)
	DM + HTN	24 (9.8)
Raised Lp(a) (>125 nmol/L)	Yes	98 (40.0)
	No	147 (60.0)

Stratified analysis demonstrated that raised Lp(a) was significantly more prevalent in the 31–40 year age group compared to the 20–30 year group (45.1% vs. 33.9%; p=0.042), in smokers versus non-smokers (48.8% vs. 35.6%; p=0.038), in participants with combined diabetes mellitus and hypertension versus those without this combination (58.3% vs. 38.0%; p=0.046), and in those with any comorbidity compared to those without (44.9% vs. 32.7%; p=0.036). Gender, BMI, residential status, educational level, socioeconomic status, occupation, and relation to the index patient did not show statistically significant associations with raised Lp(a). Detailed stratification results are presented in Table 3.

Table 3: Stratification of Raised Lipoprotein (a) by Demographic and Clinical Parameters (n=245)

Characteristic	Category	Raised Lp(a) Yes n(%)	Raised Lp(a) No n(%)	p-value
Age (years)	20–30 (n=112)	38 (33.9)	74 (66.1)	**0.042***
	31–40 (n=133)	60 (45.1)	73 (54.9)	
Gender	Male (n=122)	52 (42.6)	70 (57.4)	0.387
	Female (n=123)	46 (37.4)	77 (62.6)	
BMI (kg/m²)	<25 (n=98)	34 (34.7)	64 (65.3)	0.169
	≥25 (n=147)	64 (43.5)	83 (56.5)	
Residence	Urban (n=147)	62 (42.2)	85 (57.8)	0.361
	Rural (n=98)	36 (36.7)	62 (63.3)	
Education	Matric or below (n=82)	36 (43.9)	46 (56.1)	0.563
	Intermediate (n=81)	33 (40.7)	48 (59.3)	
	Graduate+ (n=82)	29 (35.4)	53 (64.6)	

Characteristic	Category	Raised Lp(a) Yes n(%)	Raised Lp(a) No n(%)	p-value
Profession	Unemployed (n=73)	27 (37.0)	46 (63.0)	0.509
	Employed (n=172)	71 (41.3)	101 (58.7)	
SES	Low (n=110)	48 (43.6)	62 (56.4)	0.412
	Middle (n=105)	40 (38.1)	65 (61.9)	
	High (n=30)	10 (33.3)	20 (66.7)	
Relation to Patient	Brother (n=58)	26 (44.8)	32 (55.2)	0.678
	Sister (n=61)	23 (37.7)	38 (62.3)	
	Son (n=64)	26 (40.6)	38 (59.4)	
	Daughter (n=62)	23 (37.1)	39 (62.9)	
Smoking	Yes (n=82)	40 (48.8)	42 (51.2)	**0.038***
	No (n=163)	58 (35.6)	105 (64.4)	
Diabetes Mellitus	Yes (n=48)	24 (50.0)	24 (50.0)	0.112
	No (n=197)	74 (37.6)	123 (62.4)	
Hypertension	Yes (n=75)	36 (48.0)	39 (52.0)	0.074
	No (n=170)	62 (36.5)	108 (63.5)	
DM + HTN (Combined)	Yes (n=24)	14 (58.3)	10 (41.7)	**0.046***
	No (n=221)	84 (38.0)	137 (62.0)	
Any Comorbidity	Yes (n=147)	66 (44.9)	81 (55.1)	**0.036***
(DM, HTN, or both)	No (n=98)	32 (32.7)	66 (67.3)	

*Chi-square test; *p <0.05 statistically significant; SES = Socioeconomic Status*

DISCUSSION

The frequency of raised Lp(a) observed in our study (40.0%) considerably exceeds the 20–30% prevalence reported across European and North American populations,(10) as well as the 6.1% documented by Mehta et al. in a population-based cohort.(9) This disparity is attributable to the deliberate selection of a genetically enriched cohort: all participants were first-degree relatives of patients with established IHD, a criterion that naturally concentrates individuals with atherogenic lipid phenotypes including elevated Lp(a). Given that Lp(a) levels are 70–90% heritable, the enrichment of family-history-positive subjects creates an inherent upward selection bias relative to unselected community samples.(1,11) Accordingly, the present findings are not intended to reflect population-level prevalence, but rather to characterize the burden of this risk factor within a clinically identifiable high-risk group amenable to screening.

Comparisons with South Asian literature lend contextual support. A systematic review of Indian populations reported Lp(a) elevation in 35–45% of subjects studied,(21) a range consistent with our findings. Enas et al. documented that individuals of South Asian heritage harbor higher Lp(a) levels than Western counterparts, potentially attributable to population-specific LPA gene polymorphisms.(22) Waldeyer et al., studying European cohorts, reported substantially lower prevalences of 20–25%.(10) These cross-ethnic differences underscore that threshold values and risk interpretations derived from Western populations may not translate directly to South Asian clinical contexts, strengthening the rationale for locally derived prevalence data.

Within the study sample, raised Lp(a) was significantly more frequent in participants aged 31–40 years compared to those aged 20–30 years. This age-related gradient is consistent with the broader literature on dyslipidemia trajectories: Kaneko et al. demonstrated progressive deterioration of lipid profiles in young adults, with accelerating cardiovascular risk beyond the fourth decade.(4) Dugani et al. similarly identified age at IHD onset as correlated with increasingly unfavorable metabolic profiles in younger women.(5) From a preventive standpoint, these findings suggest that the third decade of life represents a critical intervention window, prior to the consolidation of irreversible atherosclerotic changes.

Smoking was significantly associated with elevated Lp(a) in the present cohort. As a potent modifiable risk factor for IHD, smoking amplifies atherogenesis through oxidative modification of LDL, endothelial dysfunction, and induction of a proinflammatory state.(15) Aaseth et al. demonstrated that smoking compounds the cardiovascular impact of residual cholesterol and plasma triglycerides in young adults.(6) The cumulative interaction between genetically predetermined Lp(a) elevation and acquired smoking exposure in this high-risk group underscores the particular importance of smoking cessation interventions targeted at young individuals with a family history of IHD.

The combined burden of diabetes mellitus and hypertension, but not either condition individually, was significantly associated with raised Lp(a). When examined independently, neither hypertension (p=0.074) nor diabetes mellitus (p=0.112) reached statistical significance, suggesting that isolated comorbidities may be insufficient to substantially alter Lp(a) in this predominantly young cohort. This observation aligns with the concept that the cumulative metabolic dysregulation associated with concurrent insulin resistance and pressure overload may be required to potentiate lipid abnormalities beyond the individual contributions of each condition.(5,16) Taylor et al. demonstrated that co-existing modifiable risk factors additively amplify future cardiovascular risk.(7) The clinical implication is that young individuals with multiple concurrent comorbidities represent a particularly high-priority group for Lp(a) evaluation.

BMI did not demonstrate a significant association with raised Lp(a) in this study (p=0.169). This is consistent with the fundamentally genetic nature of Lp(a) regulation, which renders it relatively resistant to metabolic or nutritional

influences in contrast to LDL and triglycerides, which are substantially diet-sensitive.(12) Smith et al. described metabolically healthy obesity as a recognized phenotype in which individuals with elevated BMI maintain near-normal metabolic profiles;(17) however, Neeland et al. cautioned that this represents a transitional rather than a stable state.(18) The absence of a BMI–Lp(a) association reinforces that weight-based risk stratification alone would be an inadequate trigger for Lp(a) screening in this population.

The absence of significant associations between raised Lp(a) and gender, residential status, educational level, socioeconomic status, or relation to the index patient supports the view that genetic determinism overrides sociodemographic heterogeneity in this risk factor's expression. Mezhal et al. and Wahrenberg et al. independently confirmed that family history of cardiovascular disease predicts adverse lipid profiles and IHD recurrence irrespective of socioeconomic strata or educational attainment, lending further credence to this interpretation.(19,20)

Several limitations merit acknowledgment. The cross-sectional design precludes causal inference, and the single-center setting at PIMS Islamabad may limit generalizability to populations with differing demographic profiles or healthcare access. The absence of a comparator group without family history of IHD — a constraint of the primary study aim, which was to characterize frequency within a defined high-risk group rather than to establish relative risk prevents direct quantification of family history-attributable Lp(a) elevation. Additionally, data on lipid-lowering therapy, dietary patterns, and physical activity which could influence co-existing lipid parameters were not captured. Genetic characterization of LPA isoforms or other inherited lipid disorders was not undertaken due to resource constraints. Prospective multicenter studies incorporating comparator groups and longitudinal outcome data are warranted to delineate the absolute risk conferred by raised Lp(a) in this population.

CONCLUSION

Among young individuals with a first-degree family history of ischemic heart disease, elevated lipoprotein (a) is prevalent, affecting two in five individuals in this series. Raised Lp(a) is significantly associated with age above 30 years, active smoking, and the presence of comorbidities particularly the concurrent occurrence of diabetes mellitus and hypertension. These associations, combined with the largely genetic determination of Lp(a) levels, underscore the value of early targeted screening in this high-risk group rather than reliance on conventional lipid panels alone. Proactive management of modifiable co-risk factors particularly smoking cessation and rigorous comorbidity control should be prioritized from the third decade of life onwards in individuals with a family history of IHD.

Conflict Of Interest
None
Funding
None

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