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Association of Lipoprotein(A) with Angiographic Severity of Coronary Artery Disease.

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Abstract

Background: Lipoprotein(a) [Lp(a)] is a genetically determined lipoprotein with atherogenic, pro-inflammatory, and prothrombotic properties. Elevated Lp(a) has emerged as an important contributor to residual cardiovascular risk; however, its relationship with the anatomical severity and complexity of coronary artery disease (CAD) requires further evaluation. Objectives: To determine the association between serum Lp(a) concentration and angiographic severity of CAD and to evaluate whether elevated Lp(a) is independently associated with more complex coronary artery disease. ***Materials and Methods***: This prospective observational study was conducted in the Department of Cardiology, S.N. Medical College, Agra, Uttar Pradesh, from April 2025 to March 2026. A total of 120 adult patients undergoing clinically indicated coronary angiography were enrolled. Demographic characteristics, cardiovascular risk factors, lipid parameters, Lp(a) concentrations, number of diseased vessels, and SYNTAX scores were recorded. Lp(a) ≥50 mg/dL was considered elevated for categorical analysis. Associations between Lp(a) and angiographic severity were assessed using correlation and multivariable regression analyses. ***Results***: The mean age was 58.7 ± 10.4 years and 82 (68.3%) patients were male. Median Lp(a) concentration was 34 mg/dL (IQR 18–62), and 39 (32.5%) patients had Lp(a) ≥50 mg/dL. Median Lp(a) increased progressively from 18 mg/dL in patients without significant CAD to 26 mg/dL, 37 mg/dL, and 61 mg/dL in patients with single-, double-, and triple-vessel disease, respectively ($p<0.001$). Lp(a) demonstrated a significant positive correlation with SYNTAX score (Spearman $\rho=0.48$, $p<0.001$). Patients with elevated Lp(a) had a higher prevalence of triple-vessel disease (53.8% vs 19.8%, $p<0.001$) and SYNTAX score ≥23 (71.8% vs 35.8%, $p<0.001$). Elevated Lp(a) remained independently associated with SYNTAX score ≥23 after multivariable adjustment (adjusted OR 3.15; 95% CI 1.42–7.01; $p=0.005$). ***Conclusion***: Elevated Lp(a) was significantly associated with greater extent and angiographic complexity of CAD. Lp(a) measurement may provide additional information regarding coronary atherosclerotic burden beyond conventional cardiovascular risk factors.
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Keywords: Lipoprotein(a); coronary artery disease; coronary angiography; SYNTAX score; atherosclerosis; cardiovascular risk.

INTRODUCTION

Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide and is particularly important in South Asian populations, in whom atherosclerotic cardiovascular disease often develops at a younger age. Traditional cardiovascular risk factors such as hypertension, diabetes mellitus, smoking, dyslipidemia, obesity, and family history account for a major proportion of risk; however, substantial residual cardiovascular risk persists even when conventional risk factors are adequately treated.

Lipoprotein(a) [Lp(a)] has emerged as an important genetically determined cardiovascular risk factor. Structurally, Lp(a) consists of an LDL-like particle containing apolipoprotein B-100 covalently linked to apolipoprotein(a). Plasma Lp(a) concentrations are predominantly determined by the LPA gene and remain relatively stable throughout adult life. Unlike conventional lipid fractions, Lp(a) is only modestly influenced by lifestyle measures.

Lp(a) may promote atherosclerotic cardiovascular disease through several complementary mechanisms. The LDL-like component contributes cholesterol to the arterial wall, while oxidized phospholipids carried by Lp(a) promote endothelial dysfunction and vascular inflammation. In addition, structural homology between apolipoprotein(a) and plasminogen has provided a mechanistic basis for potential antifibrinolytic and prothrombotic effects. These properties make Lp(a)

biologically capable of influencing not only the occurrence of CAD but also the extent and complexity of coronary atherosclerosis.

Large epidemiological, genetic, and Mendelian-randomization studies support a causal relationship between elevated Lp(a) and atherosclerotic cardiovascular disease. Contemporary consensus statements recommend that Lp (a) be considered a cardiovascular risk modifier, especially in patients with premature cardiovascular disease, recurrent events despite optimal LDL-C reduction, familial hypercholesterolemia, or otherwise unexplained cardiovascular risk.

The relationship between Lp(a) and angiographically defined CAD severity has been evaluated using the number of diseased vessels, Gensini score, and SYNTAX score. The SYNTAX score provides an anatomical measure of coronary complexity by incorporating lesion location, bifurcation involvement, chronic total occlusion, calcification, tortuosity, and other lesion characteristics. Several studies have shown higher Lp(a) levels in multivessel and more complex CAD, although results have not been fully uniform across populations.

Data relating Lp(a) to angiographic disease severity remain comparatively limited in contemporary Indian clinical populations. Differences in genetic background, metabolic risk profile, and distribution of Lp(a) may affect the observed relationship. The present study was therefore undertaken to assess the association between serum Lp(a) concentration and angiographic severity of CAD in patients undergoing coronary angiography at a tertiary-care teaching hospital in North India.

Aim and Objectives

Aim: To evaluate the association between serum lipoprotein(a) concentration and angiographic severity of coronary artery disease.

Primary objective: To determine the correlation between serum Lp(a) concentration and angiographic CAD severity as assessed by the SYNTAX score.

Secondary objectives: To compare Lp(a) concentrations according to the number of significantly diseased coronary vessels; determine the proportion of patients with elevated Lp(a); compare clinical and biochemical characteristics between elevated and non-elevated Lp(a) groups; and evaluate whether elevated Lp(a) is independently associated with intermediate-to-high angiographic complexity after adjustment for conventional cardio vascular risk factors.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based prospective observational study conducted in the Department of Cardiology, S.N. Medical College, Agra, Uttar Pradesh, India, over a 12-month period from April 2025 to March 2026.

Study population

Adult patients undergoing clinically indicated invasive coronary angiography during the study period were screened for eligibility. Eligible consecutive patients were recruited until the required sample size was achieved.

Sample size

A total of 120 patients were included. The sample size was selected to provide approximately 80% power to detect a small-to-moderate correlation between Lp(a) and angiographic severity at a two-sided alpha level of 0.05, while allowing for incomplete or non-evaluable data.

Inclusion criteria

Patients aged ≥18 years; patients undergoing coronary angiography for a clinically established indication; suspected or established CAD; availability of Lp(a) estimation and angiographic assessment; and written informed consent.

Exclusion criteria

Previous coronary artery bypass graft surgery; coronary anatomy unsuitable for reliable SYNTAX scoring; severe valvular heart disease as the primary cardiac diagnosis; nephrotic syndrome; dialysis-dependent renal failure or severe chronic kidney disease; severe hepatic dysfunction; active systemic inflammatory or infectious disease; active malignancy; pregnancy; incomplete clinical, biochemical, or angiographic data; or refusal to participate.

Data collection

Demographic characteristics, cardiovascular risk factors, clinical presentation, blood pressure, body mass index, routine biochemical parameters, lipid profile, Lp(a), left ventricular ejection fraction, number of significantly diseased coronary vessels, and SYNTAX score were recorded using a structured case-record form.

Definitions
Hypertension was defined by a previous diagnosis, ongoing antihypertensive therapy, or blood pressure fulfilling accepted diagnostic criteria. Diabetes mellitus was defined by a previous diagnosis, current glucose-lowering treatment, or standard biochemical criteria. Current smoking included active cigarette, bidi, or other smoked tobacco use. Family history of premature CAD was defined as CAD in a first-degree male relative before 55 years of age or female relative before 65 years of age.

Biochemical assessment
Venous blood samples were obtained according to institutional laboratory protocol. Total cholesterol, triglycerides, HDL-C, LDL-C, serum creatinine, and Lp(a) were measured using routine laboratory methods. Lp(a) was analyzed primarily as a continuous variable. For secondary categorical analyses, Lp(a) ≥50 mg/dL was considered elevated and <50 mg/dL was considered lower. Where Lp(a) is reported in nmol/L, direct unit conversion should not be performed using a single fixed conversion factor.

Coronary angiography
Coronary angiography was performed through radial or femoral arterial access according to operator preference and clinical circumstances. Multiple orthogonal projections were obtained to visualize the coronary circulation. Significant obstructive CAD was defined as ≥70% luminal stenosis in a major epicardial coronary artery or major branch, while ≥50% stenosis was used for left-main coronary disease. Patients were categorized as having no significant obstructive CAD, single-vessel disease, double-vessel disease, or triple-vessel disease.

Assessment of angiographic severity
The anatomical complexity of CAD was evaluated using the SYNTAX score. Lesion characteristics required by the standard scoring algorithm were assessed. SYNTAX scores were categorized as low (≤22), intermediate (23–32), and high (≥33). For selected analyses, intermediate and high scores were combined as SYNTAX score ≥23.

Study outcomes
The primary outcome was the association between continuous serum Lp(a) concentration and continuous SYNTAX score. Secondary outcomes were the relationship of Lp(a) with number of diseased vessels, differences in Lp(a) across SYNTAX categories, and the independent association between elevated Lp(a) and SYNTAX score ≥23.

Statistical analysis
Data were analyzed using standard statistical software. Normally distributed continuous variables were expressed as mean ± standard deviation; skewed variables were expressed as median with interquartile range. Categorical variables were expressed as frequency and percentage. Independent-samples t test or Mann–Whitney U test was used for two-group comparisons, while ANOVA or Kruskal–Wallis test was used for three or more groups. Categorical variables were compared using chi-square or Fisher exact test. The association between Lp(a) and SYNTAX score was examined using Spearman correlation. Multivariable logistic regression was used to identify independent predictors of SYNTAX score ≥23. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A two-sided p value <0.05 was considered statistically significant.

Ethical considerations
The study was conducted after approval from the Institutional Ethics Committee of S.N. Medical College, Agra. Written informed consent was obtained from all participants. Confidentiality was maintained and the study was conducted in accordance with the principles of the Declaration of Helsinki.

RESULTS
A total of 120 patients undergoing coronary angiography were included. The mean age of the study population was 58.7 ± 10.4 years (range 34–79 years). Eighty-two (68.3%) were male and 38 (31.7%) were female. Hypertension was present in 70 (58.3%) patients, diabetes mellitus in 53 (44.2%), current smoking in 41 (34.2%), and a family history of premature CAD in 18 (15.0%). The mean LDL-C concentration was 112.6 ± 31.8 mg/dL. Median Lp(a) concentration was 34 mg/dL (IQR 18–62), and 39 (32.5%) patients had Lp(a) ≥50 mg/dL.

Table 1. Baseline clinical and biochemical characteristics of the study population

Variable	Overall (n=120)
Age, years	58.7 ± 10.4
Male sex, n (%)	82 (68.3)
Female sex, n (%)	38 (31.7)
BMI, kg/m²	25.8 ± 3.7
Hypertension, n (%)	70 (58.3)
Diabetes mellitus, n (%)	53 (44.2)
Current smoking, n (%)	41 (34.2)
Family history of premature CAD, n (%)	18 (15.0)
Total cholesterol, mg/dL	184.7 ± 39.6
LDL-C, mg/dL	112.6 ± 31.8
HDL-C, mg/dL	41.8 ± 9.6
Triglycerides, mg/dL	151 (118–198)
Lp(a), mg/dL	34 (18–62)
Lp(a) ≥50 mg/dL, n (%)	39 (32.5)
Serum creatinine, mg/dL	1.02 ± 0.24
LVEF, %	52.6 ± 8.7

Values are expressed as mean ± SD, median (IQR), or n (%), as appropriate.

Coronary angiography demonstrated no significant obstructive CAD in 10 (8.3%) patients, single-vessel disease in 35 (29.2%), double-vessel disease in 38 (31.7%), and triple-vessel disease in 37 (30.8%). Significant left-main coronary disease was present in 8 (6.7%) patients. The median SYNTAX score was 19 (IQR 11–29); 63 (52.5%) patients had a low score, 38 (31.7%) had an intermediate score, and 19 (15.8%) had a high score.

Table 2. Angiographic characteristics of study participants

Angiographic variable	n (%) / Value
No significant obstructive CAD	10 (8.3)
Single-vessel disease	35 (29.2)
Double-vessel disease	38 (31.7)
Triple-vessel disease	37 (30.8)
Left-main disease	8 (6.7)
SYNTAX score	19 (11–29)
SYNTAX ≤22	63 (52.5)
SYNTAX 23–32	38 (31.7)
SYNTAX ≥33	19 (15.8)
SYNTAX ≥23	57 (47.5)

Lp(a) concentrations increased progressively with the extent of coronary disease. Median Lp (a) was 18 mg/dL (IQR 11–27) in patients without significant CAD, 26 mg/dL (IQR 15–39) in single-vessel disease, 37 mg/dL (IQR 21–59) in double-vessel disease, and 61 mg/dL (IQR 39–82) in triple-vessel disease. The difference across groups was statistically significant (Kruskal–Wallis $p<0.001$).

Table 3. Association of Lp(a) with extent of coronary artery disease

CAD extent	n	Lp(a), mg/dL, median (IQR)	p-value
No significant CAD	10	18 (11–27)	
Single-vessel disease	35	26 (15–39)	
Double-vessel disease	38	37 (21–59)	
Triple-vessel disease	37	61 (39–82)	<0.001

Median Lp(a) also increased significantly across SYNTAX categories. Patients with low SYNTAX scores had a median Lp(a) of 24 mg/dL (IQR 14–39), compared with 48 mg/dL (IQR 29–70) in the intermediate group and 68 mg/dL (IQR 47–91) in the high group ($p<0.001$). Spearman correlation demonstrated a moderate positive relationship between Lp(a) and continuous SYNTAX score ($\rho=0.48$, $p<0.001$).

Table 4. Lp(a) according to angiographic complexity

SYNTAX category	n	Lp(a), mg/dL, median (IQR)	p-value
Low (≤22)	63	24 (14–39)	
Intermediate (23–32)	38	48 (29–70)	
High (≥33)	19	68 (47–91)	<0.001

Patients were divided into Lp(a) <50 mg/dL (n=81) and Lp(a) ≥50 mg/dL (n=39). Triple-vessel disease was substantially more frequent among patients with elevated Lp(a) (53.8% vs 19.8%, p<0.001). Similarly, SYNTAX score ≥23 occurred in 71.8% of patients with elevated Lp(a), compared with 35.8% in the lower Lp(a) group (p<0.001).

Table 5. Comparison of patients according to Lp(a) concentration

Variable	Lp(a) <50 mg/dL (n=81)	Lp(a) ≥50 mg/dL (n=39)	p-value
Age, years	57.9 ± 10.6	60.3 ± 9.8	0.238
Male sex, n (%)	54 (66.7)	28 (71.8)	0.571
Hypertension, n (%)	43 (53.1)	27 (69.2)	0.094
Diabetes mellitus, n (%)	31 (38.3)	22 (56.4)	0.061
Current smoking, n (%)	25 (30.9)	16 (41.0)	0.273
LDL-C, mg/dL	109.8 ± 30.6	118.4 ± 33.8	0.164
Triple-vessel disease, n (%)	16 (19.8)	21 (53.8)	<0.001
SYNTAX score	15 (9–23)	28 (20–36)	<0.001
SYNTAX score ≥23, n (%)	29 (35.8)	28 (71.8)	<0.001

On multivariable logistic regression analysis, elevated Lp(a) remained independently associated with intermediate-to-high angiographic complexity after adjustment for age, sex, hypertension, diabetes mellitus, smoking, and LDL-C concentration. Lp(a) ≥50 mg/dL was associated with approximately three-fold higher odds of a SYNTAX score ≥23 (adjusted OR 3.15; 95% CI 1.42–7.01; p=0.005). Diabetes mellitus was also independently associated with higher angiographic complexity (adjusted OR 2.18; 95% CI 1.01–4.72; p=0.047).

Table 6. Multivariable logistic regression for factors associated with SYNTAX score ≥23

Variable	Adjusted OR	95% CI	p-value
Age, per year	1.03	0.99–1.07	0.118
Male sex	1.28	0.56–2.94	0.556
Hypertension	1.61	0.72–3.61	0.246
Diabetes mellitus	2.18	1.01–4.72	0.047
Current smoking	1.52	0.67–3.46	0.316
LDL-C, per 10 mg/dL increase	1.09	0.98–1.21	0.112
Lp(a) ≥50 mg/dL	3.15	1.42–7.01	0.005

DISCUSSION

The present study demonstrated a significant association between serum Lp(a) concentration and both the extent and anatomical complexity of coronary artery disease. Lp(a) concentrations increased progressively from patients without significant obstructive CAD to those with single-, double-, and triple-vessel disease. In addition, Lp(a) showed a moderate positive correlation with the SYNTAX score, supporting a relationship with increasing coronary anatomical complexity. Approximately one-third (32.5%) of the study population had Lp(a) ≥50 mg/dL. The prevalence of triple-vessel disease was 53.8% in the elevated Lp(a) group compared with 19.8% among patients with lower Lp(a). Likewise, a SYNTAX score ≥23 was observed in 71.8% of patients with elevated Lp(a) compared with 35.8% of those with lower concentrations. These findings suggest that elevated Lp(a) may identify a subgroup with a greater burden of diffuse and complex coronary atherosclerosis.

The biological relationship between Lp(a) and CAD severity is plausible. Lp(a) contributes cholesterol to the arterial wall and is an important carrier of oxidized phospholipids, which promote endothelial activation, inflammation, and foam-cell formation. Apolipoprotein(a) also shares structural similarity with plasminogen, providing a possible link with impaired fibrinolysis and thrombosis. The combination of atherogenic, inflammatory, and prothrombotic properties may therefore contribute to progression of coronary lesions.

Our observations are consistent with earlier angiographic studies. Ashfaq et al. reported higher Lp(a) concentrations among patients with CAD and a progressive increase in Lp(a) with the number of diseased vessels in a North Indian population. They also demonstrated a significant relationship between Lp(a) and SYNTAX score. Other studies have similarly described associations between Lp(a), multivessel disease, Gensini score, and coronary lesion complexity.

The finding that median Lp(a) increased from 18 mg/dL in patients without significant obstructive CAD to 61 mg/dL in those with triple-vessel disease is particularly noteworthy. The graded pattern argues against an association limited only to the presence or absence of disease and instead supports a relationship with the overall coronary atherosclerotic burden. The SYNTAX score provides information beyond simple vessel counting because it incorporates lesion location and morphological complexity, including bifurcation disease, total occlusions, severe tortuosity, calcification, and diffuse

disease. The positive correlation between Lp(a) and SYNTAX score in the present study therefore suggests an association with both the extent and complexity of coronary atherosclerosis.

On multivariable analysis, Lp(a) ≥50 mg/dL remained independently associated with a SYNTAX score ≥23 after adjustment for conventional cardiovascular risk factors, with an adjusted OR of 3.15. This is clinically relevant because Lp(a) concentration is largely genetically determined and may remain elevated despite otherwise acceptable conventional lipid parameters. Consequently, Lp(a) may help explain residual or disproportionate coronary risk in some patients. Diabetes mellitus was also independently associated with greater angiographic complexity. This is consistent with the established tendency of diabetes to produce diffuse, multivessel, and anatomically complex coronary disease. The coexistence of diabetes and elevated Lp(a) may therefore identify patients who warrant particularly intensive risk-factor management.

Current clinical recommendations increasingly recognize Lp(a) as a cardiovascular risk-enhancing factor. Although specific Lp(a)-lowering therapies are under active investigation, the immediate clinical implication of detecting elevated Lp(a) is optimization of all modifiable risk factors, particularly LDL-C, blood pressure, glycemic control, smoking cessation, weight management, and adherence to evidence-based secondary prevention.

The present study adds data from a North Indian tertiary-care population and supports incorporation of Lp(a) assessment into comprehensive risk evaluation, especially in patients with premature CAD, extensive multivessel disease, recurrent events, a strong family history, or apparently severe disease despite acceptable conventional lipid levels.

Strengths

- Direct invasive coronary angiographic assessment of CAD.
- Use of the validated SYNTAX scoring system to quantify anatomical complexity.
- Evaluation of Lp(a) both continuously and using a clinically relevant categorical threshold.
- Multivariable adjustment for important conventional cardiovascular risk factors.
- Study population drawn from a North Indian tertiary-care cardiology setting.

Limitations

This study has several limitations. It was a single-centre observational study with a relatively modest sample size, which may limit generalizability. The cross-sectional relationship between Lp(a) and angiographic severity cannot establish temporal causality. Conventional coronary angiography assesses luminal narrowing rather than total plaque volume or plaque composition. Lp(a) was assessed at a single time point, and assay-related variability may influence mass-based Lp(a) measurements. Medication use, particularly lipid-lowering therapy, may affect conventional lipid parameters. Finally, long-term cardiovascular outcomes were not assessed.

CONCLUSION

Elevated serum lipoprotein(a) was significantly associated with greater extent and anatomical complexity of coronary artery disease in patients undergoing coronary angiography. Lp(a) concentrations increased progressively with the number of diseased coronary vessels and demonstrated a significant positive correlation with SYNTAX score. Patients with Lp(a) ≥50 mg/dL had substantially higher prevalences of triple-vessel disease and intermediate-to-high SYNTAX scores. After adjustment for conventional cardiovascular risk factors, elevated Lp(a) remained independently associated with greater angiographic complexity. Lp(a) measurement may therefore provide additional information regarding coronary atherosclerotic burden and may help identify patients who warrant especially intensive management of modifiable cardiovascular risk factors.

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