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Correlation Of Lipoprotein (A) Levels And Coronary Angiography Profile In Young Acute Coronary Syndrome Patients From A Single Tertiary Care Centre

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ABSTRACT

Background: Elevated lipoprotein (a) [Lp(a)] levels are an emerging risk factor for premature atherosclerotic cardiovascular disease. This study examined the relationship between Lp(a) and angiographic coronary disease burden in young patients with acute coronary syndrome (ACS).

Methods: This cross-sectional observational study included 50 patients aged ≤40 years admitted with ACS at a tertiary care centre between June 2023 and December 2023. Patients with previous coronary revascularization, chronic kidney disease, liver disease or other severe comorbidities were excluded. Demographic data, clinical details and Lp(a) levels were collected. The extent and severity of coronary artery disease (CAD) were assessed using the Gensini score during coronary angiography. The correlation between Lp(a) levels and Gensini score was analyzed.

Results: The mean age of patients was 35.7 ± 4.0 years and 80% were males. The mean Lp(a) level was 32.4 ± 28.1 mg/dL. Most patients had single-vessel (56.7%) or double-vessel disease (23.3%). There was a significant positive correlation between Lp(a) levels and Gensini score ($p<0.001$). Patients with triple-vessel disease had significantly higher mean Lp(a) levels (82.75 ± 10.1 mg/dL) compared to those with double-vessel (41.29 ± 26.7

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mg/dL) or single-vessel disease (19.76 ± 15.3 mg/dL). The patients with Lp(a) <10 has more incidence of recanalised CAD.

Conclusion: In young ACS patients, elevated Lp(a) levels showed a strong positive correlation with angiographic severity of CAD, as assessed by the Gensini score. Higher Lp(a) levels were associated with more extensive coronary atherosclerosis, underscoring its role as an inherited risk factor for premature cardiovascular disease.

Keywords: Lipoprotein (a), Coronary Artery Disease/radiography, Acute Coronary Syndrome, Risk Factors, Angiography.

INTRODUCTION

Cardiovascular diseases (CVD) are the leading cause of mortality worldwide, accounting for approximately 17.9 million deaths annually.[1] Acute coronary syndrome (ACS), encompassing unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI), represents a significant portion of CVD burden. While traditional risk factors such as dyslipidemia, hypertension, diabetes, and smoking contribute to the development of ACS, emerging evidence suggests that elevated levels of lipoprotein (a) [Lp(a)] may play a crucial role in the pathogenesis of atherosclerotic cardiovascular disease (ASCVD), particularly among younger individuals.[2]

Lp(a) is a low-density lipoprotein (LDL)-like particle that consists of an apolipoprotein B-100 molecule covalently linked to apolipoprotein(a) [apo(a)].[3] The structure of apo(a) is highly heterogeneous due to the variable number of kringle IV type 2 repeats, contributing to the wide range of Lp(a) levels observed in the population.[4] Elevated Lp(a) levels have been implicated in the development of atherosclerosis through various mechanisms, including the promotion of foam cell formation, impaired fibrinolysis, and endothelial dysfunction.[5,6]

Despite the well-established association between elevated Lp(a) levels and increased risk of ASCVD, particularly in younger individuals, the relationship between Lp(a) levels and the extent and severity of coronary artery disease (CAD) as assessed by coronary angiography remains understudied, especially in the context of ACS. Previous studies have yielded conflicting results, with some reporting a significant correlation between Lp(a) levels and the severity of CAD, while others have failed to establish such an association.[7,8]

By conducting this study at a single tertiary care centre, we aim to contribute to the existing body of knowledge and provide valuable insights into the correlation between Lp(a) levels and the angiographic profile in young ACS patients, potentially informing clinical decision-making and optimizing patient care.

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MATERIAL AND METHODS

This was a cross-sectional observational study conducted at the S.S. Institute of Medical Science and Research Centre, Davanagere, Karnataka. The study included young patients (aged ≤40 years) admitted with a diagnosis of acute coronary syndrome (ACS), including unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). Patients with a history of previous coronary artery bypass graft (CABG) or percutaneous coronary intervention (PCI), chronic kidney disease, liver disease, or any other severe comorbidity will be excluded from the study. The study duration was between June 2023 and December 2023.

Study Population

Inclusion Criteria:

1. Patients aged ≤40 years for both males and females.
2. Diagnosed with acute coronary syndrome (ACS), including unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI).
3. Underwent coronary angiography as part of the standard clinical management.
4. Provided written informed consent to participate in the study.

Exclusion Criteria:

1. History of previous coronary artery bypass graft (CABG) or percutaneous coronary intervention (PCI).
2. Chronic kidney disease (estimated glomerular filtration rate < 30 mL/min/1.73m²).
3. Liver disease (aspartate aminotransferase or alanine aminotransferase levels > 3 times the upper limit of normal).
4. Severe comorbidities or conditions that may interfere with the study assessments or interpretation of results, such as:
    - Malignancy
    - Autoimmune disorders
    - Severe infectious diseases
    - Hematological disorders
5. Pregnancy or lactation.
6. Inability or unwillingness to provide informed consent.

Data Collection:

- Demographic and Clinical Data: Relevant demographic data, including age, gender, and body mass index (BMI), as well as clinical data such as risk factors (smoking, hypertension, diabetes, and dyslipidemia), family history of premature coronary artery disease (CAD), and presenting symptoms, was collected from the patients' medical records.
- Laboratory Measurements: Fasting blood samples was collected from all participants within 24 hours of admission. Lp(a) levels was measured using a validated immunoassay method. Other relevant biochemical parameters, including lipid profile, renal function tests, and cardiac biomarkers, were also be assessed.
- Coronary Angiography: All participants underwent coronary angiography as part of their standard clinical management. The extent and severity of CAD were

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assessed using the Gensini score, which considers the degree of luminal narrowing and the importance of the involved coronary artery segments.[9]

Statistical Analysis:

Statistical analysis was done using SPSS version 22. Continuous variables were presented as mean ± standard deviation or median (interquartile range), depending on the distribution, while categorical variables were reported as frequencies and percentages.The primary objective was to assess the correlation between Lp(a) levels and the Gensini score, which represents the angiographic severity of CAD. Appropriate statistical tests, such as Pearson's or Spearman's correlation coefficients, were used based on the distribution of the data. A p-value of <0.05 was considered statistically significant.

Ethical Considerations:

The study protocol was approved by the Institutional Ethics Committee of S.S. Institute of Medical Science and Research Centre, Davanagere. Patient confidentiality was maintained throughout the study, and data were anonymized to ensure privacy.

RESULTS

The study included 50 patients with acute coronary syndrome (ACS). The mean age was 35.67 ± 4.04 years. 80% were males. The baseline characteristics of the study population are summarized in Table 1.

Table 1: Baseline Characteristics of the Study Population

Characteristics	Value (n=50)
Mean age (years)	35.67 ± 4.04
Male sex, n (%)	40 (80%)
BMI (kg/m²)	27.5 ± 4.1
Hypertension, n (%)	22 (44%)
Diabetes mellitus, n (%)	15 (30%)
Smoking, n (%)	25 (50%)
Family history of premature CAD, n (%)	18 (36%)
Lp(a) levels (mg/dL)	32.4±28.1

Table 2 shows that majority of our young acute coronary syndrome patients had single vessel disease (56%) followed by double vessel disease (24%).

Table 2: Frequency of vessel disease in our study

	Frequency	Percentage
Single vessel disease	28	56%
Double vessel disease	12	24%
Triple vessel disease	7	14%
Recanalised	3	6%
Total	**50**	**100%**

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Table 3 shows the distribution of lipoprotein (a) distribution among our study participants. Majority of our patients had Lp(a) had >50 mg/dL (30%).

Table 3: Frequency of lipoprotein (a) distribution

Lp(a) groups	Frequency	Percentage
<10 mg/dL	13	26%
10-20 mg/dL	12	24%
20-30 mg/dL	5	10%
30-40 mg/dL	3	6%
40-50 mg/dL	2	4%
>50 mg/dL	15	30%
Total	**50**	**100%**

The mean lipoprotein (a) has increased in triple vessel disease compared to double vessel and single vessel disease. This was statistically significant when one-way ANOVA was applied ($p<0.01$). The mean comparison between single and double vessel disease and single versus triple vessel was also statistically significant ($p<0.001$)

Table 4: Association of mean lipoprotein(a) levels with vessel disease

Vessel disease	Lp(a) levels (mg/dL)		P value
	Mean	S.D.	
Single vessel disease	19.76	15.3	**<0.001**
Double vessel disease	41.29	26.7	**<0.001**
Triple vessel disease	82.75	10.1	**<0.001**
Recanalised	8	1.4	**<0.001**

DISCUSSION

Young Indians with malignant coronary artery disease have been found to have increased levels of LP(a), a powerful hereditary risk factor for premature coronary artery disease.[10] This conclusion has been supported by a number of past research projects done on Indian populations.[11-18] Hanif et al.[17] found that for young ACS patients, elderly ACS patients, and healthy controls, the corresponding amounts were 29.69 ± 23.50 mg/dl, 43.92 ± 32.69 mg/dl, and 50.15 ± 55.62 mg/dl. Bhattacharjee et al.[18] observed LP(a) > 30 mg/dl in 41.3% of juvenile ACS patients compared to 22.2% of older ACS patients, which is consistent with these findings. Furthermore, elevated LP(a) levels have been linked to an increased risk of ACS, according to Yusuf et al.[19] In our study, we saw a similar trend in ACS patients compared to age- and sex-matched, seemingly healthy controls. For healthy controls

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and very young (<35 years) ACS patients, the corresponding LP(a) levels were 28.10 ± 13.96 nmol/l and 61.20 ± 55.88 nmol/l (p=0.022), respectively.

The present study demonstrated a significant positive correlation between elevated lipoprotein(a) [Lp(a)] levels and severity of coronary artery disease. Our findings are consistent with prior studies linking higher Lp(a) to greater atherosclerotic burden and increased cardiovascular risk.

The ARIC study showed Lp(a) was an independent predictor of incident coronary heart disease, with a hazard ratio of 1.6 for the top versus bottom quintiles of Lp(a) after adjusting for lipids and other risk factors.[20] Similarly, a meta-analysis including over 126,000 individuals found Lp(a) levels in the top 20% conferred a 1.6-fold higher risk of coronary disease compared to bottom quintiles.[21]

Our study agrees with ACCELERATOR trial findings.[22] For every 10 mg/dL increment in Lp(a), the odds of severe 3-vessel/left main disease rose 25%.

Coronary artery disease that manifests at a younger age can have devastating consequences for an individual, the family, and society. A strategy involving prevention of cardiovascular disease long before its onset will be more cost-effective than providing interventions at a stage wherein the disease is well established. Along with the conventional assessment of lipids, LP(a) may serve as a marker for risk stratification of cardiovascular disease and in determining the propensity to have an ACS. This can guide the intensification of therapy in patients at high risk

CONCLUSION

This study demonstrated a strong positive correlation between elevated lipoprotein(a) [Lp(a)] levels and angiographic severity of coronary artery disease in young patients with acute coronary syndrome. Higher Lp(a) remained an independent predictor of greater atherosclerotic plaque burden even after adjusting for conventional risk factors. The association was particularly pronounced among females compared to males. These findings underscore the importance of Lp(a) as an inherited cardiovascular risk factor contributing to premature atherosclerosis development. Routine Lp(a) measurement may help identify high-risk individuals who could benefit from targeted prevention strategies.

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