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Internal Medicine

ASSOCIATION OF SERUM LIPOPROTEIN(A) LEVELS WITH ISCHEMIC STROKE AND ITS SEVERITY: A HOSPITAL-BASED OBSERVATIONAL STUDY

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ABSTRACT **Background:** Ischemic stroke is a leading cause of death and disability worldwide, with lipoprotein(a) [Lp(a)] emerging as an important, genetically determined biomarker. (1)This study aimed to assess the association between Lp(a) levels and ischemic stroke, focusing on stroke severity. **Methods:** A hospital-based observational study was conducted on 110 acute ischemic stroke patients at ACPM Medical College, Dhule. Serum Lp(a) levels were measured within 24 hours of admission, and stroke severity was assessed using NIHSS. **Results:** Elevated Lp(a) (≥30 mg/dL) was present in 80% of patients and significantly correlated with higher NIHSS scores (p=0.028). Dyslipidemia was significantly more common in the elevated Lp(a) group (p=0.014). Other vascular risk factors showed no statistically significant association with Lp(a) levels. **Conclusion:** Elevated Lp(a) is common among ischemic stroke patients and correlates with greater severity. Routine measurement may aid in risk stratification and prevention strategies.

KEYWORDS : Ischemic stroke, Lipoprotein(a), NIHSS, Biomarkers, Atherothrombosis.

INTRODUCTION

Ischemic stroke accounts for approximately 80–85% of all stroke cases and remains one of the leading causes of mortality and long-term disability globally. In India, stroke incidence is increasing, with younger age of onset compared to Western populations. While established risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking, and atrial fibrillation are well recognized, emerging biomarkers have gained attention for their potential role in risk stratification and prognosis.

Lipoprotein(a) [Lp(a)] is a low-density lipoprotein (LDL)-like particle with an additional apolipoprotein(a) component covalently linked to apolipoprotein B-100. It is largely genetically determined (2) and minimally influenced by lifestyle or diet. Elevated Lp(a) promotes atherosclerosis by enhancing endothelial dysfunction, oxidative stress, and thrombosis, and has been implicated in coronary artery disease and ischemic stroke. The structural similarity of apo(a) to plasminogen impairs fibrinolysis, increasing thrombotic risk.

This study focuses on evaluating the relationship between serum Lp(a) levels and ischemic stroke, with emphasis on stroke severity measured by NIHSS.

MATERIALS AND METHODS

A prospective observational study was conducted at the Department of Medicine, ACPM Medical College, Dhule, from January 2023 to December 2024. A total of 110 patients with acute ischemic stroke confirmed by CT/MRI brain were included. Exclusion criteria were hemorrhagic stroke, chronic kidney disease, chronic liver disease, malignancy, and recent myocardial infarction.

Detailed demographic and clinical data were recorded, including vascular risk factors such as hypertension, diabetes, dyslipidemia, smoking, and alcohol consumption. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) at admission. Serum Lp(a) was measured within 24 hours using an immunoturbidimetric assay. Patients were categorized as normal (<30 mg/dL) or elevated (≥30 mg/dL) Lp(a).

Statistical Analysis:

Data were analyzed using SPSS v26. Chi-square test and Student's t-test were used, with $p<0.05$ considered statistically significant.

RESULTS

Table 1: Distribution Of Lp(a) Levels In Ischemic Stroke Patients

Lp(a) Level	No. of Patients	Percentage (%)
<30 mg/dL	22	20.0
≥30 mg/dL	88	80.0

In this study, elevated Lp(a) levels (≥30 mg/dL) were present in **80%** of ischemic stroke patients, indicating a high prevalence in the cohort.

Table 2: Relation of Lp(a) with Stroke Severity (NIHSS Score)

NIHSS Category	<30 mg/dL (n=22)	≥30 mg/dL (n=88)	p-value
Mild (0–4)	14 (63.6%)	28 (31.8%)	0.028
Moderate (5–15)	6 (27.3%)	36 (40.9%)	
Severe (>15)	2 (9.1%)	24 (27.3%)	

Stroke severity was significantly higher in patients with elevated Lp(a) (≥30 mg/dL), with severe NIHSS scores (>15) observed in **27.3%** compared to **9.1%** in the normal Lp(a) group ($p = 0.028$).

Table 3: Relation of Lp(a) With Major Vascular Risk Factors

Risk Factor	<30 mg/dL (n=22)	≥30 mg/dL (n=88)	p-value
Hypertension	10 (45.5%)	54 (61.4%)	0.178
Diabetes Mellitus	6 (27.3%)	38 (43.2%)	0.174
Dyslipidemia	7 (31.8%)	54 (61.4%)	0.014
Smoking	4 (18.2%)	28 (31.8%)	0.223
Alcohol	3 (13.6%)	22 (25.0%)	0.254

Among vascular risk factors, **dyslipidemia** showed a significant association with elevated Lp(a) levels ($p = 0.014$), while hypertension, diabetes, smoking, and alcohol use were not statistically significant.

DISCUSSION

In this study, elevated lipoprotein(a) [Lp(a)] levels were found in 80% of ischemic stroke patients, a proportion significantly higher than reported in some Western cohorts but consistent with previous Indian studies (3). The high prevalence of elevated Lp(a) in our cohort may reflect the underlying genetic predisposition in the South Asian population, which is known to have higher Lp(a) concentrations compared to other ethnic groups.

The association between elevated Lp(a) and stroke severity, as measured by the NIHSS score, was statistically significant ($p = 0.028$) (4). Patients with higher Lp(a) levels had more severe neurological deficits at presentation. This finding is consistent with studies by Kamstrup et al. and Virani et al., which reported that Lp(a) is linked not only to stroke occurrence but also to poorer functional outcomes.

Mechanistically, Lp(a) contributes to atherogenesis through multiple pathways: enhancement of endothelial dysfunction, upregulation of adhesion molecules, promotion of smooth muscle proliferation, and inhibition of fibrinolysis via its structural similarity to plasminogen (5). Furthermore, the presence of oxidized phospholipids (6) on Lp(a) particles amplifies vascular inflammation and plaque instability.

Although dyslipidemia was significantly associated with elevated Lp(a) in this study ($p = 0.014$), other vascular risk factors such as hypertension, diabetes, smoking, and alcohol use did not show statistically significant associations. This could be due to overlapping risk factor profiles and sample size limitations.

Our findings support the growing evidence that elevated Lp(a) is an independent risk factor for ischemic stroke, particularly in large artery atherosclerosis. Routine measurement of Lp(a) in patients at risk for cerebrovascular events could improve risk stratification and inform preventive strategies.

CONCLUSION

Elevated Lp(a) levels are highly prevalent in ischemic stroke patients and are significantly associated with greater stroke severity. Given its genetic determination and limited response to lifestyle modification, Lp(a) represents a valuable biomarker (7) for identifying high-risk individuals. Incorporating Lp(a) screening into routine clinical evaluation for stroke patients may facilitate early detection and implementation of targeted preventive measures. Future research should explore Lp(a)-lowering interventions and their impact on stroke recurrence and outcomes.

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