International Journal of Health Sciences and Research
[Indexed, Peer-Reviewed Journal]
Volume 16; Issue: 4; April 2026
Website: www.ijhsr.org
Original Research Article *ISSN: 2249-9571*

Serum Lipoprotein (a) Levels in Patients with Ischemic Stroke

Priya Theresa Job[1], T. Sobhakumari[2], Thomas Iype[3]

[1,2]Department of Physiology, [3]Department of Neurology,
Govt. Medical College, Thiruvananthapuram, KUHS University, Kottayam, India.

Corresponding Author: Dr. Priya Theresa Job priyatheresajob@gmail.com

DOI: https://doi.org/10.52403/ijhsr.20260402

ABSTRACT

Introduction: A great number of studies have suggested that high levels of Lipoprotein (a) Lp (a) [Lp (a)] predispose the individuals to atherosclerotic vascular diseases specifically, ischemic stroke and coronary artery disease. But only a few studies have investigated the distribution of serum Lp (a) levels in the Indian population so far. In the present study, an attempt has been made to find out the levels of serum Lp (a) in a group of newly diagnosed ischemic stroke patients.
Objective: To study the serum levels of Lipoprotein (a) in patients who developed ischemic stroke in comparison with a group of people who do not have the clinical outcome.
Methods: The levels of serum Lp (a) were measured in forty newly diagnosed ischemic stroke patients, aged 45-60 yrs, who were admitted to the Department of Neurology, Government Medical College Thiruvananthapuram.
Results: The mean ± SD of Lp (a) in stroke patients was 24.76 ±10.74 whereas in person without stroke it was 17.44 ±10.50. This difference was statistically significant. ($p<0.05$) Among the stroke patients 37.5% had Lp (a) values >30mg/dl and among persons without stroke only 12.5% had elevated Lp (a) values. The difference was statistically significant. ($p<0.05$)**.** The current research has observed no significant correlation of serum Lipoprotein (a) and other markers like blood pressure, fasting blood glucose, total cholesterol, LDL –c, HDL –c, triglycerides and BMI.
Conclusion: The study revealed that the mean serum Lp (a) levels in patients with stroke were significantly higher than persons without stroke.

Keywords: Lipoprotein (a), stroke, ischemic stroke

INTRODUCTION

Stroke or cerebrovascular accident has been an escalating public health problem globally. As reported by WHO, stroke accounts for 5.7 million deaths worldwide. In India, prevalence of stroke is detected to be 1.54 with a death rate of 0.6 per 1000 population.[1] The conventional risk factors for stroke are hypertension, metabolic disorders such as diabetes and dyslipidemia, lifestyle factors like smoking, alcoholism, lack of physical activity and non-modifiable factors including age and gender and other rare causes.[2] Modification of these factors has been shown to reduce the risk of stroke.

However, these factors account for only 50-60% of strokes, leaving a significant gap in the current understanding of the pathogenesis of stroke.[3] This knowledge gap has led to considerable research on nontraditional risk

factors and genetic drivers of stroke. There is growing evidence that biological markers, like lipoprotein (a) [Lp(a)], add to the prognostic value of conventional risk factors and may serve as useful tools in identifying subjects at risk.[4] Lipoprotein (a), for the first time described in 1963 by Berg belongs to the lipoproteins with the strongest atherogenic effect. Lp (a) consists of an LDL particle covalently bound to apolipoprotein (a) via a disulfide bridge.[5] Lp (a) is also highly prothrombotic and antifibrinolytic, due to its structural resemblance to the fibrinolytic enzyme plasminogen. Furthermore, the atherogenicity of Lp (a) is 10 times higher than that of LDL.[6] Thus, with its dual lipid and plasminogen like components, Lp (a) provides a critical link between atherothrombosis and atherosclerosis.

Over many years, Lp(a)) has been proven as a genetically determined predictor of atherosclerotic vascular diseases mainly coronary artery disease. But the relationship between high Lp(a) levels and stroke had been controversial. In 2006, a metanalysis of thirty-one studies, encompassing over 56,010 subjects with 4609 stroke events concluded that high Lp (a) is a risk factor for incident stroke also[7].

Lp (a) is an important risk factor for premature stroke among Asian Indians[8] and south Asians especially in those younger than 40 years of age[9]. A study was conducted among North Indian patients by Chakraborty and Vishnoi in 2012[10]. They recruited 100 consecutive patients with acute ischemic stroke and 120 controls. Lp (a) levels were consistently higher among cases than controls ($P < 0001$).

Lp (a) levels, unlike LDL-cholesterol, do not vary with the age of the subject. Lp (a) is completely expressed in the first year of life itself. Hence, its tracking provides greater prognostic value than any other lipids.

A high Lp (a) level is particularly detrimental in the presence of other risk factors like hypertension, dyslipidemia, diabetes etc. Therefore, a wise strategy should involve proactive management of all established risk factors in patients exhibiting high serum Lp (a) levels.

Objectives

1. To study the serum levels of Lipoprotein (a) in patients who developed ischemic stroke in comparison with a group of people who do not have the clinical outcome.
2. To find out the correlation between the serum Lipoprotein (a) and other parameters like blood pressure, fasting blood sugar and lipid profile in stroke patients.
3. To compare stroke patients and persons without stroke with respect to factors such as hypertension, diabetes mellitus and dyslipidemia.

MATERIALS & METHODS

Study Design: Cross-sectional study

Inclusion Criteria

Newly diagnosed 40 ischemic stroke patients in the age group of 45-60 yrs, who were admitted to the Department of Neurology. All patients had clinical and neuroimaging (CT or MRI) findings suggestive of ischemic stroke. Another group of 40 age matched subjects, who had not experienced ischemic stroke were included in the study as a comparison group. Majority of the persons in this group were the relatives of the patients who accompanied them to the hospital.

Exclusion Criteria

The following exclusion criteria were applied to all study subjects.

1. Haemorrhagic stroke
2. Stroke with cardiogenic source for embolism
3. Prior history of stroke
4. Prior history of coronary artery disease
5. Hepatic and renal diseases

STUDY SETTING

The study was conducted in the Department of Neurology at the Government Medical College, Thiruvananthapuram

SAMPLE SIZE

Sample size was calculated using the formula.

$$N = \left(Z_\alpha + Z_\beta\right)^2 \times \frac{\sigma 1^2 + \sigma 2^2}{(\mu 1 - \mu 2)^2}$$

$\sigma_1 = 12$

$\sigma_2 = 21$

$\mu_1 = 26$

$\mu_2 = 15$

$$= 7.9 \times \frac{(12^2 + 21^2)}{11^2}$$

$$= 7.9 \times \frac{(144 + 441)}{121}$$

$= 38$

For the present study sample size was fixed at 40 which included 40 ischemic stroke patients and 40 persons without history of stroke.

Data Collection

The study was conducted after getting clearance from the ethical committee. The details were recorded in the Proforma after obtaining informed consent from each participant. A detailed history, clinical examination and laboratory investigations were done.

Procedure

Fasting serum lipoprotein (a) levels were estimated in patients using TRAI / LP (A)/AUT-000 kit.

Method: Measurement of antigen-antibody reaction by the end point method.

Reference Values for Lp (a)

Normal values: 0 – 30 mg/dl.

Estimation of fasting blood glucose was done by Glucose Oxidase method. Those persons with Fasting blood sugar > 126 mg/ dl were considered as diabetics according to ADA criteria[11].

Individuals whose BP > 140/90 mm Hg were diagnosed as hypertensives based on JNC-7 report criteria[12].

ESTIMATION OF LIPID PROFILE

According to National Cholesterol Education Programme, Adult Treatment panel III ((NCEP-ATP III) criteria,[13] Fasting serum total cholesterol (TC) value > 200 mg/dl, Triglyceride (TG) > 150 mg%, HDL – cholesterol (HDL-C) < 40 mg% and LDL – cholesterol (LDL-C) >130 mg% was taken as abnormal.

Statistical Analysis

Statistical analysis was performed using Statistical Package for Social Services [SPSS] version 16. For all analyses, p value of <0.05 was considered significant.

RESULT

Table 1: Comparison of mean Lipoprotein (a) levels between Stroke patients and persons without stroke

Category	N	Lpa		t	p
		Mean	SD		
Stroke patients	40	24.76	10.74		
Persons without stroke	40	17.44	10.50	3.082	0.003*

The statistical significance of the difference in mean Lp (a) levels between the two groups was analyzed using the Student's t-test. The mean ± SD of Lp (a) in stroke patients was 24.76 ±10.74 whereas in person without stroke it was 17.44 ±10.50. The difference was statistically significant. (p<0.05)

Table 2: Distribution of study subjects based on serum Lp (a) levels

Lp(a)	Stroke patients		Persons without stroke	
	N	%	N	%
>30mg/dl	15	37.5	5	12.5
<30mg/dl	25	62.5	35	87.5
Total	40	100.0	40	100.0

$\chi 2$ =6.667, df =1, p=0.010*, OR=4.200, 95% CI=1.350-13.065

The distribution of study subjects according to l Lp (a) status was compared using the Pearson's Chi-square test.
Among the stroke patients 37.5% had Lp (a) >30mg/dl and among persons without stroke only 12.5% had high Lp (a) values. The difference was statistically significant. ($p<0.05$)

Table 3: Correlation of serum Lp (a) with other variables

Variables	Case (N=40)	
	Pearson Correlation coefficient (r)	**p value**
FBS	-.037	.821
T.C.	.208	.199
LDL- C	.190	.240
HDL- C	.301	.059
TG	-.090	.581
SBP	.193	.234
DBP	.075	.646

"The relationship between serum Lipoprotein (a) levels and other continuous variables was assessed using the Pearson Correlation Coefficient.
The present study has observed no significant correlation of serum Lipoprotein (a) and other variables like fasting blood sugar, Systolic blood pressure (SBP), Diastolic Blood Pressure (DBP), total cholesterol, LDL –C, HDL –C and triglycerides.

Table 4: Distribution of study subjects based on hypertension

Hypertension	Stroke patients		Persons without Stroke	
	N	**%**	**N**	**%**
Present	29	72.5	17	42.5
Absent	11	27.5	23	57.5
Total	40	100.0	40	100.0

$\chi 2$ =7.366, df =1, p=0.007*, OR=3.567 95%, CI=1.400-9.088

The distribution of study subjects based on hypertension was performed between the two groups using the Pearson's Chi-square test.
Among the stroke patients, 72.5% were hypertensives whereas in the other group, 42.5% were hypertensives. A statistically significant difference was noted. ($p <0.05$)

Table 5: Distribution of study subjects based on Diabetes

Diabetes	Stroke patients		Persons without Stroke	
	N	**%**	**N**	**%**
Present	15	37.5	5	12.5
Absent	25	62.5	35	87.5
Total	40	100.0	40	100.0

$\chi 2$ = 6.667, df =1, p=0.010*

The distribution of study subjects based on diabetes was performed using the Pearson's Chi-square test.
Diabetes was found to be present in 37.5% of stroke patient and 12.5% of persons without stroke. The difference noted was significant statistically. ($p<0.05$)

Table 6: Distribution of Study subjects based on Lipid Profile

Variables	Category (mg/dl)	Stroke Patients	Persons without stroke	p value
Total cholesterol	>200	23 (57.5%)	10 (25%)	0.003*
	<200	17 (42.5%)	30 (75%)	
Triglycerides	>150	19 (47.5%)	11 (27.5%)	0.065
	<150	21 (52.5%)	29 (72.5%)	
HDL cholesterol	<40	25 (62.5%)	22 (55%)	0.49
	>40	15 (37.5%)	18 (45%)	
LDL cholesterol	>130	21 (52.5%)	12 (30%)	0.04*
	<130	19 (47.5%)	28 (70%)	

The distribution of study subjects based on lipid profile was performed using the Pearson's Chi-square test.

Among stroke patients, 57.5% had abnormal total cholesterol values whereas in persons without stroke, only 25% had high TC values. Chi square analysis showed the difference to be statistically significant. ($p< 0.05$) LDL cholesterol levels were high in 52.5% of stroke patients and 30% of persons without stroke. The difference was statistically significant. ($p< 0.05$) Elevated triglycerides were present in 47.5% of stroke patients while in persons without stroke, it was present in 27.5%. The difference was statistically not significant. ($p> 0.05$) Among stroke patients, 62.5% showed low HDL cholesterol levels whereas in the other group, 55% had low HDL- cholesterol. The two groups showed no significant difference.

DISCUSSION

In the current study, the mean value of serum Lp (a) level in ischemic stroke patients was 24.76 mg /dl whereas in the group without stroke, it was 17.44 mg/dl (Table 1). The observed difference between the two groups was significant. (p value < 0.05). Researchers have found that white men with Lp(a) levels 80th centile or higher face a significantly elevated risk of cerebrovascular atherosclerotic diseases.[14] However, very few clinical researches have mapped the distribution of serum Lp (a) levels in Indian population. Therefore, the cut-off levels of Lp (a) were fixed at 30 mg/dl for the present study. Among the

stroke patients, 37.5% had Lp (a) > 30mg/dl whereas only 12.5% in the comparison group had high Lp(a) values (Table 2). This difference was found to be statistically significant ($p<0.05$).

Contribution of Lp(a) in Atherosclerosis

Lipoprotein (a) has been proved to exert both proatherogenic and prothrombotic effects in multiple stages of the atherosclerosis process.

I. Prothrombotic action

Due to its structural homology with plasminogen, Lp (a) can competitively interferes with the formation of active plasmin. By competing with both plasminogen and tissue plasminogen activator (tPA) for fibrin binding sites, Lp (a) maintains a prothrombotic environment.[15,16] Repeated subclinical thrombotic events may promote arterial injury at points of incipient plaque formation. Lp (a) also promotes atherosclerosis by impairing fibrin clearance in the injured blood vessel and by accumulating on fibrin-containing atherosclerotic plaques.[17]

II. Proatherogenic Action -

Lp (a) was proposed to act as the main scavenger for oxidized phospholipids which are inflammatory inducing in human circulation.[18] Lp (a) induces expression of tumour necrosis factor alpha (TNF- α), IL-1β, IL-8, and monocyte chemoattractant protein (MCP-1) in macrophages.[19] By modulating endothelial cell function, Lp (a) induces monocyte recruitment and chemotaxis via both direct and indirect mechanisms. Lp (a) has an affinity for many components of the subendothelial matrix including proteoglycans[20], fibrinogen and fibronectin.[21] LDL binds to the extra cellular

matrix of the arterial smooth muscle cells, which then increases two- to threefold the specific binding of LDL.[22] Lp (a) also binds to triglyceride-rich lipoproteins, a property which contributes to the accumulation of lipid in the arterial wall.[23] The development of atherosclerosis is characterised by endothelial dysfunction and proliferation and migration of SMC. It has been proposed that Lp (a) modulates vascular remodeling by proliferation of smooth Muscle Cells. Lp (a) stimulates smooth cell growth via apo (a) and LDL motifs, primarily by inhibiting the activation of TGF-β, a potent negative regulator of smooth muscle cell proliferation.[24,25]

Correlation of serum Lp (a) with other variables

The present study found no significant correlation between Lipoprotein (a) and other variables like fasting blood glucose, total cholesterol, LDL cholesterol, HDL cholesterol and Body Mass Index (Table 3). Similar results were obtained in a study conducted in 3915 CHD patients in Prague in 2008 in which serum Lp (a) levels were not significantly correlated with lipid and clinical parameters tested[26]. Many other studies also agree with similar results.[27]

Therapeutic Modification of Lp (a)

With varying degrees of success, a number of agents have been reported to lower circulating levels of Lp (a). Niacin or nicotinic acid is currently the first-choice therapy to specifically reduce Lp (a) levels.[28] It consistently decreases Lp (a) levels by 20-25%, when given in high doses (2 to 3 g/day).[29] The action of niacin appears to be through reducing Lp (a) production rates. LDL apheresis is a proven therapeutic method which should be reserved for individuals presenting with extreme elevations of Lp (a).[30] Diet, exercise, and standard pharmacologic measures to lower plasma LDL are considered as the most efficacious approach to treat Lp (a)-initiated atherogenesis because emerging evidence demonstrates that the atherogenic properties of Lp(a) are intimately related to that of LDL.[31] Substantial reduction in LDL- c attenuates the clinical threat of persistent elevation of Lp (a). From this perspective, LDL reduction with hydroxymethyl glutaryl coenzyme A reductase inhibitors (statins), fibrates, and ion exchange resins all play a very important role. In patients with documented atherosclerotic vascular disease, particularly in those with elevated serum Lp (a) levels, aspirin has been found to induce a reduction in Lp (a) levels,[32] This offers an affordable and efficient method of lowering L p (a) while reaping the anti-thrombogenic benefits of aspirin therapy. Other agents reported to lower Lp (a) levels include 2 g per day Lcarnitine[33], a combination of L-lysine and ascorbate (3g/day of each)[34] and the cholestin extract, Xuezhikang (1.2 g/day).[35]

Recommendations for the Determination of Serum Lp (a)

The European Atherosclerosis Society recommends that patients with a moderate or high risk of cardiovascular disease should have their lipoprotein (a) levels measured[36]. Any patient with one of the following risk factors should be screened;

- Premature cardiovascular disease
- Familial hypercholesterolaemia
- Family history of early cardiovascular disease
- Family history of elevated lipoprotein (a)
- Recurrent cardiovascular disease despite ongoing statin therapy

CONCLUSION

The study revealed the following findings:

1. The mean serum Lp (a) levels were significantly higher in stroke patients compared to individuals without a history of stroke.
2. No significant correlation was obtained between Lp(a) and Fasting Blood Sugar, Total Cholesterol, LDL- c, HDL- c, and Triglycerides.
3. Among the stroke patients, the proportion of hypertensives and diabetics were significantly higher compared with the comparison group.

4. Total and LDL cholesterol levels were found to be significantly elevated in stroke patients compared with persons without stroke.
5. There was no significant difference in triglyceride values and HDL cholesterol levels between the two groups.

Till date, our understanding of the precise mechanisms of vascular pathology of Lp (a) remains relatively poor. Further research is required to fully identify the genetic determinants and environmental factors controlling the circulating concentrations of Lp (a) also need further definition. There is lack of a universally accepted, standardized method for determination of serum Lp (a). An accurate and efficient means of estimating plasma Lp (a) levels using reagents that do not cross-react with plasminogen is essential. The development of a targeted therapeutic agent which is capable of specifically lowering the lower Lp (a) levels remains a primary challenge. The availability of a safe and cost effective Lp (a) lowering therapies will enable researchers to conduct intervention trials to clarify Lp (a)'s contribution to the development of stroke.

Declaration by Authors

Ethical Approval: Approved
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Deoke A, Deoke S, Saoji A, Hajare S. Profile of Modifiable and Non-Modifiable Risk Factors in Stroke in a Rural Based Tertiary Care Hospital – A Case Control Study. Glob J Health Sci. 2012;4(3):158–63. doi:10.5539/gjhs. v4n3p158
2. O'Donnell MJ, Chin SL, Rangarajan S, Xavier D, Liu L, Zhang H, et al. Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study. Lancet. 2016;388(10046):761–75. doi:10.1016/S0140-6736(16)30506-2
3. Dhamija RK, Arora S, Gaba P, Jais M, Kaintura A, Kumar M, et al. Study of genetic, metabolic and inflammatory risk factors in patients of acute ischemic stroke. Indian J Clin Biochem. 2008;23(2):136–43. doi:10.1007/s12291-008-0031-5
4. Christogiannis L, Milionis HJ, Elisaf M. Lipoprotein (a) and Stroke: An Overview. The Open Clinical Biochemistry Journal. 2010; 3: 38-43. doi:10.2174/1874241601003010038
5. Dahlén GH. Lp(a) lipoprotein in cardiovascular disease. Atherosclerosis. 1994;108(2):111–26. doi:10.1016/0021-9150(94)90106-6
6. Kronenberg F, Utermann G. Lipoprotein(a): resurrected by genetics. J Intern Med. 2013;273(1):6–30. doi:10.1111/j.1365-2796.2012. 02592.x
7. Smolders B, Lemmens R, Thijs V. Lipoprotein (a) and Stroke. Stroke. 2007;38(6):1959–66. doi:10.1161/STROKEAHA.106.480657
8. Nagayama M, Shinohara Y, Nagayama T. Lipoprotein(a) and ischemic cerebrovascular disease in young adults. Stroke. 1994;25(1):74–8. doi: 10.1161/01.STR.25.1.74
9. Sharobeem KM, Patel JV, Ritch AES, Lip GYH, Gill PS, Hughes EA. Elevated lipoprotein (a) and apolipoprotein B to AI ratio in South Asian patients with ischaemic stroke. Int J Clin Pract. 2007;61(11):1824–8. doi:10.1111/j.1742-1241.2007. 01521.x
10. Chakraborty B, Vishnoi G, Goswami B, Gowda SH, Chowdhury D, Agarwal S. Lipoprotein(a), ferritin, and albumin in acute phase reaction predicts severity and mortality of acute ischemic stroke in North Indian Patients. J Stroke Cerebrovasc Dis. 2013;22(7): e159-167. doi: 10.1016/j.jstrokecerebrovasdis.2012.10.013
11. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care. 2012;36(Supplement_1): S67–74. doi:10.2337/dc13-S067
12. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo J Joseph L, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. The JNC 7 Report. JAMA. 2003;289(19):2560–71. doi:10.1001/jama.289.19.2560
13. Expert Panel on Detection E and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of

the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285(19):2486–97. doi:10.1001/jama.285.19.2486
14. Milionis HJ, Winder AF, Mikhailidis DP. Lipoprotein (a) and stroke. Journal of Clinical Pathology. 2000;53(7):487–96. doi:10.1136/jcp.53.7.487
15. Loscalzo J, Weinfeld M, Fless GM, Scanu AM. Lipoprotein(a), fibrin binding, and plasminogen activation. Arteriosclerosis. 1990;10(2):240–5. doi: 10.1161/01.atv.10.2.240
16. Rouy D, Grailhe P, Nigon F, Chapman J, Anglés-Cano E. Lipoprotein(a) impairs generation of plasmin by fibrin-bound tissue-type plasminogen activator. In vitro studies in a plasma milieu. Arterioscler Thromb. 1991;11(3):629–38. doi: 10.1161/01.atv.11.3.629
17. Hajjar KA, Gavish D, Breslow JL, Nachman RL. Lipoprotein(a) modulation of endothelial cell surface fibrinolysis and its potential role in atherosclerosis. Nature. 1989;339(6222):303–5. doi:10.1038/339303a0
18. Bergmark C, Dewan A, Orsoni A, Merki E, Miller ER, Shin MJ, et al. A novel function of lipoprotein [a] as a preferential carrier of oxidized phospholipids in human plasma. Journal of Lipid Research. 2008;49(10):2230–9. doi:10.1194/jlr.M800174-JLR200
19. Riches K, Porter KE. Lipoprotein(a): Cellular Effects and Molecular Mechanisms. Cholesterol. 2012;2012(1):923289. doi:10.1155/2012/923289
20. Lundstam U, Hurt-Camejo E, Olsson G, Sartipy P, Camejo G, Wiklund O. Proteoglycans Contribution to Association of Lp(a) and LDL With Smooth Muscle Cell Extracellular Matrix. Arteriosclerosis, Thrombosis, and Vascular Biology. 1999;19(5):1162–7. doi: 10.1161/01.ATV.19.5.1162
21. Koschinsky ML, Tomlinson JE, Zioncheck TF, Schwartz K, Eaton DL, Lawn RM. Apolipoprotein(a): expression and characterization of a recombinant form of the protein in mammalian cells. Biochemistry. 1991;30(20):5044–51. doi:10.1021/bi00234a029
22. Morrisett JD. The role of lipoprotein[a] in atherosclerosis. Curr Atheroscler Rep. 2000;2(3):243–50. doi:10.1007/s11883-000-0026-z PubMed PMID: 11122750.
23. Gaubatz JW, Hoogeveen RC, Hoffman AS, Ghazzaly KG, Pownall HJ, Guevara J, et al. Isolation, quantitation, and characterization of a stable complex formed by Lp[a] binding to triglyceride-rich lipoproteins. J Lipid Res. 2001;42(12):2058–68. PubMed PMID: 11734579.
24. Takahashi A, Taniguchi T, Fujioka Y, Ishikawa Y, Yokoyama M. Effects of lipoprotein(a) and low-density lipoprotein on growth of mitogen-stimulated human umbilical vein endothelial cells. Atherosclerosis. 1996;120(1–2):93–9. doi:10.1016/0021-9150(95)05686-6
25. Jamieson DG, Usher DC, Rader DJ, Lavi E. Apolipoprotein(a) deposition in atherosclerotic plaques of cerebral vessels. A potential role for endothelial cells in lesion formation. Am J Pathol. 1995;147(6):1567–74.
26. Zlatohlávek L, Zídková K, Vrablík M, Haas T, Prusíková M, Svobodová H, et al. Lipoprotein(a) and its position among other risk factors of atherosclerosis. Physiol Res. 2008;57(5):777–84. doi:10.33549/physiolres.931133
27. Shai I, Schulze MB, Manson JE, Stampfer MJ, Rifai N, Hu FB. A prospective study of lipoprotein(a) and risk of coronary heart disease among women with type 2 diabetes. Diabetologia. 2005;48(8):1469–76. doi:10.1007/s00125-005-1814-3
28. Scanu AM, Bamba R. Niacin and lipoprotein(a): facts, uncertainties, and clinical considerations. Am J Cardiol. 2008;101(8A):44B-47B. doi: 10.1016/j.amjcard.2008.02.031
29. Crouse JR. New developments in the use of niacin for treatment of hyperlipidemia: new considerations in the use of an old drug. Coron Artery Dis. 1996;7(4):321–6. doi:10.1097/00019501-199604000-00009
30. Siekmeier R, Scharnagl H, Kostner GM, Grammer T, Stojakovic T, März W. Lipoprotein(a) – Structure, Epidemiology, Function and Diagnostics of a Cardiovascular Risk Marker. The Open Clinical Biochemistry Journal. 2008; 1: 79-91. doi:10.2174/1874241600801010079
31. Kostner GM, Grillhofer HK. Lipoprotein(a) mediates high affinity low density

lipoprotein association to receptor negative fibroblasts. J Biol Chem. 1991;266(31): 21287–92.
32. Akaike M, Azuma H, Kagawa A, Matsumoto K, Hayashi I, Tamura K, et al. Effect of aspirin treatment on serum concentrations of lipoprotein(a) in patients with atherosclerotic diseases. Clin Chem. 2002;48(9):1454–9.
33. Dalessandri KM. Reduction of lipoprotein(a) in postmenopausal women. Arch Intern Med. 2001;161(5):772–3. doi:10.1001/archinte.161.5.772-a
34. Sirtori CR, Calabresi L, Ferrara S, Pazzucconi F, Bondioli A, Baldassarre D, et al. L-carnitine reduces plasma lipoprotein(a) levels in patients with hyper Lp(a). Nutr Metab Cardiovasc Dis. 2000;10(5):247–51. PubMed PMID: 11213533.
35. Liu L, Zhao SP, Cheng YC, Li YL. Xuezhikang decreases serum lipoprotein(a) and C-reactive protein concentrations in patients with coronary heart disease. Clin Chem. 2003;49(8):1347–52. doi:10.1373/49.8.1347
36. Nordestgaard BG, Chapman MJ, Ray K, Borén J, Andreotti F, Watts GF, et al. Lipoprotein(a) as a cardiovascular risk factor: current status. Eur Heart J. 2010;31(23):2844–53. doi:10.1093/eurheartj/ehq386

How to cite this article: Priya Theresa Job, T. Sobhakumari, Thomas Iype. Serum lipoprotein (a) levels in patients with ischemic stroke. *Int J Health Sci Res.* 2026; 16(4):8-16. DOI: *https://doi.org/10.52403/ijhsr.20260402*

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