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Review Article

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Prevalence and Distribution of High Lipoprotein(a) Levels in Indian Populations: A Systematic Review

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Abstract

Background: Lipoprotein(a) [Lp(a)] is a genetically determined and independent risk factor for atherosclerotic cardiovascular disease (ASCVD). South Asians, particularly Indians have a greater burden of premature cardiovascular disease. However, prevalence and distribution of elevated Lp(a) levels across Indian populations remain scattered and inconsistently reported. Objective: To systematically evaluate the prevalence and distribution of elevated Lp(a) in Indian adults across healthy populations and various clinical conditions.

Methods: A systematic search of electronic databases was conducted to identify studies reporting Lp(a) levels in Indian adults. Randomized controlled trials (RCTs), post hoc analyses, and all observational investigations, including cross-sectional, cohort, and registry-based studies, retrospective studies, conference abstracts evaluating Lp(a) concentrations in healthy Indian individuals or Indian patients with cardiovascular or metabolic conditions were included.

Results: A total of 53 studies comprising 20,486 Indian adults were included in the qualitative synthesis. Mean Lp(a) levels in healthy or control Indian populations ranged from 6.68 to 33.51 mg/dL, with median levels typically between 12–20 mg/dL. Among Indian patients with coronary artery disease, the prevalence of elevated Lp(a) levels >30 mg/dL ranged from 32% to 70%, with some studies reporting Lp(a) ≥50 mg/dL in 34% and 37% of Indian patients with ACS and ASCVD, respectively. Elevated Lp(a) levels were also reported in Indian patients with dyslipidemia, stroke, diabetes, and familial hypercholesterolemia.

Conclusion: Indian adults have a high burden of elevated Lp(a) across both healthy and clinical populations. These findings highlight the need for greater awareness and broader implementation of Lp(a) testing to improve cardiovascular risk stratification in India.

Keywords: Lipoprotein(a); Cardiovascular disease; Dyslipidemia; Prevalence; Atherosclerotic cardiovascular disease; Systematic review.

Introduction

Atherosclerotic cardiovascular disease (ASCVD) is a major cause of morbidity and mortality worldwide, with cardiovascular diseases (CVD) responsible for nearly 17.9 million deaths annually [1]. South Asians, particularly Indians, suffer from premature and more severe ASCVD compared to other ethnicities, often manifesting at younger ages and lower body mass indices [2]. The incidence of acute myocardial infarction (MI) is

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reported to be three- to fivefold higher among young Indians compared with individuals from other ethnic groups [3]. This excess burden and early manifestation in young Indians are not fully explained by traditional cardiovascular (CV) risk factors, which has triggered increasing interest in non-traditional and emerging risk markers [3].

Lipoprotein (a) [Lp(a)] is an inherited, independent, and causal risk factor for ASCVD, and has also been associated with premature coronary artery disease (CAD) [3]. Elevated Lp(a) concentrations are frequently observed among Indians with aggressive or malignant forms of CAD [3,4]. High Lp(a) levels confer a two- to threefold increased risk of developing CAD [3,5]. Moreover, Lp(a) is considered substantially more atherogenic than LDL-C, with evidence suggesting that, on a particle-for-particle basis, Lp(a) may be up to six times more atherogenic due to its unique apolipoprotein(a) component [6]. Among different ethnic groups, South Asians have the second highest Lp(a) concentrations and the greatest risk of acute MI attributable to elevated Lp(a) [3]. While Lp(a) levels ≥50 mg/dL are widely considered as high and clinically significant, with increased risk of premature and aggressive ASCVD, emerging expert opinions suggest that lower thresholds may also be clinically relevant [7-11]. In an Indian expert consensus, panelists emphasized that Lp(a) levels >30 mg/dL are associated with an increased risk of CV events and should be incorporated into CV risk assessment, particularly in high-risk populations [11].

Despite its clinical relevance and recommendations by major guidelines for Lp(a) testing in high-risk opulations and once in a lifetime, routine testing for Lp(a) is low worldwide and particularly in India, both in the general population and in patients with established ASCVD [12,13]. Considering the fact that measuring Lp(a) can help to identify patients who may benefit from more aggressive risk factor optimization, including intensification of treatment, low testing frequency is concerning [14]. In fact, Lp(a) testing can improve CV risk stratification, especially in individuals with very high Lp(a), and increases CVD risk prediction, allowing re-classification of individuals or patients into higher risk categories who may otherwise not meet guideline criteria for lipid-lowering therapy [8,15].

Despite compelling evidence that elevated Lp(a) is a causal and independent CV risk factor, its epidemiologic profile in India is poorly defined. Systematic data on Lp(a) levels and prevalence in Indian populations are fragmented. No prior systematic review has comprehensively synthesized Indian evidence across diverse clinical groups. In the context of India's disproportionately high rates of premature ASCVD and the growing relevance of therapies that lower Lp(a), a rigorous synthesis of all available Indian data is essential. This systematic review addresses these gaps by providing the first consolidated estimation of Lp(a) burden, and disease-specific patterns in Indian adults, thereby generating evidence needed to inform screening policies, improve risk stratification, and guide future therapeutic decision-making.

Methods

This study is designed as a systematic review of published literature and eligible conference abstracts reporting Lp(a) levels in Indian populations. The study was conducted in accordance with the updated Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 Guidelines.[16] The review protocol was prospectively registered with the International Prospective Register of Systematic Reviews (PROSPERO) under the registration number CRD420251228987. This study aims to determine the prevalence and distribution of elevated Lp(a) levels (>30 mg/dL, >50 mg/dL, >70 mg/dL) in Indian populations; to compare mean/median Lp(a) levels across clinical subgroups (ASCVD, diabetes, stroke, familial hypercholesterolemia (FH), and healthy individuals); and to evaluate demographic variation in Lp(a) levels.

Search Strategy

A comprehensive search was conducted in the following electronic databases: PubMed/MEDLINE, EMBASE, Scopus, Web of Science, Cochrane Central Register of Controlled Trials (CENTRAL), and Google Scholar (first 200 results) for grey literature. Additionally, literature was identified through conference proceedings and abstract repositories of Cardiological Society of India (CSI), Lipid Association of India (LAI), European Society of Cardiology (ESC), American College of Cardiology (ACC), and European Atherosclerosis Society (EAS). The search strategy focused on studies reporting Lp(a) levels in Indian populations, published between January 1, 2000, and July 31, 2025. A combination of controlled vocabulary (e.g., MeSH terms) and free-text keywords was used to maximize retrieval sensitivity.

Eligibility Criteria

Inclusion Criteria

Population: Indian adults (≥18 years), including both healthy individuals and patients with established or high risk for ASCVD, diabetes mellitus, stroke, familial hypercholesterolemia, or other CV conditions, in whom Lp(a) levels have been measured.

Intervention / Exposure: Measurement and reporting of Lp(a) levels**.**

Comparator(s) or control(s)**:** Patients tested vs not tested within the same study population; Lp(a) levels in patients with ASCVD, diabetes, stroke, familial hypercholesterolemia, healthy individuals; demographic factors such as age groups, and sex.

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Study Designs: We included randomized controlled trials (RCTs), post hoc analyses, and observational investigations, including cross-sectional, cohort, and registry-based studies, retrospective studies, and conference abstracts that reported measured Lp(a) levels in Indian populations. Studies were eligible if they provided prevalence estimates of normal, or elevated Lp(a), mean or median Lp(a) levels, either overall or stratified by clinical subgroups.

Language: Only studies published in English were considered.

Exclusion criteria

Case reports, case series, letters to the editor, narrative reviews, systematic reviews, and meta-analyses; studies conducted in non-Indian populations, or where Indian data could not be extracted separately; and studies conducted in children or adolescents (<18 years), animal models, and those reporting only genetic determinants of Lp(a) without actual plasma level measurements.

Outcome measures

The outcomes included prevalence of elevated Lp(a) levels at defined thresholds (e.g., >30 mg/dL, >50 mg/dL, or >70 mg/dL), and mean/median Lp(a) levels across different populations and clinical groups.

Data Extraction

Two reviewers independently screened all retrieved studies by titles and abstracts, followed by full-text evaluation of potentially relevant studies against predefined inclusion and exclusion criteria. Discrepancies in study selection or data interpretation were resolved through discussion or consultation with a third reviewer. For each eligible study, data were extracted using a standardized form. All extracted data were cross-verified for accuracy and completeness before inclusion in the final evidence synthesis.

Quality assessment

Risk of bias and methodological quality were appraised independently by two reviewers for all included studies, with disagreements resolved by discussion or a third reviewer. We used design-appropriate, validated tools. For randomized controlled trials, Cochrane Risk of Bias 2 (RoB_2) was used [17]. Cross-sectional studies were assessed using Joanna Briggs Institute (JBI) critical appraisal tool of 8 items [18]. Observational studies underwent evaluation with Newcastle Ottawa Scale (NOS) (ranging for 0-9) [19].

Results

Search results

A total of 208 articles were initially retrieved through the database search. After screening titles and abstracts, 150 articles were selected for full-text review, along with three conference abstracts. Following full-text assessment, 97 studies were excluded for the following reasons: not conducted in Indian population (n=26), study design did not match inclusion criteria (n=26), involved pediatric population (n=1), Lp(a) assessment either not undertaken or performed in a heterogeneous population (n=41), non-English publication (n=1), full text unavailable (n=1), and duplicate publication (n=1). Overall, 50 studies met the predefined eligibility criteria and were included in the qualitative synthesis [2,4,20-28,30-47,49-69]. In addition, three eligible conference abstracts published in peer-reviewed journals were also included in this review [29,48,70]. Thus, a total of 53 records reporting Lp(a) data in Indian population, including healthy individuals, patients with various clinical conditions, and individuals of Indian origin residing outside India were included in the final analysis (Figure 1). The findings presented in the subsequent sections relate to Indian populations included in this review, comprising both individuals residing in India and, where applicable, populations of Indian origin residing outside India.

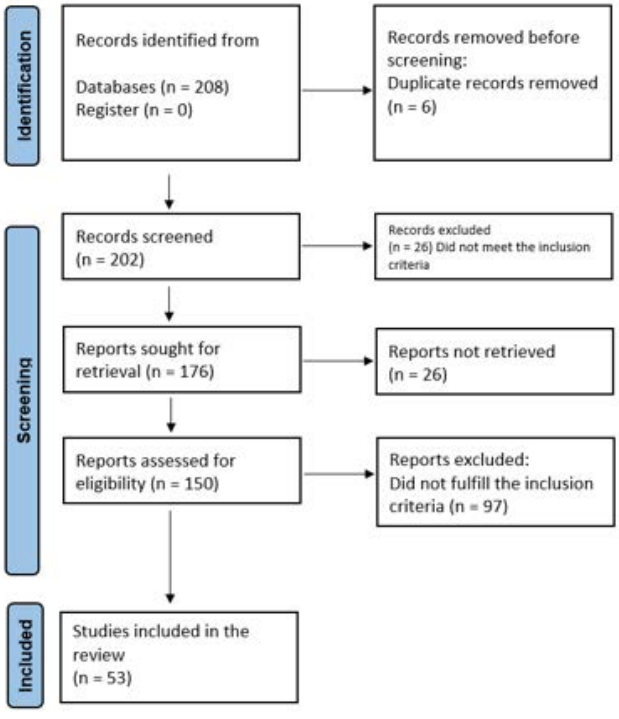

Figure 1: PRISMA flow diagram of included studies.

Study characteristics

A total of 53 studies with 20,486 Indian participants were analyzed.[2,4,20–70] Most studies were observational studies; case-control (n=24), [4,22,23,27,32,34,35,37,41-44,46,47,49-51,56,57,61,65,66,68,69] cross-sectional

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(n=18), [2,21,24,25,33,38,39,45,52,54,55,58-60,62-64,67] retrospective cohort (n=3), [20,28,36] prospective (n=2), [30,53] retro-prospective registry (n=1), [26] pre-post intervention (n=1), [40] and randomized clinical trial (n=1) [31]. For three studies, the study design was not reported (Table 1) [29,48,70]. The mean age of patients ranged from 20 years to 56 years. The study cohort predominantly comprised men, with 53.49% males (n=10,957) and 18.02% (n=3,692) females. For 5,837 participants, sex distribution was not provided. The majority of studies (n=48) were conducted in a hospital setting, 4 studies were community-based, [2,45,52,67] and one reported data from both hospital and community settings [54]. Clinical populations most frequently included ASCVD (n=5,914), CAD (n=3,616), type 2 diabetes (T2D) (n=2,066), and acute coronary syndrome (ACS) (n=1,717), cohorts, followed by MI (n=1,199), CHD (n=560), dyslipidemia (n=200), stroke (n=214), FH (n=181), and hypertension (n=100) groups. Lp(a) concentrations were variably reported as mean, median, or percentage above predefined thresholds (Table 1). Units were reported mainly in mg/dL; a few studies used nmol/L or ng/mL. Although prevalence of elevated Lp(a) levels >70 mg/dL was a predefined outcome, none of the included studies reported prevalence estimates using this threshold.

Lipoprotein (a) Assessment in Healthy Indian Individuals / Controls

A total of 32 studies (n=4016) included Lp(a) assessment data from a healthy Indian population.[2,4,22,24,27,32,34-37,41-47,49-52,54-57,61,62,65-69]. The healthy Indian individuals presented with mean Lp(a) levels ranging from 6.68 ± 3.4 mg/dL to 33.51 ± 23.0 mg/dL (Table 1) [2,22,24,32,34-37,41-47,49,50,54-56,61,62,65-69]. The median Lp(a) ranged from 12 mg/dL to 20 mg/dL [34,42,51,57]. Joseph et al. (2022) reported 28.9% of Indian non-CAD controls with elevated Lp(a) of >30 mg/dL [4]. Sontakke et al. (2014) reported 46% of healthy Indian women had elevated Lp(a), although the study did not define a specific threshold or criterion for elevated Lp(a) [67].

Table 1: Characteristics of the included studies.

Author (Year)	Study Design (study location)	Study Population and Sample Size	Mean Age (years) and Gender	Lp(a) Threshold	Key findings
Amin et al. (2024)[30]	Prospective Observational (Manipal, India)	CAD; n= 600	475 M / 125 F	30	58.3% had Lp(a) ≥30 mg/dL
Dudum et al. (2024)[20]	Retrospective (California)	ASCVD risk patients; n=2670 (Asian Indians)	44.7 ± 9.6 186 M / 824 F	50	Median (IQR): 34 mg/dL (18-66) 35.7% had Lp(a) ≥50 mg/dL
Sandhu et al. (2024)[21]	Cross-sectional (Kolkata, India)	CAD; n= 100	52.56 ± 12.84 84 M / 16 F	30	59% had Lp(a) ≥30 mg/dL
Sawhney et al. (2024)[29]	NA	Familial hypercholesterolemia; n=100	46.97 ± 8.56	50	43% had Lp(a) ≥50 mg/dL
Sawhney et al. (2023)[70]	NA	ACS; n=1021	NA	50	34% had Lp(a) ≥50 mg/dL 37% young ACS patients had Lp(a) ≥50 mg/dL vs 32% elderly patients 40% patients with multivessel disease had Lp(a) ≥50 mg/dL
Jain et al. (2023)[28]	Retrospective study	ACS; n= 575	NA	NA	Baseline: 49.15 ± 15.03 mg/dL 21% patients tested for Lp(a) 1 year post treatment: 26.57 ± 16.23 mg/dL (p<0.001)
Kalaivani et al. (2023)[65]	Case-control study (Chennai, India)	CKD: n=70 Controls; n=70	73 M / 67 F	NA	Mean ± SD Control: 17.88 ± 14.5 mg/dL Cases: 53.26 ± 39.56 mg/dL
Muheeb et al. (2023)[22]	Case-control (Delhi, India)	STEMI; n=110 Control; n=110	38.8 ± 6.7 201 M / 19 F	NA	Mean ± SD Cases- 87.56 ± 74.28 mg/dL Control- 25.81 ± 24.66 mg/dL p < 0.0001

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Singh et al. (2023)[48]	NA	Familial hypercholesterolemia; n=81	46.34 ± 9.0	50	39.5% had Lp(a) ≥50 mg/dL
Joseph et al. (2022)[4]	Case-control	CAD; n=682 Control; n=246	673 M / 255 F	30	Lp(a) ≥30 mg/dL
	(Kerala, India)				Cases: 32%
					Control: 28.9%
Loh et al. (2022)[23]	Case-control	CAD; n=316	NA	NA	Distribution of Lp(a) is a right skew pattern
	(Singapore)				
Mahto et al. (2022)[24]	Cross-sectional observational	Hypertension; n=100	Cases- 43.06 ± 10.68	NA	Mean ± SD
	(New Delhi, India)	Control; n=50	Control- 45.24 ± 11.31		Case: 34.03 ± 7.55 mg/dL
			90 M / 60 F		Control: 24.13 ± 4.41 mg/dL
					p < 0.001
Nissen et al. (2022)[25]	Multicentre, cross-sectional epidemiological study	ASCVD; (n=3,244 Indians)	NA	NA	Median (IQR) measured in mg/dL (n=3068 Indians): 25.5 (11.2, 57.9) mg/dL
	(Global)				Median (IQR) measured in nmol/L (n=176 Indians): 60.5 (24.5, 142.0) nmol/L
Rohit et al. (2020)[64]	Cross-sectional study	T2D; n=300	189 M / 111 F	NA	Mean ± SD
	(Vadodra, India)				T2D >5yrs: 19.94 ± 11.78 mg/dL
					T2D <5yrs: 19.64 ± 9.06 mg/dL
Shukla et al. (2019)[26]	Retro-prospective registry study	STEMI; n=787	35.58 ± 4.27	30	15.83% had Lp(a) ≥30 mg/dL
	(Gujarat, India)		736 M / 51 F		
Behera et al. (2019)[66]	Case-control study	CHD; n=50	68 M / 32 F	NA	Mean ± SD
	(Bhubaneswar, India)	Control; n=50			Cases: 50.85 ± 23.42 mg/dL
					Control: 17.10 ± 5.18 mg/dL
					p< 0.001
Wadhwa et al. (2019)[27]	Case-control study	Chronic plaque psoriasis; n=132	Cases- 40.76 ± 13.8	NA	Median ± IQR:
	(Himachal Pradesh, India)	Control; n=132	Control- 42.89 ± 14.56		Cases: 412.47 ± 222.99 ng/ml
			188 M / 76 F		Control: 334.95 ± 403.15 ng/ml
					p<0.001
Asre et al. (2018)[68]	Case-control study	AMI; n= 50	NA	NA	Mean ± SD
	(Agra, India)	Control; n=20			AMI: 21.60 ± 2.96 mg/dL
					Control: 19 ± 3.03 mg/dL
Chaudhary et al. (2017)[39]	Cross-sectional Observational	CAD; n=96	77 M / 19 F	30	70% of young adults (<40 years; n=60) had Lp(a) >30 mg/dL
	(North India)				
Gulati et al. (2017)[40]	Pre-post intervention	T2D; n=50	45.8 ± 9.3	NA	Median (IQR): 21.4 mg/dL (9.4, 122)
	(New Delhi, India)		27 M / 23 F		

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Vijayakumar et al. (2016)[31]	Randomized clinical trial	CAD; n=198 Group 1: Coconut oil	58.97 ± 8	NA	Mean ± SD
	(Kerala, India)	Group 2: Sunflower oil	185 M / 13 F		*Baseline:*
					Group 1: 21.81± 21.89
					Group 2: 25.13 ± 28.73
					2 years post-treatment:
					Group 1: 22.46 ± 20.24
					Group 2: 30.64 ± 31.13
Bansal et al. (2015)[32]	Case-Control	CAD; n=30	Cases- 42.47	NA	Mean ± SD
	(New Delhi, India)	Control; n=30	Control- 41.13		Cases: 43.17 ± 10.23 mg/dL
			38 M / 22 F		Control: 17.62 ± 3.18 mg/dL
					p<0.0001
Mukherjee et al. (2015)[33]	Cross-sectional study	ACS; n=70	48 M / 22 F	NA	41.43% of cases had high Lp(a)
	(Kolkata, India)				≥40 years: 50%
					<40 years: 26.92%
					Male: 41.67%
					Female: 40.9%
Sontakke et al. (2014)[67]	Cross-sectional study	Control; n=200	200 F	NA	Mean ± SD
	(Pune, India)				21-45 years: 32.20 ± 27.6 mg/dL
					50-55 years: 33.51 ± 23.0 mg/dL
					p=0.71
					The prevalence of elevated Lp(a) was 41% and 46% in premenopausal and menopausal women respectively.
Yusuf et al. (2014)[34]	Case-control study.	CAD; n=450	Cases: 53.74 ± 0.48	NA	Median Levels
	(North India)	Control; n=150	Control: 52.34 ± 0.88		Cases: 30.30 mg/dL
			490 M / 110 F		Control: 20 mg/dL
Ashfaq et al. (2013)[62]	Cross-sectional study	CAD; n=270	54.31 ± 8.35	NA	Mean ± SD
	(Lucknow, India)	Control; n=90	300 M / 60 F		CAD: 48.73 ± 23.85 mg/dL
					Non-CAD: 18.95 ± 11.15 mg/dL
					p<0.0001
Chakraborty et al. (2013)[35]	Case-control study	Acute ischemic stroke; n=100	Cases - 54.0± 10.9	NA	Mean ± 2 SD
	(New Delhi, India)	Control; n=120	Control- 52.5 ± 9.8		Cases: 82.3 ± 52.9 mg/dL
			152 M / 68 F		Control: 24.4 ± 5.0 mg/dL
					p=0.000
Mishra et al. (2013)[36]	Retrospective study	MI or Stroke; N=85	Cases :37.2	NA	Mean
	(Mumbai, India)	(MI, n= 37; Stroke, n=48) Control; n=50	Control: 34		Stroke Patients: 37 mg/dL
			124 M / 11 F		MI Patients: 40 mg/dL
					Control: 21 mg/dL

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Chandni et al. (2012)[59]	Cross-sectional study (Kozhikode, India)	T2D; n=144	53.93 ± 10.74 81 M / 63 F	30	26.4% had Lp(a) ≥30 mg/dL
Himabindu et al. (2012)[37]	Case-control study	ACS; n= 51	NA	NA	mean ± SEM
	(Andhra Pradesh, India)	Control; n=50			Cases: 14.50 ± 1.70 mg/dL Control: 13.20 ± 1.34 mg/dL p=0.551
Banerjee et al. (2011)[38]	Cross-sectional, retrospective (USA)	CVD; n=295	NA 213 M / 82 F	NA	Median Male: 35 nmol/L Female: 41 nmol/L
Goswami et al. (2010)[41]	Case-control study	AMI; n=150	Cases: 55.1 ± 9.6	NA	Mean ± SD
	(New Delhi, India)	Control; n=150	Control: 53.7 ± 10.2 300 M		Cases: 40.2 ± 6.54 mg/dL Control: 10.5 ± 2.34 mg/dL
Dhamija et al. (2009)[69]	Case-control study	Acute ischemic stroke; n= 66	Cases- 54.43 ± 1.97	NA	Mean ± SEM
	(New Delhi, India)	Control; n=72	Control: 53.86 ± 1.88 62 M / 76 F		Cases: 57.33 ± 4.40 mg/dL Control: 23.46 ± 1.09 mg/dL p<0.001
Singla et al. (2009)[61]	Case-control study	Diabetes; n=60	Cases: 52.3 ± 8.17	NA	Cases: 70.7 mg/dL
	(Patiala, India)	Control; n=50	Control: 54.12 ± 8.17 58 M / 52 F		Control: 19.83 mg/dL p< 0.001
Gambhir et al. (2008)[42]	Case-control study	CAD; n=220	Cases: 36.2 ± 3.8	NA	Median
	(Delhi and Noida, India)	Control; n=160	Control: 34.6 ± 5.0 329 M / 51 F		Cases: 30 mg/dL Control: 12.7 mg/dL
Chopra et al. (2007)[60]	Cross-sectional study (Ludhiana, India)	Diabetes; n=200	54.9-55.1 97 M/ 103 F	NA	Mean Diabetes: 25.1 mg/dL Diabetes with retinopathy: 68.5 mg/dL
Rajappa et al. (2006)[43]	Case-control study	CAD; n=106 Control; n=52	Group 1 (NIDDM with CAD): 56 ± 9	NA	Mean ± S.D.
	(Pondicherry, India)		Group 2 (NIDDM without CAD): 54 ± 5		Group 1: 67.92 ± 25.67 mg/dL
			Group 3(Control): 55 ± 11		Group 2: 18.38 ± 7.2 mg/dL
			NA		Group 3: 17.41 ± 7.06 mg/dL
Superko et al. (2005)[44]	Case-control study	Control;	Asian Indian: 49.0 ± 11.6	NA	Mean ± SD
	(United States)	173 Asian Indian	Non-Asian Indian: 49.2 ± 10.1		Asian Indian: 22.2 ± 17.2 mg/dL
		239 non-Asian Indian	412 M		Non-Asian Indian: 15.3 ± 14.0 mg/dL
Mahajan et al. (2004)[45]	Cross-sectional study (Sydney, Australia and India)	Control; n=250	Indians residing in Australia: 39 ± 10.3 Indians residing in India: 40 ± 11.5 150 M / 100 F	NA	India Group: 178 mg/l

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Rajasekhar et al. (2004)[46]	Case-control study	CHD; n= 151	Cases: 53.30 ± 9.12	NA	Mean ± S.D
	(Andhra Pradesh, India)	Control; n=49	Control: 48.71 ± 8.35		Cases: 24.79 ± 18.99 mg/dL
			158 M / 42 F		Control: 16.04 ± 17.53 mg/dL
Singh et al. (2004)[47]	Case-control study	CAD; n=54	Cases: 49.5 ± 4.2	NA	Mean ± SD
	(Moradabad, India)	Control; n=85	Control: 52.1 ± 5.2		Cases: 21.6 ± 5.0 mg/dL
			121 M/ 18 F		Control: 15.7 ± 3.6 mg/dL
Angeline et al. (2003)[49]	Case-control study	MI; n=65	<45	NA	Cases: 58.6 ± 3.20 mg/dL
	(Madurai, India)	Control; n=50	115 M		Control: 19.6 ± 0.10 mg/dL
					p<0.05
Geethanjali et al. (2003)[50]	Case-control study	CHD; n=254	Cases: 51.6	NA	Median
	(Tamil Nadu and New Delhi, India)	Control; n=480	Control: 43.1		Cases: 27.4 mg/dL
			571 M / 163 F		Control: 17.6 mg/dL
Tan et al. (2003)[51]	Case-control study	Indians	Cases: 58.1 ± 9.78	NA	Median
	(Singapore)	CAD; n=164	Control: 46.9 ± 14.15		Cases: 23.78 mg/dL
		Control; n=231	395 M		Control: 12.0 mg/dL
Tavridou et al. (2003)[52]	Cross-sectional study	Control; n=251	NA	NA	Mean (95% CI)
	(United Kingdom)		103 M / 148 F		Male: 177 (143–219) mg/L
					Female: 162 (137–192) mg/L
Velmurugan et al. (2003)[63]	Cross-sectional study	T2D; n=587	55 ± 10	NA	Mean ± SD
	(Chennai, India)		418 M / 169 F		18.9 ± 3.1 mg/dL
Deepa et al. (2002)[58]	Cross-sectional study	T2D; n=725	54 ± 10	NA	Mean ± SD
	(Chennai, India)		508 M / 217 F		15.5 ± 3.2 mg/dL
Goyal et al. (2002)[53]	Prospective Study	Dyslipidemia; n=200	NA	30	Mean ± SD
	(Punjab, India)		140 M / 60 F		44.8 ± 26.8 mg/dL
					40% had Lp(a) >30 mg/dL
Palaniaapan et al. (2002)[2]	Cross-sectional study	Control; n=70	American Indian: 20 ± 2	NA	Mean ± SD
	(Michigan, USA)		70 F		Asian Indian: 0.3 ± 0.3 g/L
Hoogeveen et al. (2001)[54]	Cross-sectional study	CHD; n=57	Cases: 52 ± 9 Controls: 40 ± 11	NA	Mean ± SD
	(Delhi and USA)	Control; n=46	USA Group: 43 ± 14		Cases: 12.65 ± 9.40 mg/dL
		Asian Indian immigrants in the USA = 206	215 M / 94 F		Control: 9.15 ± 7.33 mg/dL
					USA Group: 8.67 ± 8.24 mg/Dl
Ramachandran et al. (2001)[55]	Cross-sectional study	CAD; n=280	Cases: 56.2 ± 8.8	NA	Mean ± SD
	(Madras, India)	Control; n=167	Controls: 47.8 ± 10.5		Cases: 23.0 ± 3.3 mg/dL
			447 M		Control: 20.0 ± 3.4 mg/dL

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Gupta et al. (2000)[56]	Case-control study	CHD; n=48	NA	NA	Mean ± SD
	(Jaipur, India)	Control; n=23			Cases: 11.95 ± 2.8 mg/dL
					Control: 6.68 ± 3.4 mg/dL
					p = 0.041
					≤50 years
					Cases: 10.27 ± 2.8 mg/dL
					Control: 7.27 ± 3.4 mg/dL
					p<0.05
					>50 years
					Cases: 12.99 ± 2.9 mg/dL
					Control: 4.91 ± 3.5 mg/dL
					p<0.05
Gambhir et al. (2000)[57]	Case-control study	CAD; n=50	NA	NA	Mean ± SD
	(Delhi, India)	Control; n=50			Cases: 35.0 ± 32.4 mg/dL
					Control: 20.3 ± 17.0 mg/dL
					p<0.002
					Median
					Cases: 26.7 mg/dL
					Control: 13.8 mg/dL
					p<0.015

Note: ACS: Acute Coronary Syndrome; AMI: Acute Myocardial Infarction; ASCVD: Atherosclerotic Cardiovascular Disease; CAD: Coronary Artery Disease; CHD: Coronary Heart Disease; CKD: Chronic Kidney Disease; CVD: Cardiovascular Disease; ECG: Electrocardiogram; F: Female; HR: Hazard Ratio; IQR: Interquartile Range; Lp(a): Lipoprotein(a); M: Male; MI: Myocardial Infarction; NA: Not Available; OR: Odds Ratio; SD: Standard Deviation; SEM: Standard Error of Mean; STEMI: ST-Elevation Myocardial Infarction; T2D: Type 2 Diabetes; USA: United States of America.

Lipoprotein (a) Assessment in Indian Patients with Cardiovascular Diseases

Coronary Artery Disease

Total 15 studies assessed the association of Lp(a) and CAD in Indian population (n=3616) [4,21,23,30–32,34,39,42,43,47,51,55,57,62]. The reported prevalence of elevated Lp(a) (≥30 mg/dL) in Indian patients with CAD ranged between 32-70%, [4,21,30,39] and was 28.9% in Indian patients without coronary disease (Table 2) [4]. The majority of studies reported higher Lp(a) levels in Indian patients with CAD compared to the healthy Indian population [31,32,34,42,43,47,51,55,57,62]. Mean Lp(a) levels in CAD cases ranged from 21.6 ± 5.0 mg/dL to 67.92 ± 25.67 mg/dL, [31,32,43,47,55,57,62]. While healthy Indian individuals had mean Lp(a) levels from 15.7 ± 3.6 mg/dL to 20.3 ± 17.0 mg/dL [32,43,47,55,57,62]. Median Lp(a) levels in Indian patients with CAD ranged from 23.78 mg/dL to 30.30 mg/dL, with median in healthy Indian individuals ranging from 12 mg/dL to 20 mg/dL [34,42,51,57]. Four studies reported outcomes in Indian patients with ACS (n=1717) [28,33,37,70]. Mukherjee et al. (2015) found that 41.43% of Indian patients with ACS had high Lp(a), although the study did not define a specific threshold or criterion for high Lp(a) [33]. Sawhney et al. (2023) reported that 34% Indian ACS patients had Lp(a) ≥50 mg/dL [70]. Overall, 37% young Indian ACS patients had Lp(a) ≥50 mg/dL vs 32% elderly Indian patients and 40% Indian patients with multivessel disease had Lp(a) ≥50 mg/dL.[70] Jain et al. (2023) reported mean Lp(a) levels of 49.15 ± 15.03 mg/dL in Indian ACS patients.[28] Himabindhu et al. (2012) found mean Lp(a) levels of 14.50 ± 1.70 mg/dL in Indian ACS patients and 13.20 ± 1.34 mg/dL in healthy Indian individuals, but the difference was not statistically significant [37].

Coronary Heart Disease

Five studies investigated the Lp(a) levels in Indian patients with coronary heart disease (CHD) (n=560) (Table 2) [46,50,54,56,66]. Mean Lp(a) levels in Indian CHD patients ranged from 11.95 ± 2.8 mg/dL to 50.85 ± 23.42 mg/dL [46,54,56,66]. Mean levels in the healthy Indian cohort ranged from 6.68 ± 3.4 mg/dL to 17.10 ± 5.18 mg/dL [46,54,56,66]. Median Lp(a) levels were higher in Indian CHD patients (27.4 mg/dL) than healthy Indian individuals (17.6 mg/dL) [50]. Lp(a) concentrations increased with disease severity, with significantly higher levels in patients with triple-vessel disease compared to those with single-vessel disease or normal coronaries [50].

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Table 2: Lp(a) levels among healthy Indian population and diseased cohorts across included studies.

Study population	Mean Lp(a) range	Median Lp(a) range	% of patients with Lp(a) >30mg/dL	% of patients with Lp(a) >50mg/dL
Healthy Indian Controls (n=4,016)	6.68 - 33.51 mg/dL [2,22,24,32,35-37,41-47,49,50,54-56,61,62,65-69]	12 - 20 mg/dL [34,42,51,57]	28.9% [4]	NA
CAD (n=3,616)	21.6 - 67.92 mg/dL [31,32,43,47,55,57,62]	23.78 - 30.3 mg/dL [34,42,51,57]	32-70% [4,21,30,39]	NA
ACS (n=1,717)	14.50 – 49.15 mg/dL[28,37]	NA	NA	34%[70]
CHD (n=560)	11.95 - 50.85 mg/dL [46,54,56,66]	27.4 mg/dL [50]	NA	NA
MI (n=1,199)	21.60 - 87.56 mg/dL [22,36,41,49,68]	NA	15.83% [26]	NA
ASCVD (n=5,914)	NA	25.5 - 34 mg/dL [20,25]	NA	35.7% [20]
Type 2 Diabetes (n=2,066)	15.5 - 70.7 mg/dL [58,60,61,63,64]	21.4 mg/dL [40]	26.4% [59]	NA
Stroke (n=214)	37 - 82.3 mg/dL [35,36,69]	NA	NA	NA
Familial hypercholesterolemia (n=181)	NA	NA	NA	39.5-43% [29,48]
Dyslipidemia (n=200)	44.8 mg/dL[53]	NA	40%[53]	NA

Myocardial Infarction

Lp(a) levels in Indian patients with MI were reported in six studies (n=1199) (Table 2) [22,26,36,41,49,68]. A study by Shukla et al. (2019) reported that 15.83% of Indian patients with ST-Elevation Myocardial Infarction (STEMI) had Lp(a) ≥30 mg/dL [26]. Mean Lp(a) in Indian patients with MI ranged from 21.60 ± 2.96 mg/dL to 87.56 ± 74.28 mg/dL, while the healthy Indian population generally exhibited lower mean levels, ranging from 19.0 ± 3.03 mg/dL to 25.81 ± 24.66 mg/dL [22,36,41,49,68].

Atherosclerotic Cardiovascular Disease

Two studies evaluated the association between Lp(a) and ASCVD in Indian patients (n=5914) (Table 2) [20,25]. A study by Dudum et al. (2024) in Indian patients reported a median Lp(a) level of 34 mg/dL across various ASCVD risk levels, with 35.7% of the population having Lp(a) levels >50 mg/dL [20]. The global Lp(a) HERITAGE study found a median Lp(a) of 25.5 mg/dL in Indian ASCVD patients [25].

Risk Factors for Cardiovascular Disease

Lp(a) has been reported in relation to established CV risk factors in Indian patients, including FH (n=181), [29,48] T2D (n=2066), [40,58–61,63,64] dyslipidemia (n=200), [53] and stroke (n=214) (Table 2) [35,36,69]. Seven studies reported Lp(a) levels in the Indian diabetic population (n=2066) [40,58–61,63,64]. Chandini et al. (2012) observed that 26.4% of Indian T2D patients had Lp(a) ≥30 mg/dL [59]. The mean Lp(a) in Indian T2D patients ranged from 15.5 ± 3.2 mg/dL to 70.7 mg/dL; [58,60-64] median was reported as 21.4 mg/dL (9.4,122) [40]. A study in Indian patients by Singla et al. (2009) reported that Lp(a) concentrations were significantly higher in diabetic subjects than in healthy individuals (70.7 mg/dL vs. 19.83 mg/dL; $p < 0.001$) [61]. Among Indian diabetic subgroups, patients with retinopathy exhibited elevated Lp(a) levels compared to those without complications (68.5 mg/dL vs. 25.1 mg/dL) [60]. In Indian patients with FH, a significant proportion of patients exhibited elevated Lp(a) levels. Sawhney et al. (2024) and Singh et al. (2023) reported that 43% and 39.5%, respectively, of FH patients had Lp(a) greater than 50 mg/dL [29,48]. Goyal et al. (2002) found a mean Lp(a) level of 44.8 ± 26.8 mg/dL in Indian dyslipidemic patients and 40% of these patients had high serum Lp(a) levels (>30 mg/dL) [53].

Stroke

Three studies investigated the association between Lp(a) levels and stroke in Indian patients (n=214) [35,36,69]. Chakraborty et al. (2013) found mean Lp(a) levels of 82.3 ± 52.9 mg/dL in stroke cases versus 24.4 ± 5.0 mg/dL in the healthy Indian cohort (p=0.000) [35]. Dhamija et al. (2009) reported mean Lp(a) of 57.33 ± 4.40 mg/dL in acute ischemic stroke cases compared to 23.46 ± 1.09 mg/dL in healthy Indian individuals [69]. Mishra et al. (2013) observed a mean Lp(a) of 37 mg/dL in stroke patients, while healthy Indian individuals had 21 mg/dL [36]. High admission levels of Lp(a) were associated with increased stroke severity, a poorer long-term prognosis, and increased mortality, with an admission level Lp(a) >77 mg/dL associated with a significant increase in mortality (p=0.000) [35].

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Demographic

Across the limited studies reporting age-specific Lp(a) patterns in Indian populations, findings were inconsistent [4,33,56,67]. In ACS cohort, prevalence of elevated Lp(a) was numerically higher in older patient (≥40 years; 50%), compared to younger patients (<40 years; 26.92%), though the difference was not statistically significant [33]. Gupta et al. (2000) observed higher Lp(a) levels in the younger (≤50 years) than older (>50 years) individuals in healthy Indian control groups [56] whereas, Sontakke et al. (2014) found no significant age-related differences in healthy Indian women [67]. Overall, current evidence remains inconclusive, and no consistent age-related pattern has been established for Indian population. Across studies reporting sex-specific data, no consistent gender differences in Lp(a) levels were observed for Indian population. In ACS patients, the proportion of elevated Lp(a) was nearly identical in males (41.67%) and females (40.9%) [33]. Similar findings were reported in CVD cohorts, where median Lp(a) levels were comparable between men and women [38]. In a general population cohort, Tavridou et al. (2003) found similar mean Lp(a) concentrations between males and females [52].

Study Quality

Based on the NOS assessment, a total of 26 studies were evaluated for methodological quality. Of these, 8 studies were rated as good quality (scores 7–9), 15 studies as moderate quality (scores 4–6), and 3 studies as low quality (scores 0–3). (Table 3) Based on the Joanna Briggs Institute (JBI) critical appraisal checklist for analytical cross-sectional studies, the overall methodological quality of 20 studies was high across most domains. (Table 4) Nearly all studies clearly defined inclusion criteria, adequately described study subjects and settings, and used valid and reliable methods to measure exposure and outcomes. A consistent limitation across the majority of studies was the inadequate identification and management of confounding factors. A single RCT was assessed using the ROB_{-2} tool and was found to have low risk of bias.[31] Abstracts were not assessed for quality and are considered low-quality evidence [29,48,70].

Discussion

This systematic review provides the most comprehensive synthesis to date of Lp(a) levels across diverse Indian populations, spanning healthy individuals, cardiometabolic disease cohorts, and high-risk subgroups. The findings consistently demonstrate a substantial burden of elevated Lp(a) in India. Disease cohorts including CAD, ACS, MI, stroke, diabetes, and FH showed uniformly higher Lp(a) levels, with a large proportion exceeding clinically important thresholds. Even among healthy or non-diseased Indian adults, nearly one-third exhibited Lp(a) concentrations >30 mg/dL, [4] suggesting a high background prevalence that likely contributes to India's disproportionately early and aggressive manifestation of ASCVD. This finding is particularly concerning in light of recent Indian expert consensus recommendations, which emphasize Lp(a) levels >30 mg/dL as clinically significant and should be considered as a CV risk-enhancing factor during risk assessment [11]. However, comparative Indian data demonstrating a higher ASCVD event risk at this threshold than in other populations are currently lacking [11].

Importantly, while Lp(a) levels between 30 and 50 mg/dL are often considered an intermediate risk category, a substantial proportion of Indian patients exceeded the more widely accepted high-risk threshold of 50 mg/dL (Table 2). In the studies included in this review, 34% of patients with CAD/ACS,[70] 35.7% of patients with ASCVD,[20] and 39.5–43% of patients with familial hypercholesterolemia had Lp(a) concentrations >50 mg/dL [29,48]. These observations suggest that a considerable proportion of Indian patients with established or inherited CV risk conditions carry markedly elevated Lp(a)-associated risk. These findings highlight a substantial burden of both elevated and markedly elevated Lp(a) among Indian populations, including apparently healthy individuals. This underscores the critical need for broader Lp(a) screening in both healthy and high-risk individuals, as many high-risk individuals remain unidentified until the occurrence of a CV event.

As outlined in Figure 2, the majority of contemporary guidelines recommend once-in-a-lifetime measurement of Lp(a) [71-83]. In addition, all major guidelines advocate Lp(a) testing in individuals with premature CV events, while several also recommend testing in those with recent or recurrent ASCVD events and in individuals with a family history of CV events. Collectively, these recommendations support a targeted yet scalable approach to Lp(a) assessment, where identification of elevated Lp(a) may meaningfully refine risk stratification and guide intensification of preventive strategies.

Approximately 90% of the interindividual variability in plasma Lp(a) levels is explained by genetic variation within the LPA gene locus [84]. South Asian populations, including Indians, have higher frequency of small apo(a) isoforms, which are associated with markedly elevated plasma Lp(a) and increased atherothrombotic risk [85,86]. This genetic predisposition explains the high baseline Lp(a) levels observed in Indian populations and the substantial burden identified in the present review. Importantly, as Lp(a) levels are not much influenced by diet, physical activity, or conventional lifestyle interventions, dependence on lifestyle modification alone is not sufficient to reduce Lp(a)-mediated CV risk [87,88]. This highlights the need to incorporate Lp(a) measurement into routine CV risk assessment and lipid evaluation, enabling more accurate risk stratification, and informed therapeutic

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decision-making, particularly in populations with a high prevalence of elevated Lp(a).

In the present study, elevated Lp(a) was strongly associated with various CVDs, which is consistent with published literature. A recent meta-analysis by Tian et al. (2024), which included studies from diverse populations worldwide, demonstrated that high Lp(a) levels significantly increased the risk of ASCVD (odd ratio [OR]: 2.15) and CAD (OR: 2.44) [89]. The risk of premature CAD with elevated Lp(a) was even greater in the South Asian cohort (OR:3.71) [89]. Another systematic review and meta-analysis of studies from diverse populations worldwide found that elevated Lp(a) in patients with existing ischemic heart disease increased the risk of MI, major adverse CV events (MACEs), and death [90]. These findings further support the adverse prognostic significance of elevated Lp(a) across different populations. Moreover, a linear concentration-response was reported in a meta-analysis by Amiri et al. (2023), where every 50 mg/dL increase in Lp(a) corresponded to a 31% greater risk of CV death in the general population [91].

Emerging evidence supports the clinical relevance of Lp(a) thresholds as low as 30 mg/dL for CV risk stratification. In a large multi-institutional study of Taiwanese individuals, Chen et al. demonstrated that Lp(a) levels ≥30 mg/dL independently predicted adverse CV outcomes irrespective of baseline ASCVD status [92]. Lp(a) ≥30 mg/dL was associated with a significantly increased risk of MACE (adjusted subdistribution hazard ratio [aSHR]: 1.24; 95% CI: 1.07–1.43) in ASCVD-free individuals and also in patients with established ASCVD (aSHR: 1.36; 95% CI: 1.07–1.74) [92]. In the present review, several Indian studies reported a substantial proportion of patients with Lp(a) levels >30 mg/dL across high-risk clinical groups, including CAD/ACS (32%-70%), [4,21,30,39] MI (15.83%), [26] diabetes (26.4%), [59] and dyslipidemia (40%) [53], as summarized in Table 2. These findings suggest that the 30–50 mg/dL range may be clinically relevant in Indian patients, particularly when interpreted alongside overall CV risk. While, most contemporary international guidelines, including ESC/EAS, consider Lp(a) ≥50 mg/dL as the threshold associated with

Table 3: Risk of bias of observational studies assessed using the NOS scale.

Author (year)	Selection	Comparability	Exposure/Outcome	Total (Out of 9)
Amin et al. (2024)	2	0	2	4
Dudum et al. (2024)	3	0	2	5
Kalaivani et al. (2023)	2	1	3	6
Muheeb et al. (2023)	2	1	3	6
Joseph et al. (2022)	3	0	3	6
Loh et al. (2022)	3	1	3	7
Behera et al. (2019)	2	1	3	6
Wadhwa et al. (2019)	2	1	3	6
Asre et al. (2018)	2	1	2	5
Bansal et al. (2015)	2	1	3	6
Yusuf et al. (2014)	3	2	3	8
Chakraborty et al. (2013)	2	1	3	6
Himabindu et al. (2012)	3	1	3	7
Goswami et al. (2010)	3	1	3	7
Dhamija et al. (2009)	3	1	3	7
Singla et al. (2009)	2	1	3	6
Gambhir et al. (2008)	3	1	3	7
Rajappa et al. (2006)	3	0	3	6
Superko et al. (2005)	4	1	3	8
Rajasekhar et al. (2004)	4	1	3	8
Singh et al. (2004)	4	1	3	8
Angeline et al. (2003)	3	1	3	7
Geethanjali et al. (2003)	4	1	3	8
Tan et al. (2003)	4	1	3	8
Goyal et al. (2002)	2	0	1	3

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Table 4: Risk of bias of cross-sectional studies assessed using the JBI appraisal scale.

JBI tool	Domain 1	Domain 2	Domain 3	Domain 4	Domain 5	Domain 6	Domain 7	Domain 8
Sandhu et al. (2024)	Y	Y	Y	NA	N	N	Y	Y
Mahto et al. (2022)	Y	Y	Y	Y	N	N	Y	Y
Nissen et al. (2022)	Y	Y	Y	Y	N	N	U	Y
Rohit et al. (2020)	Y	Y	N	NA	N	N	Y	Y
Chaudhary et al. (2017)	Y	Y	NA	Y	N	N	U	Y
Mukherjee et al. (2015)	Y	N	U	U	N	N	N	Y
Sontakke et al. (2014)	Y	Y	NA	NA	NA	NA	Y	Y
Ashfaq et al. (2013)	Y	Y	Y	Y	N	N	Y	Y
Chandini et al. (2012)	N	Y	NA	Y	N	N	Y	Y
Banerjee et al. (2011)	Y	Y	U	U	N	N	Y	Y
Chopra et al. (2007)	Y	Y	Y	NA	N	N	Y	Y
Mahajan et al. (2004)	Y	Y	Y	NA	N	N	Y	Y
Tavridou et al. (2003)	Y	Y	NA	NA	N	N	Y	Y
Velmurugan et al. (2003)	Y	Y	Y	NA	N	N	Y	Y
Deepa et al. (2002)	Y	Y	Y	NA	N	N	Y	Y
Palaniaapan et al. (2002)	Y	Y	Y	Y	N	N	Y	Y
Hoogeveen et al. (2001)	Y	Y	Y	Y	N	N	Y	Y
Ramachandran et al. (2001)	Y	Y	Y	Y	N	N	Y	Y

Note: *N: No; NA: Not applicable; U: Unclear; Y: Yes.*

increased ASCVD risk, recent Indian expert opinion has suggested that Lp(a) levels >30 mg/dL may be clinically relevant for cardiovascular risk assessment, particularly in high-risk individuals [8-11]. Therefore, these observations suggest that Lp(a) levels between 30 and 50 mg/dL may warrant greater attention in Indian populations and may support inclusion of Lp(a) into CV risk assessment frameworks in Indian population; however, prospective Indian outcome studies are needed to determine whether this lower threshold independently predicts ASCVD events or warrants population-specific risk reclassification.

Lp(a) is recognized as a major contributor to residual CV risk despite achieving target LDL-C levels with current lipid-lowering therapies [93]. Evidence suggests that elevated Lp(a) is an independent and additive risk for ASCVD even in patients receiving intensive statin therapy and achieving recommended LDL-C targets [94]. The high prevalence of elevated Lp(a) observed in this review suggests that genetically mediated Lp(a)-related risk may account for a substantial proportion of unexplained CV events, highlighting the need to incorporate Lp(a) assessment into secondary prevention frameworks and comprehensive CV risk reduction strategies.

The reviewed studies also substantiate literature demonstrating that high Lp(a) levels are linked to ischemic stroke. Across all three studies included in this review, Indian patients experiencing a stroke had significantly higher Lp(a) levels (37 – 82.3 mg/dL) than their healthy counterparts (21.0 – 24.4 mg/dL) [35,36,69]. In alignment with this finding, a recent meta-analysis of studies done in Asian and Caucasian populations identified a significant association between elevated Lp(a) concentrations and increased risk of ischemic stroke compared with controls (standardized mean difference [SMD] 0.76; 95% confidence interval [CI] 0.53–0.99) [95]. Smolders et al. (2007), in a pooled analysis of 31 studies, found higher Lp(a) concentrations to be significantly linked with overall stroke risk (SMD:0.39; 95% CI:0.23–0.54) [96]. Similarly, Nave et al. (2015) confirmed the association, reporting an elevated risk of ischemic stroke among individuals with high Lp(a) levels (OR 1.19; 95% CI 1.00–1.41) [97]. However, the overall evidence remains heterogeneous, with some studies and meta-analyses reporting weaker or non-significant associations [98-100]. Differences in ethnicity, stroke subtype, study design, and adjustment for confounding factors may partly explain these inconsistencies. Therefore, while Indian case-control data suggest higher Lp(a) levels in stroke patients, these findings should be interpreted cautiously.

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Recommendations for LP(a) Testing					
Guidelines	**PREMATURE CV EVENTS**	**FAMILY H/O CV EVENTS**	**RECURRENT CV EVENTS**	**ONCE IN LIFETIME**	**RECENT CV EVENTS**
✓ ACC/AHA (2026)	✓	✓		✓	
✓ ESC/EAS FU (2025)	✓	✓		✓	
✓ NLA4* (2024)	✓	✓		✓	
✓ CSI (2024)	✓	✓		✓	
✓ Spain† (2023)	✓	✓	✓	✓	✓
✓ AAS (2023)	✓	✓	✓		✓
✓ BHS (2022)	✓	✓		✓	
✓ EAS (2022)	✓	✓		✓	
✓ NSFA (2021)	✓	✓	✓		
✓ CCS (2021)	✓	✓		✓	
✓ LAI (2020)	✓	✓	✓	✓	✓
✓ AACE/ACE (2020)	✓	✓	✓		
✓ NLA (2019)	✓	✓	✓		
✓ HEART UK (2019)	✓	✓		✓	
✓ ESC/EAS (2019)	✓	✓			
✓ ACC/AHA (2018)	✓	✓			

Figure 2: Guidelines recommendations for Lp(a) Testing [8,9,71-83].

Note: *A check mark indicates that the guideline specifically recommends or supports Lp(a) measurement in the corresponding clinical scenario. Recommendations were interpreted based on explicit statements regarding Lp(a) testing.*

An important limitation of the existing Indian literature on Lp(a) is the underrepresentation of women, with most studies enrolling predominantly male cohorts. Although studies reporting sex-specific data in this review did not demonstrate consistent differences in Lp(a) levels between men and women, these findings must be interpreted cautiously due to limited female sample sizes and the absence of analyses stratified by menopausal status. Lp(a) levels are usually 5%–10% higher in women compared to men [75]. Postmenopausal reductions in estrogen have been associated with further increases in Lp(a), which can be attenuated with hormone replacement therapy [101,102]. These gaps highlight the need for sex-specific and menopause-stratified studies in Indian populations to better define the contribution of Lp(a) to CV risk in women and to inform tailored prevention strategies.

Clinical and Public Health Implications

The consistent elevation of Lp(a) across cardiometabolic diseases in Indians reinforces its role as a key driver of premature and aggressive ASCVD. The increased prevalence of Lp(a) ≥30 mg/dL suggests that routine assessment may help to identify high-risk individuals who would otherwise be missed by using traditional risk factors. However, despite, substantial evidence demonstrating its role in risk reclassification, intensification of lipid-lowering therapy, and identification of individuals at very high residual risk, Lp(a) testing is suboptimal in India. The findings of this review emphasize the importance of incorporating Lp(a) into CV risk profiling in India, where premature and severe CAD is common. ESC/EAS guidelines and the 2019 ACC/AHA primary prevention guideline recommend once-in-a-lifetime Lp(a) testing, particularly in high-risk ethnic groups and those with premature ASCVD [75,103]. Thus, considering the high prevalence observed in this review, routine Lp(a) testing in Indian adults, especially patients with premature ASCVD or a family history, is likely to improve early detection and allow more aggressive risk factor optimization, including intensified LDL-C lowering therapy.

Achieving very low LDL-C targets of <55 mg/dL is especially critical in patients with elevated Lp(a) as Lp(a) is an independent and additive atherothrombotic risk, amplifying plaque burden and residual CV risk, thus making aggressively lowering LDL-C essential to combat the dual threat and prevent CV events [90,104,105]. Moreover, routine Lp(a) testing assumes greater clinical relevance in light of novel, targeted therapies specifically designed to reduce Lp(a). Multiple Lp(a) targeted therapies are currently in advanced phases of clinical development and have demonstrated the ability to achieve substantial and sustained reductions in Lp(a) levels [106]. Hence, Lp(a) testing will ensure timely access to Lp(a)-targeted treatments as they become clinically available. Thus, it is imperative for clinicians to change the

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status quo, implement at least once-in-a-lifetime Lp(a) testing in at-risk individuals, use Lp(a) to refine risk assessment, and intensify lipid-lowering and global risk-reduction strategies in those with elevated Lp(a) to reduce premature CV morbidity and mortality in Indian patients.

Study Limitations and Future Directions

This systematic review has few limitations. First, there was significant variability across studies in the reporting of Lp(a) levels, variable thresholds for defining elevated Lp(a), study design, sample sizes, and population, all of which limit the comparability of findings. Assay heterogeneity is another important limitation of this review. The included studies might have used diverse assay methods for Lp(a) measurement. Furthermore, Lp(a) concentrations were reported using different units (mg/dL, nmol/L, ng/mL), which reflects variability in calibration and reporting practices. As many conventional immunoassays are affected by apo(a) isoform size, such heterogeneity may limit direct comparability of Lp(a) levels across studies, affect prevalence estimates at specific thresholds, and complicate clinical interpretation. These limitations highlight the need for future Indian studies to adopt standardized, isoform-independent assays and uniform reporting units, which would improve comparability, allow more accurate burden estimation, and help in translation of Lp(a) data into clinical practice. Also, we synthesized the findings qualitatively, as a meta-analysis was not feasible due to heterogeneity in the included studies. Finally, most included studies were single-center with relatively small sample sizes, and few accounted for key confounders such as diet, socioeconomic status, or family history, further limiting the robustness of conclusions.

Future research in India should focus on establishing national, multicentre registries to generate representative data on Lp(a) levels across diverse geographic, ethnic, and socioeconomic populations, including rural and underserved communities. There is an unmet need for adoption of standardized, isoform-independent Lp(a) assays with uniform reporting units. Large prospective cohort studies are needed to define the impact of elevated Lp(a) on long-term CV outcomes and residual risk despite optimal LLT. Moreover, integration of Lp(a) into India-specific CV risk prediction models may improve risk stratification and guide preventive strategies tailored to the Indian population.

Conclusion

Indian adults exhibit a high burden of elevated Lp(a) across both healthy and clinical populations, with markedly higher levels in individuals with CAD, MI, stroke, FH, and diabetic complications. Elevated Lp(a) levels correlated with greater disease severity and adverse clinical outcomes, highlighting its role as an independent and clinically relevant CV risk factor. Given the strong genetic determination, prognostic importance, and availability of emerging Lp(a)-lowering therapies, routine Lp(a) testing and risk-stratified management strategies are urgently needed in Indian clinical practice.

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Declaration of Interest Statement/Conflict of Interest

Authors JPS, AO, CKP, DK, NS, GN have no conflict of interest to declare. Authors SC and PK are the employees of Novartis Healthcare Pvt Ltd. Mumbi, India.

Author Contributions

JPS conceptualized and designed the study, conducted the literature search, extracted the data, interpreted the findings, and drafted the manuscript. AO contributed to the literature search, data extraction, and critical revision of the manuscript. CKP assisted with data interpretation and critical revision of the manuscript. DK contributed to data analysis, interpretation of results, and manuscript editing. NS provided critical review of the manuscript, data interpretation and intellectual input. GN contributed to data verification and manuscript review. SC provided methodological input and critically revised the manuscript. PK supervised the study, provided overall guidance, and interpreted the findings. All authors reviewed the manuscript critically for important intellectual content and approved the final manuscript for submission.

Ethical Statement

This study is a systematic review of previously published literature. As the analysis was based solely on data from published studies and did not involve direct interaction with human participants or access to identifiable patient information, ethical approval and informed consent were not required. The review was performed and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

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