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AGE & GENDER BASED LP(A) LEVELS: INDIAN REFERENCE LAB DATABASE

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ABSTRACT

India is experiencing an escalating epidemic of coronary artery disease and Cardiovascular Disease. Acute Myocardial Infarction rates are threefold to fivefold higher in young Indians than in other populations. Lipoprotein(a) (Lp(a)) is increasingly recognized as the strongest known genetic risk factor for premature CAD. Numerous studies have been published on Lp(a), suggesting that south Asians have the second highest Lp(a) levels as compared to other population groups. We planned to review our lab data as we could not find any recent Indian study on Lp(a) levels based on large population. **Objective:** To analyse age and gender-based difference in Lp(a) levels in Indian population along with the lipid profile in different Lp(a) categorises. The present retrospective study is done at Dr. Lal pathlabs. Data of the patients who visited lab for routine health check- up from April 2023 to March 2025 was analyzed for Lp(a) levels and lipid profile. **Results:** In the present study, 37421 individuals underwent lp(a) testing and a subset of 18692 had their concurrent lipid profile assessed. The population was categorised into different groups based on age and gender. When the population was categorised into 4 different groups based on age, high Lp(a) levels were observed at age group >60 years with median Lp(a) value of 27 mg/dL and this was statistically significant as compared with other age group with p <0.001. It was also found that the percentage of individuals in each Lp(a) category irrespective of age is approximately constant. Approximately 26.2% (9816) of population fall in high risk Lp(a) category with median 81 mg/dL. There were 25508 (68.16%) males and 11913 (31.84%) females. Females showed had high levels of Lp(a) as compared to males with median of 25 and 21 respectively and this difference was statistically significant. Present study showed a negative correlation between Triglyceride and Lp(a) levels but a positive correlation was observed between Lp(a) levels and HDL-cholesterol, Total cholesterol and LDL-cholesterol respectively. **Conclusion:** Overall, our study suggests that LP(a) levels are significantly high for patients with >60 years of age. Levels >50mg/dL was seen in 26.2% population which roughly translates into the fact that one out of four Indians has Lp(a) above 50 mg/dL. We even found gender-based difference in Lp(a) levels with Females having high levels of LP(a) in comparison to males. Individuals in high-risk category had significantly high HDL-cholesterol, Total cholesterol and LDL-cholesterol but Low Triglyceride and this was statistically significant with low-risk category.

KEYWORDS: The present retrospective study is done at Dr. Lal pathlabs.

INTRODUCTION

Cardiovascular diseases (CVDs) is a major global health problem and a leading cause of morbidity and mortality, despite availability of advance treatment modality. As per world health organisation data base, an estimate of 17.9 million people died from CVDs in 2016, representing 31% of all global deaths. In 2016 India reported 63%[1] of total deaths due to NCDs, of which 27% were attributed to CVDs. "Lipid Association of India 2023 update on cardiovascular risk assessment and lipid management in Indian patients: Consensus statement IV", it is well recognized that Indians develop ASCVD about a decade earlier than the Western populations, despite having lower levels of low-density lipoprotein cholesterol (LDL-C). It has been reported that more than 50% of coronary artery disease (CAD)-

associated deaths in India occur before the age of 50 years and 25% of myocardial infarctions (MIs) occur before the age of 40 years. CVD is not a single disorder, it is a collective term that representing all types of affliction affecting the blood circulatory system, including the heart and vasculature. Atherosclerosis is one of the crucial risk factor leading to CVD. Although there are several well established risk factors contributing to CAD[2], but they fails to explain the cause of malignant CVD in young Indians.

Elevated Lipoprotein(a) [Lp(a)][3] has been found to be an one of important and independent risk factor that explains the high incidence of malignant CVD in young Indians. Various Epidemiological studies, Mendelian randomization data and genome-wide association data, provide clear support of its association. Lipoprotein(a) is an apoB-containing lipoprotein resembling low-density lipoprotein (LDL), but apolipoprotein B-100 is covalently linked to a unique glycoprotein, called apolipoprotein(a) or apo(a). Apo(a), encoded by the *LPA* gene, is structurally similar to plasminogen, an important protein involved in fibrinolysis. *In vitro*, apo(a) has been shown to inhibit fibrinolysis[4] although the exact function of lipoprotein(a) is still a mystery. Lp(a) was first described by Kare Berg[5] in 1963 and later purified and characterized by Ehnholm[6] and colleagues as part of human lipoproteins.

The levels of lipoprotein(a) are under strong genetic regulation. 80–90% of Lp(a) is genetically determined as an autosomal co-dominant with complete expression by 1–2 years of age. Adult levels are achieved by the age of 5-years which remains constant except in inflammatory states. The levels of Lp(a) varies several hundred-fold[7] in the general population and this variation is predominantly determined by its isoform size and other genetic variants in the *LPA* locus. 90 % of the interindividual variation in plasma Lp(a) has been attributed to the apo(a) gene while 70 %[8] is related to the size of apo(a) isoforms. Numerous studies have been published on Lp(a) that states, that south Asians have highest Lp(a) levels as compared to other population groups such as whites and chinese. They also quote that the prevalence of high Lp(a) in Indians and South Asian population is around 25%[9] but there are no recent studies establishing the same. The available data dates back to early 90's and there is no clarity regarding the sample size as well. *So*, The present study incorporate recent data of last two years to see the age and gender-wise distribution of Lp(a) in Indian population, that too with a fair sample size and thus will help to have a better insight of the trend and may help to formulate population based strategy for Lp(a) estimation.

MATERIALS AND METHODS

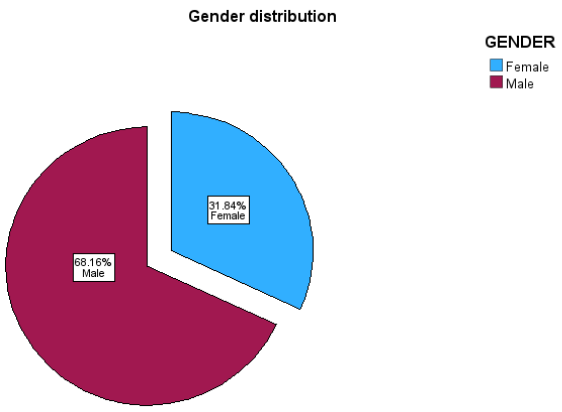
It is a Retrospective study, conducted at Dr. Lal pathlabs. The past data of 2 years spanning April 2023 to March 2025 was reviewed for Lp(a) levels and lipid profile, of the patients who visited lab for routine health check-up during this period. There were total 37,421 participants in this study. While all 37421 individuals underwent lp(a) testing, a subset of 18692 also had their concurrent lipid profile assessment.

Serum samples (both fasting and non-fasting) were analyzed for Lp(a) and other lipid parameters using the Siemens Atellica CH analyzer. Lp(a) concentrations were quantified using an isoform-insensitive immunoturbidimetric method to ensure accuracy independent of apolipoprotein(a) size. The assay demonstrated robust precision, with an intra-assay coefficient of variation (CV) of 1.5% and an inter-assay CV of 3.0%.

Data were analyzed using IBM SPSS Statistics version 30. Due to the non-normal distribution of Lp(a) levels, non-parametric statistical methods were employed. The Kruskal-Wallis test was used for comparisons across multiple groups, and the Mann-Whitney U test was applied for pairwise comparisons. Statistical significance was assessed using mean rank scores to account for the skewed distribution of the data. Pvalue <0.05 was considered as significant.

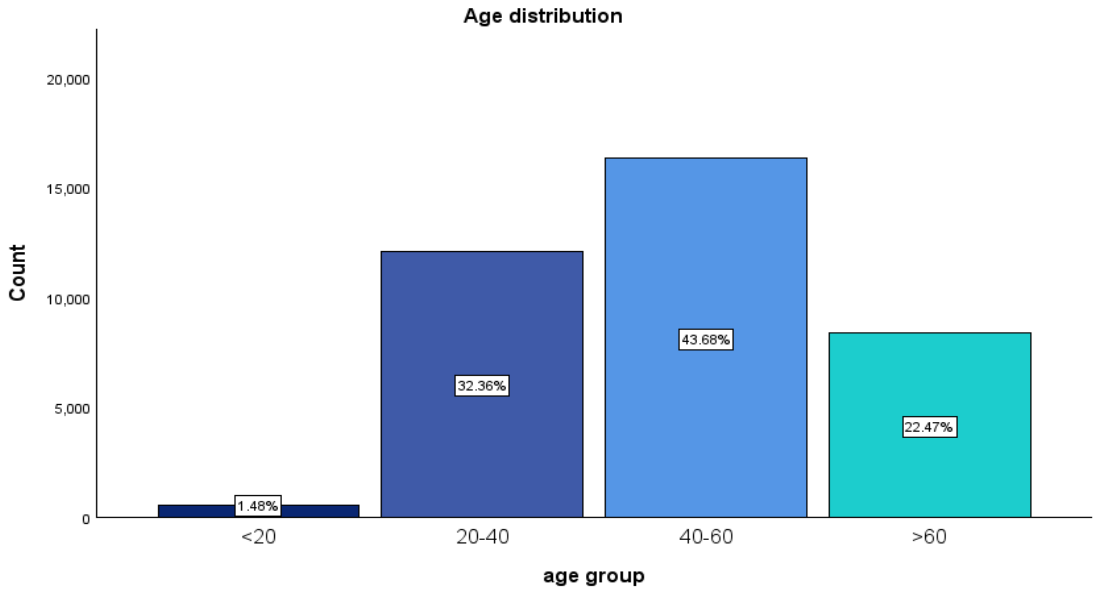
RESULTS

In the present study, 37,421 patients underwent Lp(a) analyzed. The population was categorised into different groups based on gender and age, comprising of 25508 (68.16%) males and 11913 (31.84%) females (Graph 1).



Graph 1: Gender based distribution.

The study population was divided into 4 different age-based groups. The distribution included 555 (1.5 %) participants in < 20 years age group, 12111 (32.4 %) participants in 20 - 40 years of age group, 16345 (43.7 %) in 40 – 60 years and 8410 (22.5 %) in > 60 years (Graph 2).



Graph 2: Age based distribution of total population.

There is no generalized consensus on Lp(a) risk thresholds. However, ≥50 mg/dL is an accepted target in American College of Cardiology/American Heart Association (ACC/AHA) guidelines, Canadian Cardiovascular Society (CCS) guidelines as well as in Lipid Association of India (LAI) guidelines). The same has been as a risk-enhancing cut-off in the National Lipid Association (NLA) scientific statement.[10,11]

So, in the present study Lp(a) levels were stratified into 3 categorizes: low-risk (< 30 mg/dL), moderate risk (30 – 50 mg/dL) and high-risk (>50 mg/dL). 59% (22079) individuals fall in a low-risk group with the median value of 11 mg/dL, 14.8% (9816) fall in a moderate risk category with the median value of 38 mg/dL and 26.3% (5526) fall in a high-risk category with the median value of 81 mg/dL. Overall, median was 22 mg/dL with 26.3% of Indian population in high risk category.

Table 1: Categorisation based on Lp(a) levels.

Lp(a) categories	Frequency	Percent	Median (mg/dL)
Low-risk	22079	59.0%	11
Moderate - risk	9816	14.8%	38
High-risk	5526	26.3%	81
Total	37421	100.0%	22

The distribution of patients across Lp(a) risk categories by age is summarized in Table 2. In the age group of <20 years, 332 (59.8%) patients were in the low-risk category (<30 mg/dL), 76 (13.7%) in the moderate-risk category (30–50 mg/dL), and 147 (26.5%) in the high-risk category (>50 mg/dL). For patients aged 20–40 years, 7,513 (62.0%) had low-risk levels, while 1,749 (14.4%) and 2,849 (23.5%) were in the moderate and high-risk groups, respectively. In the 40–60 years group, 9,715 (59.4%) were low risk, 2,318 (14.2%) moderate risk, and 4,312 (26.4%) high risk. Finally, among participants >60 years, 4,519 (53.7%) were low risk, 1,383 (16.4%) were moderate risk, and 2,508 (29.8%) were high risk. High Lp(a) levels were observed in age group >60 years. It was statistically significant with p <0.001.

Table 2: Study population categorization into 4 groups based on age.

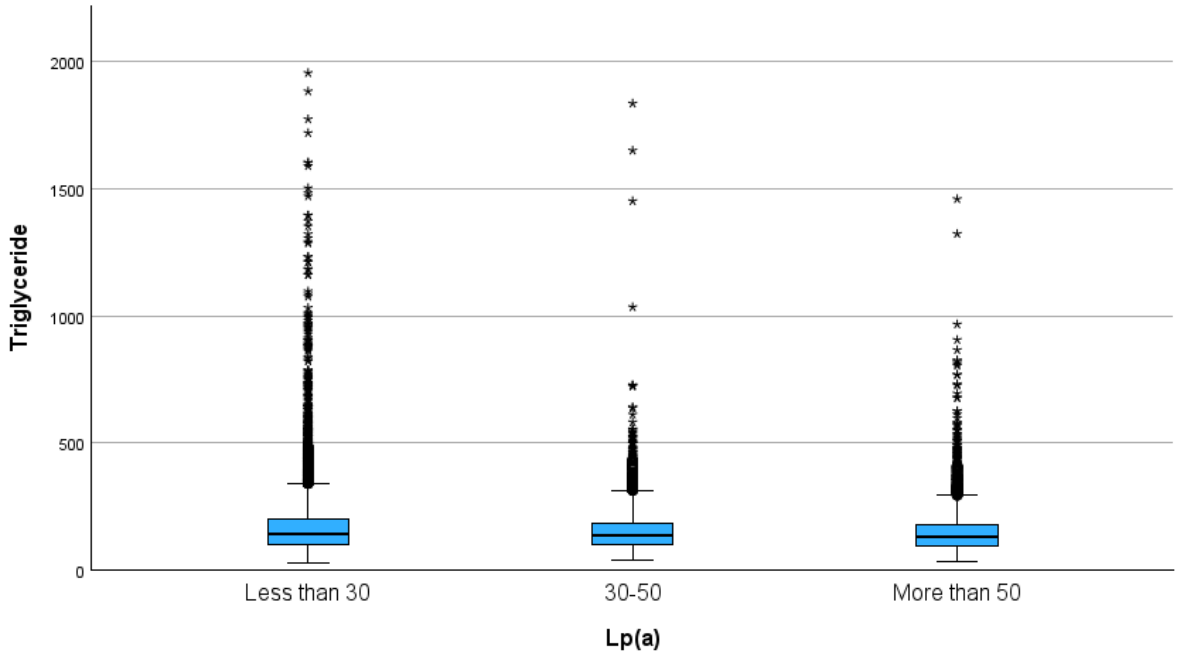
Age groups (years)	Lp(a) categories	Frequency	Percent	Median(Lp(a)) (mg/dL)
<20	Low-risk category	332	59.8	10.2
	Moderate-risk category	76	13.7	37.8
	High-risk category	147	26.5	78.4
	Total	555	100	21
20- 40	Low-risk category	7513	62	11

	Moderate-risk category	1749	14.4	38
	High-risk category	2849	23.5	79.5
	Total	12111	100	20
40- 60	Low-risk category	9715	59.4	11.4
	Moderate-risk category	2318	14.2	38
	High-risk category	4312	26.4	81
	Total	16345	100	22
> 60	Low-risk category	4519	53.7	12.1
	Moderate-risk category	1383	16.4	38.3
	High-risk category	2508	29.8	83
	Total	8410	100	27

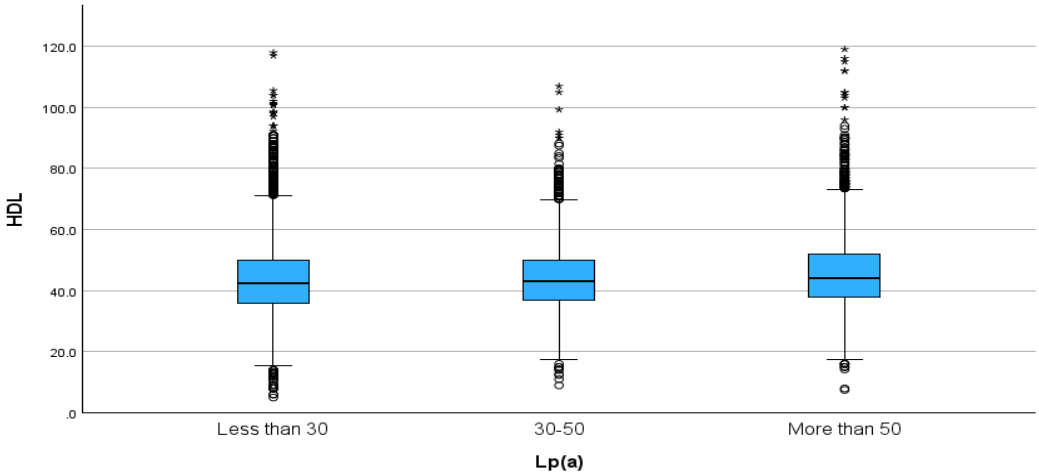
In the present study, the number of female patients in each Lp(a) category are, 6627 (55.6%) in Low-risk category, 1807 (15.2%) in Moderate risk category, 3479 (29.2%) in High-risk category whereas males are, 15452 (60.6%) in Low-risk category, 3719 (14.6%) in Moderate risk category, 6337 (24.8%) in High-risk category. Females had high median LP(a) levels as compared to males with median of 25 mg/dL v/s 21 mg/dL. This difference is statistically significant (P <0.05).

Table 3: Gender based Lp(a) levels.

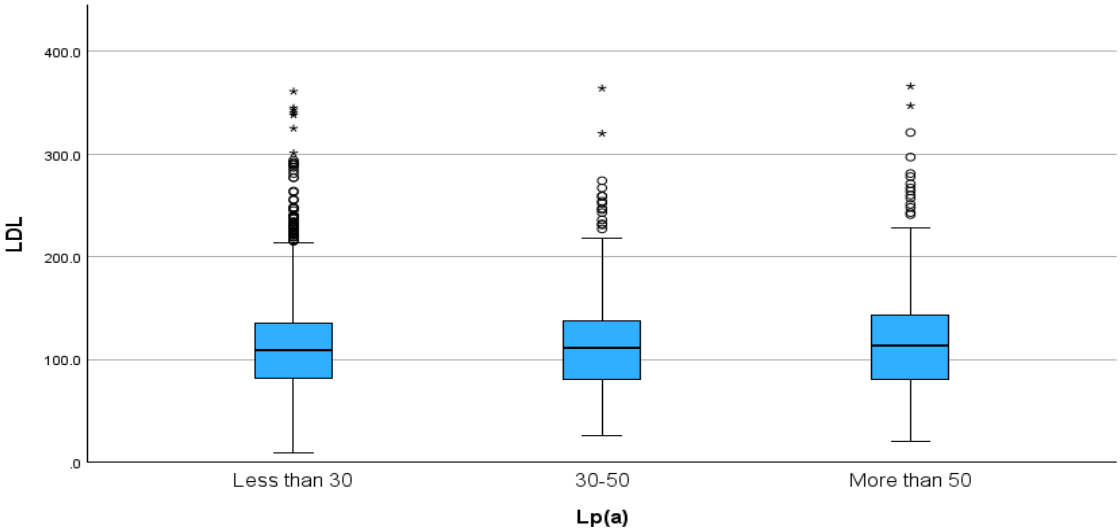
GENDER	Lp(a) categories	Frequency	Percent	Median (Lp(a)) (mg/dL)
Female	Low-risk category	6627	55.6	12
	Moderate- risk category	1807	15.2	38.21
	High-risk category	3479	29.2	83.35
	Total	11913	100	**25**
Male	Low-risk category	15452	60.6	11
	Moderate - risk category	3719	14.6	38
	High-risk category	6337	24.8	79.92
	Total	25508	100	**21**



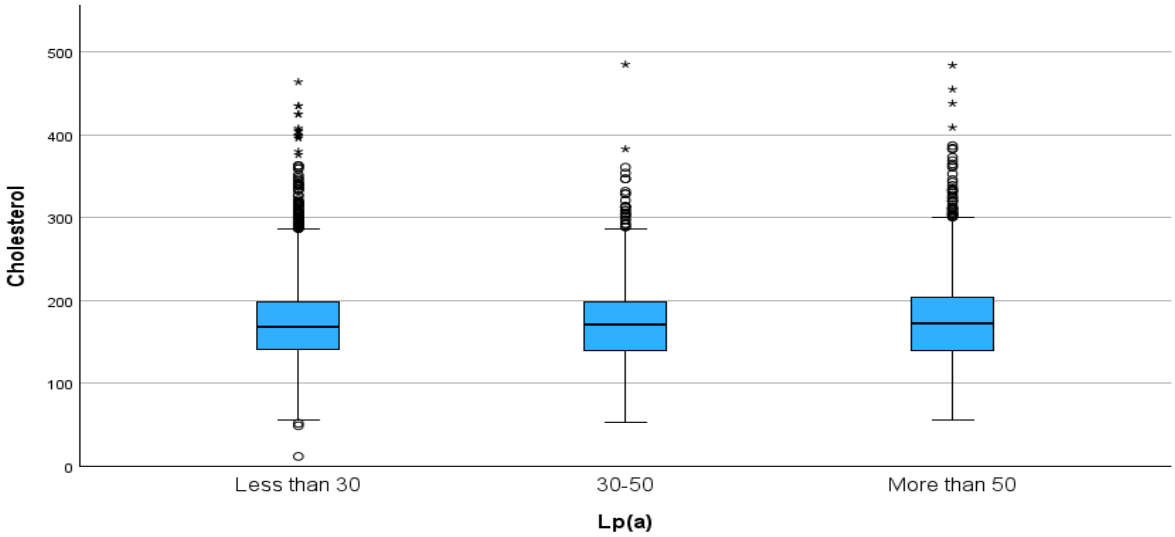
Graph 3: Triglyceride level across Lp(a) group.



Graph 4: HDL level across Lp(a) groups.



Graph 5: LDL levels across Lp(a) groups.



Graph 6: Cholesterol levels across Lp(a) groups.

Of the 37421 total participants, a subset of 18692 (49.9%) underwent a full lipid profile assessment. Within these participants, 11333 (60.6%) were from low-risk category, 2741(14.7%) were from moderate- risk category and 4618 (24.7%) were from high risk category.

Table 4: Comparison of lipid profile with different Lp(a) risk categories.

Lp(a) group	Mean (TG)	Mean (HDL)	Mean (LDL)	Mean(CHOL)
Low Risk	169.12	43.87	109.64	171.11
Moderate Risk	155.90	44.19	110.61	171.26
High Risk	148.34	45.52	113.49	174.63

Post-hoc pairwise comparisons between different lipid profile parameters and all Lp(a) groups after Bonferroni adjustment. High-risk group showed significantly lower Median of TG compared to both moderate-risk (p=0.002) and low-risk group (p <0.001). Additionally, moderate-risk group also differed significantly from Low risk Lp(a) group (p <0.001). For HDL, no significant difference was found between low-risk group and moderate- risk groups. However, High-risk group Lp(a) group had significantly higher median values than the other 2 groups. Both low-risk group and moderate-risk group had significantly low median values for LDL and CHOL as compared to the high-risk group. No significant difference was found between low-risk group and moderate-risk groups for LDL and CHOL.

Table 5: Post-hoc pairwise comparisons of lipid profile parameters between Lp(a) groups.

Comparison	P (TG)	P (HDL)	P (LDL)	P (Cholesterol)
<30 vs (30-50)	0.002	0.543	0.878	1.000
< 30 vs > 50	<0.001	0.00	0.00	0.001
30-50 vs > 50	<0.001	0.00	0.27	0.026

DISCUSSION

The epidemiology of atherosclerotic cardiovascular disease (ASCVD) in India is considerably different from that in Western countries. Considering the causal relationship of Lp(a) with ASCVD and the earlier age of ASCVD onset in Indians, it becomes even more important to have a clear understanding of trend of Lp(a) in our population. In the present study, when the population was categorised into 4 different groups based on age, high Lp(a) levels were observed at age group >60 years with median Lp(a) of 27 mg/dL (Table 2) and this was statistically significant with p <0.001. This may be due to influence of Non-genetic factors on lipoprotein(a) levels such as Hormone therapy, Chronic kidney disease, Treatment of overt hyperthyroidism with thyroidectomy, Reduction of saturated fatty acid intake and Hemodialysis.[12] It was also found that the percentage of individuals in each Lp(a) category irrespective of age is approximately constant (Table 2, which represents that the levels of Lp(a) are largely genetically determined.

Percentage of individuals in high risk Lp(a) category

In the present study, 26.2% (9816) of population fall in high risk Lp(a) category with median 81 mg/dL (Table1). Many studies supports the finding of >26% of Indian population having lp(a) >50mg/dL. Enas et al. were the first to report high Lp(a) levels among Indians residing in the US. According to him, Lp(a) levels ≥30 mg/dl were found in 25% and >20 mg/dl in 44% of Indians. In the MASALA (Mediators of Atherosclerosis in South Asians Living in America) cohort, median lipoprotein(a) levels in South Asian individuals were higher than in White, Hispanic, and Chinese individuals. Similar results were found in the study conducted by Tapan Ghosh, where he found that higher numbers of Indian and South Asian population, as high as 25% has Lp(a) levels >50mg/dL.[9]

Level of Lp (a) in female vs male

In the present study, Females showed high levels of Lp(a) as compared to males with median of 25 and 21 respectively and this difference is statistically significant (Table 3). It may be due to reduced estrogen in postmenopausal women, which may results in elevation of lipoprotein(a) up to 13%. Estrogen is a negative regulator of *LPA* as Estrogen response element was identified 26 kb upstream of the apolipoprotein(a) promoter.[13] This finding was supported by the study conducted by SV Annabelle et al. who found that Lipoprotein(a) concentrations are generally 5% to 10% higher in women than men. Virani SS et al. ARIC (Atherosclerosis Risk in Communities) study also showed that median Lp(a) levels were significantly higher in women versus men in both Black and White populations. Zeis Tm et al. also found higher proportion of females had elevated Lp(a) levels than males in his cohort. This difference was greatest at higher Lp(a) levels (>90 mg/dL).

Lipid profile parameters between different Lp(a) groups

Present study showed that mean of Triglyceride (TG) decreases with the increase in Lp(a) levels i.e High Lp(a) occurs with normal or low triglycerides. This inverse correlation between Lp(a) and TG among individuals with hypertriglyceridemia was first described by Bartens et al.[14] Large number of studies showed the similar correlation. But a study conducted by Marco-Benedí et al showed that negative correlation exists only when TG transcend >300mg/dL.[15] In order to find out the possible

reasons for this correlation a study was conducted by Gaubatz JW where he found a positive correlation between TG and the lipoprotein(a)–triglyceride-rich lipoprotein (Lp(a)–TRL) complexes and concluded that Lp(a)–TRL complex are easily metabolised in compare to isolated Lp(a).[16] other possible explanation suggested by Ramos-Cáceres et al.,[17] is that larger VLDL particles, apoE in VLDL, and VLDL refinement with TG provides a negative feedback in Lp(a) synthesis.

But same is not true with other lipid parameters. HDL-cholesterol showed positive correlation with Lp(a) levels. These findings are supported by the study conducted by I Andrikou et al.[18] where he found that Lp(a) $\geq$ 30 mg/dL had statistically significant high HDL in comparison to those with Lp(a) <30mg/dL. They also found that individuals with high Lp(a) levels were at high risk of developing CAD risk despite of having high HDL i.e good cholesterol which indicates that high Lp(a) levels attenuate the shielding effect of HDL. This could be explained by high systemic and vascular inflammation mediated by elevated Lp(a), which leads to the transformation of HDL to a malfunctioning form, eliminating its protective effects against atherosclerosis.[19]

LDL and Cholesterol showed positive correlation with Lp(a). Several studies supported the positive correlation of LDL with Lp(a)[20,21] while Saeedi R et al[22] and other such as Li KM et al[23], Bustanji Y et al.[24] showered contradictory results. A study conducted by Saxena R et al. on paediatric age group showed that children with family history of CAD had higher Lp (a) and Cholesterol when compared with group of children whose family do not had CAD history.[25]

CONCLUSION

Overall, our study suggests that LP(a) levels are significantly high for patients with >60 years of age. Levels >50mg/dL was seen in 26.2% population which roughly translates into the fact that one out of four Indians has Lp(a) above 50 mg/dL. We even found gender-based difference in Lp(a) levels with Females having high levels of LP(a) in comparison to males. Individuals in high-risk category had significantly high HDL-cholesterol, Total cholesterol and LDL-cholesterol but Low Triglyceride and this was statistically significant with low-risk category.

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