JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

Assessment of Lipoprotein(a) Levels Among Acute Myocardial Infarction (AMI) Patients

Author: Dr Gitika P patil

Co author: Dr Sudhir L Tungikar

Dr Pranav Kothari

Introduction: Myocardial infarction (MI) is an emerging public health concern, with rising incidence linked to both lifestyle and genetic factors. Lipoprotein(a) [Lp(a)], a genetically determined lipid particle, is now recognized as a significant, independent risk factor for atherosclerotic cardiovascular disease and MI, affecting about 20% globally. Despite its strong association with premature MI, effective Lp(a)-lowering therapies are lacking, and guidelines remain unclear.

Duration of study: one year

Objective:

· To determine the distribution and mean levels of Lp(a) among AMI cases.

· To analyze the correlation between elevated Lp(a) levels and clinical severity or type of AMI.

Material and methods:

This 1-year prospective study was conducted in the Department of Medicine with 50 participants. After obtaining informed consent, data were collected through medical history, clinical examination, ECG, 2D Echo, cardiac enzymes, coronary angiography, and laboratory investigations including lipid profile and serum Lp(a) levels. The aim was to aassess the serum levels of Lipoprotein(a) [Lp(a)] in patients diagnosed with acute myocardial infarction. Data analysis was performed using SPSS and OpenEpi, with statistical significance set at $p < 0.05$. Chi-square and t-tests were applied for categorical and continuous variables, respectively.

Results and observation

Thus total 50 cases having acute myocardial infarction were studied and following results were observed. The mean age was 51.7 years and predominance of males was seen. They had higher BMI and a more atherogenic lipid profile—elevated LDL, triglycerides, fasting blood sugar, and lower HDL. Mean LPa was 55.4 mg/dL. Lp(a) levels >50 mg/dL were strongly associated with CAD.

Discussion

Study by Vandana Saini et al [54] showed that mean age among cases was 59.4 and mean LPa among cases was 51.4 and Mean LPa in present study was 55.4 mg/dL. Study by Mehdi Afshar et al [52] showed that mean age was 49 years. .

Conclusion

Elevated Lp(a) was identified as a raised for AMI cases, frequently overlooked in standard lipid profiles. Early screening can help detect high-risk individuals and improve prevention, particularly among young Indians.

Keywords: Lipoprotein A Lp(a), Acute Myocardial Infarction (AMI)

Introduction

The rising incidence of myocardial infarction (MI) among younger individuals is becoming a major public health concern. Traditionally considered a disease of older adults, MI is increasingly being observed in people below the age of 45, driven by both lifestyle-related and genetic risk factors. This early-onset or "premature" MI, defined as MI occurring before the age of 55 in men and 65 in women, has prompted renewed interest in understanding non-traditional cardiovascular risk markers. One such marker is lipoprotein(a) [Lp(a)], a lipoprotein similar to LDL but containing an additional apolipoprotein(a) component. Elevated Lp(a) levels are predominantly genetically inherited and are now recognized as one of the most common inherited lipid disorders, affecting approximately 1 in 5 individuals globally.

Multiple studies have confirmed that Lp(a) is not just associated with, but also plays a causal role in the pathogenesis of atherosclerotic cardiovascular diseases, including myocardial infarction and aortic valve stenosis. Despite its clinical significance, there are currently limited therapeutic interventions specifically aimed at lowering Lp(a) levels, and no universally accepted clinical guidelines exist for its management. As a result, physicians often manage patients with high Lp(a) by controlling other modifiable cardiovascular risk factors such as LDL cholesterol, hypertension, smoking, and diabetes.

Importantly, there is still a lack of comprehensive data examining the interplay between Lp(a) and traditional cardiovascular risk factors, especially in younger populations. Young patients presenting with acute coronary syndrome (ACS) often fall outside conventional risk prediction models, and their risk may be underestimated. Understanding how elevated Lp(a) interacts with factors like family history, obesity, hypertension, and smoking can help clinicians identify vulnerable individuals who may benefit from early intervention.

Objective:

· To determine the distribution and mean levels of Lp(a) among AMI cases.

· To analyze the correlation between elevated Lp(a) levels and clinical severity or type of AMI.

Material and methods

This one-year prospective study was carried out in the Department of Medicine and included 50 participants. Informed consent was obtained from all subjects prior to enrollment. Data collection involved a comprehensive approach including detailed medical history, clinical examination, ECG, 2D echocardiography, cardiac enzyme assessment, coronary angiography, and laboratory tests such as lipid profile and serum lipoprotein(a) [Lp(a)] levels. The primary objective was to evaluate serum Lp(a) concentrations in patients diagnosed with acute myocardial infarction. Statistical analysis was conducted using SPSS and OpenEpi software, with a p-value of <0.05 considered statistically significant. Appropriate tests, including the chi-square test for categorical variables and the t-test for continuous variables, were applied.

Lp(a) Assay

Lp(a) measurements were performed in our hospital chemistry laboratory by using standard methods and reagents (Incstar Co) according to the supplier's package insert. In brief, this is an automated immunoprecipitation procedure that uses a monospecific goat antibody to Lp(a). The coefficient of variation is 13% for Lp(a) levels , 10 mg/dL and 2.5% for Lp(a) levels . 60 mg/dL. Values , 5 mg/dL are undetectable. Short-term post–myocardial infarction increases in Lp(a),19 believed to be only transient, return to normal

levels within 1 month.32 In our patients, the median time between a coronary event and presentation to our clinic was 10 months.

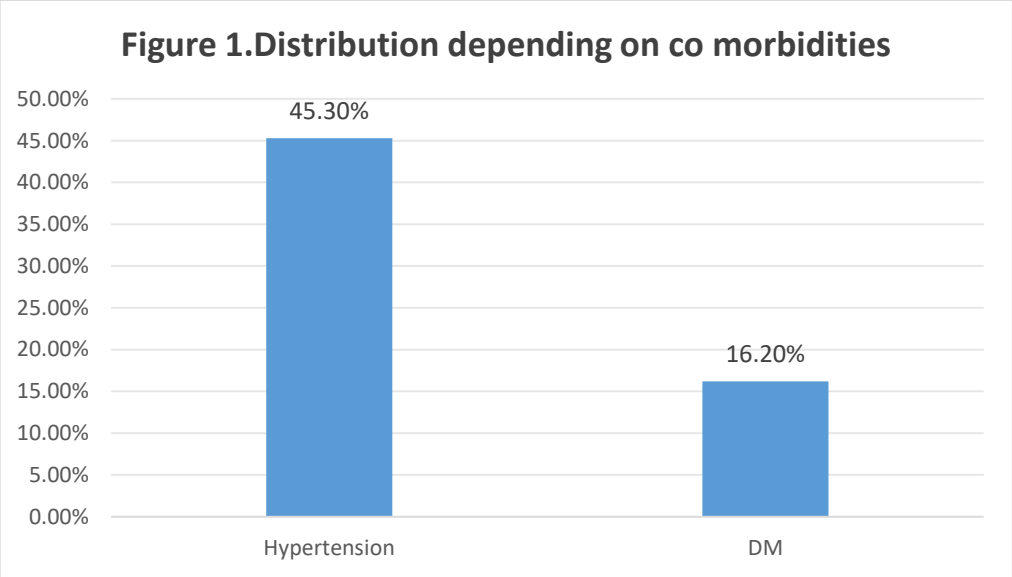
Results

Total 50 cases were studied having Acute Myocardial Infarction (AMI) and following findings were seen.

Table 1: Distribution depending on age

Parameters	Age in years	BMI	Lp(a)
Mean	51.7	28.9	55.4
SD	3.6	2.5	2.3

The mean age of the cases was 51.7 years (SD 3.6) and mean BMI was 28.9 (SD 2.5). The mean Lipoprotein(a) [Lp(a)] level among the study participants was 55.4 mg/dL, with a standard deviation of 2.3 mg/dL, indicating elevated average levels in the cohort, suggestive of increased cardiovascular risk.

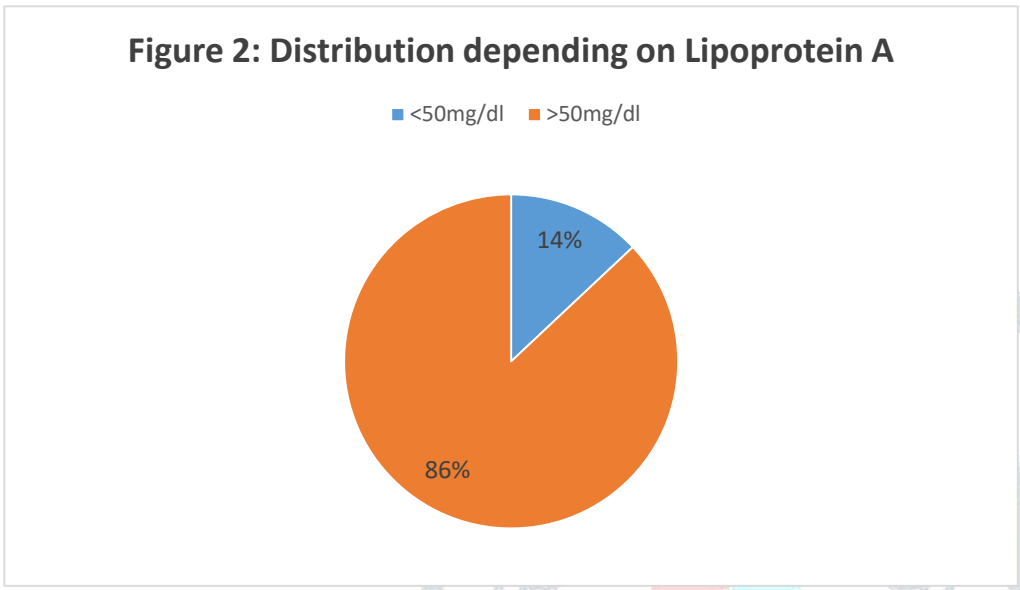


45.3% had Hypertension and 16.2% had Diabetes Mellitus.

2.Distribution depending on biochemical test findings

Biochemical test findings	Mean
Total cholesterol	167.3
LDL	118.7
HDL	22.5
Triglyceride	138.4
FBS	105.3
Urea	32.8
Creatinine	0.81
Uric acid	3.2

The biochemical profile of the study participants revealed a mean total cholesterol level of 167.3 mg/dL and an LDL level of 118.7 mg/dL, indicating moderately elevated low-density lipoprotein values. The mean HDL level was notably low at 22.5 mg/dL, suggesting an increased cardiovascular risk. Triglyceride levels averaged 138.4 mg/dL, while fasting blood sugar (FBS) was 105.3 mg/dL, pointing toward borderline glycemic status. Renal function tests showed a mean urea level of 32.8 mg/dL and creatinine at 0.81 mg/dL, both within normal limits, and the mean uric acid level was 3.2 mg/dL, which is also considered normal.



The pie chart in Figure 2 illustrates the distribution of study participants based on their serum Lipoprotein(a) [Lp(a)] levels. A significant majority—86%—had elevated Lp(a) levels exceeding 50 mg/dL, while only 14% had levels below 50 mg/dL. This suggests that high Lp(a) levels were prevalent among the participants, indicating a potential association between elevated Lp(a) and the occurrence of myocardial infarction in this cohort.

3. Distribution depending on Coronary Angiography among cases

Coronary Angiography	Frequency	Percentage
Occlusion	18	36
Stenosis	20	40
Multi vessel disease	13	26
Plaque	12	24

As cases had one or more than one finding on angiography, among them majority cases had stenosis(40%). Coronary angiography findings in the study revealed that stenosis was the most common abnormality, observed in 40% of the participants, followed by occlusion in 36%. Multi-vessel disease was present in 26% of cases, indicating more extensive coronary involvement. Additionally, plaque formation was identified in 24% of individuals, highlighting the atherosclerotic burden contributing to coronary artery disease in this population.

4. Correlation between LPa and final diagnosis values among young MI cases

Parameters	LPa		Total
	<50	>50	
Within normal limits	5	6	11
Severe	2	37	39
Total	7	43	50

In the present study, among participants with Lipoprotein(a) [Lp(a)] levels <50 mg/dl, 5 cases were within normal limits and 2 presented with severe disease. Conversely, among those with elevated Lp(a) levels >50 mg/dl, only 6 were within normal limits, while a significant majority—37 individuals—had severe disease. This indicates a strong association between elevated Lp(a) levels and the severity of clinical presentation.

Parameter	Estimate	Lower - Upper 95% CIs	Method
Sensitivity	71.43%	(35.89, 91.78[1])	Wilson Score
Specificity	86.05%	(72.74, 93.44[1])	Wilson Score
Positive Predictive Value	45.45%	(21.27, 71.99[1])	Wilson Score
Negative Predictive Value	94.87%	(83.11, 98.58[1])	Wilson Score
Diagnostic Accuracy	84%	(71.49, 91.66[1])	Wilson Score

Discussion

In this prospective study of 50 acute myocardial infarction (AMI) cases, the mean age was 51.7 years and mean BMI was 28.9, indicating a middle-aged overweight population. The average Lipoprotein(a) [Lp(a)] level was 55.4 mg/dL, with 86% of patients having levels above 50 mg/dL, suggesting a strong association between elevated Lp(a) and AMI. Comorbid conditions included hypertension in 45.3% and diabetes in 16.2% of participants. Biochemical profiles revealed moderately elevated LDL (118.7 mg/dL) and very low HDL (22.5 mg/dL), alongside normal renal function markers. Coronary angiography showed stenosis in 40%, occlusion in 36%, multivessel disease in 26%, and plaque in 24% of patients, indicating significant atherosclerotic burden. Notably, 37 of 43 patients with Lp(a) >50 mg/dL had severe disease, while only 2 of 7 with Lp(a) <50 mg/dL showed severe presentation, confirming a strong link between elevated Lp(a) and disease severity. Diagnostic performance metrics showed high specificity (86.05%) and negative predictive value (94.87%), making Lp(a) a reliable marker to rule out severe disease, despite moderate sensitivity and PPV.

Study by Vandana Saini et al [54] showed that mean age among cases was 59.4 and mean LPa among cases was 51.4 and Mean LPa in present study was 55.4 mg/dL. Study by Mehdi Afshar et al [52] showed that mean age was 49 years.

Conclusion

Elevated Lp(a) was identified as a raised for AMI cases, frequently overlooked in standard lipid profiles. Early screening can help detect high-risk individuals and improve prevention, particularly among young Indians.

References

1.Nordestgaard BG, Chapman MJ, Ray K, Boren J, Andreotti F, Watts GF, Ginsberg H, Amarenco P, Catapano A, Descamps OS, Fisher E, Kovanen PT, Kuivenhoven JA, Lesnik P, Masana L, Reiner Z, Taskinen MR, Tokgozoglu L, Tybjaerg-Hansen A; European Atherosclerosis Society Consensus Panel. Lipoprotein(a) as a cardiovascular risk factor: current status. Eur Heart J. 2010;31:2844–2853.

2.Kamstrup PR, Tybjaerg-Hansen A, Steffensen R, Nordestgaard BG. Genetically elevated lipoprotein(a) and increased risk of myocardial infarction. JAMA. 2009;301:2331–2339.

3. Thanassoulis G, Campbell CY, Owens DS, Smith JG, Smith AV, Peloso GM, Kerr KF, Pechlivanis S, Budoff MJ, Harris TB, Malhotra R, O'Brien KD, Kamstrup PR, Nordestgaard BG, Tybjaerg-Hansen A, Allison MA, Aspelund T, Criqui MH, Heckbert SR, Hwang SJ, Liu Y, Sjogren M, Van der Pals J, Kalsch H, Muhleisen TW, Nothen MM, Cupples LA, Caslake M, Di Angelantonio E, Danesh J, Rotter JL, Sigurdsson S, Wong Q, Erbel R, Kathiresan S, Melander O, Gudnason V, O'Donnell CJ, Post WS; CHARGE Extracoronary Calcium Working Group. Genetic associations with valvular calcification and aortic stenosis. N Engl J Med. 2013;368:503–512.

4.Brown WV, Ballantyne CM, Jones PH, Marcovina S. Management of Lp(a). J Clin Lipidol. 2010;4:240–247.

5.Davidson MH, Ballantyne CM, Jacobson TA, Bittner VA, Braun LT, Brown AS, Brown WV, Cromwell WC, Goldberg RB, McKenney JM, Remaley AT, Sniderman AD, Toth PP, Tsimikas S, Ziajka PE, Maki KC, Dicklin MR. Clinical utility of inflammatory markers and advanced lipoprotein testing: advice from an expert panel of lipid specialists. J Clin Lipidol. 2011;5:338–367.

6.Vandana Saini, M K Bhatnagar and Jayashree Bhattacharjee. Evaluation of Homocysteine, Lipoprotein(a) and Endothelin as diagnostic markers of Coronary Artery Disease in Indian population. Internet Journal of Medical Update. 2013 January;8(1):17-23

7. Mehdi Afshar, Louise Pilote, Line Dufresne, James C. Engert, George Thanassoulis. Lipoprotein(a) Interactions With Low-Density Lipoprotein Cholesterol and Other Cardiovascular Risk Factors in Premature Acute Coronary Syndrome (ACS). J Am Heart Assoc. 2016;5:e003012