ORIGINAL ARTICLE

Correlation of Conventional and Extended Lipid Profiles with Plaque Burden in Statin-naïve Patients with Acute Coronary Syndrome: A Prospective Observational Study from South India

Mughilan Periasamy[1], Ramu Ramadoss[2*], Avinash Anantharaj[3], Balasubramaniyan Vairappan[4]

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Abstract

Background: Traditional lipid parameters like low-density lipoprotein (LDL), high-density lipoprotein (HDL), and total cholesterol (TC) are commonly used in evaluating cardiovascular risk. Recently, emerging biomarkers such as apolipoprotein B (ApoB) and apolipoprotein A1 (ApoA1) are proposed to provide improved accuracy in assessing atherosclerotic risk. This study examined the association between conventional and novel lipid parameters and plaque burden in statin-naïve acute coronary syndrome (ACS) patients.

Methodology: We enrolled 81 statin-naïve patients with ACS. Each underwent both standard and extended lipid profiling. Coronary angiograms were evaluated using the Gensini score to quantify plaque burden. All participants were followed for 28 days to monitor for major adverse cardiac events (MACE).

Results: The average age was 51 years, with males comprising 77%. The ST-segment elevation myocardial infarction (STEMI) was observed in 58% of cases, non-ST-segment elevation myocardial infarction (NSTEMI) in 31%, and unstable angina in 11%. There was a significant correlation between the Gensini score and TC/HDL ratio ($r = 0.35$), LDL/HDL ratio ($r = 0.31$), and ApoB levels ($r = 0.24$). LDL and the ApoB/ApoA1 ratio did not exhibit significant associations with plaque burden. STEMI patients had higher LDL/HDL and TC/HDL ratios compared to those with NSTEMI or unstable angina. MACE occurred in 16% of participants, with no significant difference across ACS subtypes.

Conclusion: The ratios of TC/HDL, LDL/HDL, and ApoB levels were positively associated with coronary plaque burden. While conventional lipid parameters continue to serve well in cardiovascular risk assessment (CRA), ApoB presents a promising standalone marker for identifying atherogenic risk and may serve as a practical alternative in clinical practice.

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Introduction

Cardiovascular diseases account for 31% of deaths worldwide and 27% of deaths in India.[1] The incidence of acute coronary syndrome (ACS) is rising in the Indian population and has started to occur at a younger age. On the contrary, the resources for percutaneous interventions remain limited. Smoking, diabetes, dyslipidemia, and obesity are the modifiable risk factors for atherosclerotic cardiovascular diseases. Of these, dyslipidemia is often overlooked.[2]

Cholesterol is carried through the bloodstream by lipoproteins. Very low-density lipoprotein (VLDL) is responsible for delivering triglycerides and cholesterol from the liver to various body tissues. In the process, it gradually loses triglycerides and cholesterol, transforming first into intermediate-density lipoprotein (IDL) and eventually into low-density lipoprotein (LDL). High-density lipoprotein (HDL) helps protect against atherosclerosis by facilitating reverse cholesterol transport—carrying cholesterol from peripheral tissues back to the liver.[3] To evaluate dyslipidemia, fasting lipid profile parameters such as LDL, HDL, VLDL, and total cholesterol (TC) are commonly assessed. Additionally, lipid ratios like LDL/HDL, TC/HDL, and non-HDL/HDL are useful indicators for identifying individuals at increased risk of cardiovascular diseases.

The protein component of lipoprotein is called apolipoprotein. Apolipoprotein B (ApoB) is the major apolipoprotein in LDL. An LDL particle can carry a variable amount of cholesterol but has only one ApoB. ApoB is present in IDL and VLDL as well. So, measuring ApoB levels gives a better estimate of the number of atherogenic lipoproteins.[4] Conversely, apolipoprotein A1 (ApoA1) is the primary apolipoprotein in the antiatherogenic HDL.[5] The ApoB/ApoA1 ratio reflects the balance between lipoproteins that promote atherosclerosis and those that protect against it. An elevated ApoB/ApoA1 ratio is associated with an increased risk of acute coronary events.[6,7] However, evidence regarding its ability to predict the severity and extent of atherosclerosis remains limited. Lipoprotein(a) [Lp(a)] is a modified form of the LDL particle, distinguished by the presence of an additional protein, apolipoprotein(a), which enhances its atherogenic potential.[8]

Coronary angiography (CAG) is done to diagnose and treat ACS patients. Based on the location and percentage of coronary artery stenoses identified on CAG, the Gensini score is calculated, and it quantifies the extent of myocardial injury.[9] This scoring system provides better information on the severity of atherosclerosis rather than classifying it as single-, double-, or triple-vessel disease.

This study aimed to find whether these conventional and newer lipid parameters correlate with Gensini scores in ACS patients.

Methodology

The study was carried out from October 2022 to July 2024 after obtaining clearance from the Institutional Ethics Committee. We included ACS patients aged 18–65 years who were admitted to the departments of Medicine or Cardiology and were scheduled for CAG. Patients with known heart, kidney, or liver disease, as well as pregnant patients, were excluded. Since statin therapy affects lipid parameters earlier than coronary artery plaque, this study excluded patients who had been on statin therapy for >1 month.[10]

[1]Postgraduate; [2]Associate Professor, Department of Medicine; [3]Associate Professor, Department of Cardiology; [4]Additional Professor, Department of Biochemistry, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India; *Corresponding Author

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Data Collection

Demographic information of the patients was recorded, and clinical history included detailed questions related to the symptoms and risk factors associated with ACS. All patients underwent a comprehensive clinical examination and baseline laboratory investigations, including hemograms, renal and liver function tests. Electrocardiogram (ECG) findings, cardiac biomarkers (troponin T or CK-MB), and echocardiogram reports were also collected.

Lipid Profile

A fasting lipid profile (TC, LDL, HDL, VLDL, and triglyceride) was performed within 48 hours of admission. In addition, a 5 mL fasting blood sample was collected within 48 hours, centrifuged at 3,000 rpm for 15 minutes to isolate the serum, which was then stored at −80°C. These stored serum samples were later analyzed to determine levels of ApoB, ApoA1, and Lp(a) using enzyme-linked immunosorbent assay (ELISA) kits.

Gensini Score Calculation

Once the study participants underwent CAG as part of routine care, their angiographic data were collected. A cardiologist assessed the location and percentage of stenoses in coronary arteries and collateral circulation.[9,11] Based on these findings, a severity score was allotted to each lesion, and the total score was obtained by adding the severity scores assigned to each coronary lesion.

Follow-up

All patients were monitored throughout their hospital stay and followed up for up to 28 days to monitor major adverse cardiac events (MACE).

Sample Size

We assumed that if a lipid parameter caused a 10% variation in the Gensini score, it was considered clinically significant. For this, the correlation coefficient (*r*) had to be 0.32. Assuming a 95% confidence level and 80% power, the estimated sample size required to detect a correlation significantly different from zero was 74. Assuming a 10% attrition rate, 81 participants were recruited in this study.

Statistical Analysis

Depending on the distribution of the data, continuous variables were expressed as mean ± standard deviation (SD) or as median with interquartile range (IQR). Correlation analyses were carried out to explore the relationship between the Gensini score and lipid markers, including the LDL/HDL ratio, TC/HDL ratio, Lp(a), ApoB, and the ApoB/ApoA1 ratio. Differences in the ApoB/ApoA1 ratio across patients with unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI) were evaluated using analysis of variance (ANOVA).

Definitions

ST-segment Elevation Myocardial Infarction

ST-segment elevation myocardial infarction is defined as "new ST-segment elevation at the J-point in at least two adjacent leads. The threshold is ≥1 mm in all leads except V2 and V3. In leads V2–V3, the cutoff is ≥2 mm in men aged 40 years or older, ≥2.5 mm in men younger than 40, and ≥1.5 mm in women."[12]

Non-ST-segment Elevation Myocardial Infarction

Non-ST-segment elevation myocardial infarction is defined as "the presence of horizontal or downsloping ST-segment depression of ≥0.5 mm in at least two contiguous leads, or T-wave inversion of >1 mm with a prominent R wave or an R/S ratio >1, along with elevated cardiac biomarkers indicating myocardial injury."[12]

Unstable Angina

Unstable angina is diagnosed "when there is chest pain at rest lasting >20 minutes, new-onset angina that significantly restricts physical activity, or worsening angina that is more frequent, prolonged, or occurs with less exertion compared to previous episodes. It may be accompanied by significant ST-segment depression and T-wave inversion but without a rise in cardiac biomarkers."[12]

Gensini Score

A severity score is assigned based on the percentage of stenoses and adjusted according to collaterals. This severity score is multiplied by a factor based on stenosis location. The total Gensini score is obtained by adding the severity scores assigned to each coronary lesion.

Gensini score = [Severity score based on degree of stenosis – adjustment for collaterals] × multiplication factor based on the location of stenosis[11]

Major Adverse Cardiac Events

Recurrent MI, heart failure, cardiogenic shock, cardiac death, and stroke are considered MACE.[13]

Results

Out of 127 ACS patients screened, 81 were eligible for enrollment. An overview of the study design is presented in Figure 1. The mean age was 51 years, with males comprising 77% of the study population. The proportion of STEMI, NSTEMI, and unstable angina was 58, 31, and 11%, respectively. Table 1 summarizes the baseline characteristics and compares various clinical and laboratory parameters across various types of ACS.

The mean LDL cholesterol level was 116 ± 36 mg/dL, with similar values among STEMI, NSTEMI, and unstable angina groups. LDL level >130 mg/dL is considered the cutoff for high-risk CAD.[14] Based on this cutoff, 30% of the patients had dyslipidemia. The mean LDL/HDL ratio was 2.7 ± 0.7, which was significantly higher in the STEMI population than the other two types. However, there was no statistically notable difference in ApoB, ApoA1 levels, or the ApoB/ApoA1 ratio across all three groups. Lp(a) levels were not normally distributed and did not show significant changes among STEMI, NSTEMI, and unstable angina. Lp(a) was found to be >50 mg/dL in 74% of the patients, and 12% had Lp(a) levels exceeding 180 mg/dL.

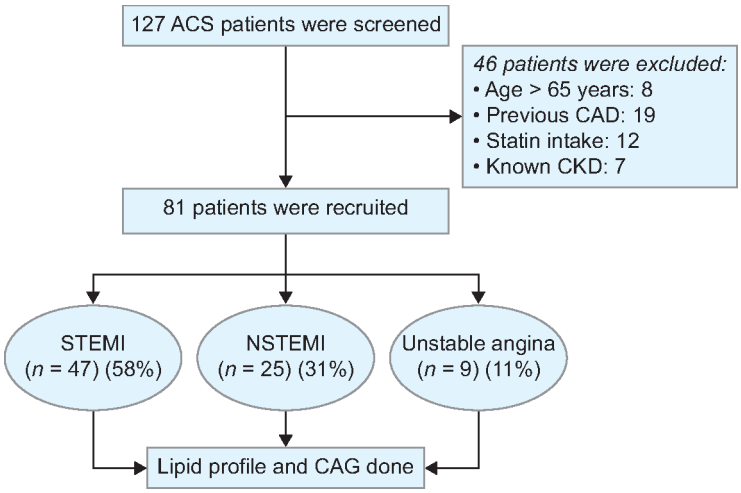

Fig. 1: Study procedure

Table 1: Baseline characteristics of the study population

Baseline characteristics	*All ACS, (n = 81)*	*STEMI, (n = 47)*	*NSTEMI, (n = 25)*	*Unstable angina, (n = 9)*	*p-value*
Number of participants, *n* (%)	81	47 (58)	25 (31)	9 (11)	<0.01
Age in years, mean ± SD	51 ± 8.5	52 ± 8	49 ± 10	50 ± 6	0.48
Male sex, *n* (%)	62 (77)	40 (85)	17 (68)	5 (56)	0.08
Smoking, *n* (%)	43 (53)	30 (64)	10 (40)	3 (33)	0.07
Alcohol consumption, *n* (%)	38 (47)	25 (53)	11 (44)	2 (22)	0.23
Hypertension, *n* (%)	31 (38)	19 (40)	8 (32)	4 (44)	0.73
Diabetes mellitus, *n* (%)	33 (41)	20 (43)	7 (28)	6 (67)	0.12
Family history of CAD, *n* (%)	1 (1.2)	1 (2)	0	0	0.70
Height (cm), mean ± SD	161 ± 8	162 ± 7	160 ± 8	160 ± 10	0.55
Weight (kg), mean ± SD	65 ± 9	66 ± 10	66 ± 8	64 ± 10	0.83
BMI >25 kg/m^2, *n* (%)	37 (46)	20 (43)	12 (48)	4 (44)	0.91
TC (mg/dL), mean ± SD	183 ± 50	188 ± 52	180 ± 51	170 ± 41	0.58
LDL (mg/dL), mean ± SD	116 ± 36	121 ± 38	113 ± 38	102 ± 23	0.28
HDL (mg/dL), mean ± SD					
Males	41 ± 8	42 ± 8	39 ± 7	46 ± 10	0.14
Females	46 ± 12	47 ± 15	44 ± 11	50 ± 10	
TC/HDL, mean ± SD	4.4 ± 0.96	4.48 ± 0.93	4.45 ± 0.97	3.61 ± 0.97	0.04
LDL/HDL, mean ± SD	2.7 ± 0.7	2.9 ± 0.7	2.78 ± 0.64	2.18 ± 0.56	0.02
ApoB (mg/dL), mean ± SD	69.7 ± 23	70.4 ± 23.7	70.5 ± 23.6	63.8 ± 18.8	0.72
ApoA1 (mg/dL), median (IQR)	90.4 (73.8, 109.1)	104.4 ± 66.9	87.8 ± 25.8	90.3 ± 16.3	0.42
Lp(a) mg/dL, median (IQR)	88 (48, 146)	70 (39, 172)	104 (58, 139)	102 (73, 136)	0.99
ApoB/ApoA1 ratio, mean ± SD	0.79 ± 0.3	0.78 ± 0.38	0.83 ± 0.28	0.71 ± 0.18	0.66
Median time to CAG, in days, median (IQR)	7 (2,15)	4 (1,9)	8 (3,20)	24 (23,26)	<0.01
Gensini score, median (IQR)	25 (13.5, 44)	26 (16, 48)	27.5 (15.5, 40)	4.5 (0, 41)	0.23

ACS, acute coronary syndrome; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; BMI, body mass index; CAD, coronary artery disease; CAG, coronary angiogram; HDL, high density lipoprotein; IQR, interquartile range; LDL, low density lipoprotein; Lp(a), lipoprotein(a); NSTEMI, non-ST-segment elevation myocardial infarction; SD, standard deviation; STEMI, ST-segment elevation myocardial infarction; TC, total cholesterol

Table 2: Spearman correlation coefficients between various lipid parameters and the Gensini score

Independent variable	*Spearman coefficient (r)*	*r-squared*	*p-value*
TC/HDL	0.35	0.12	<0.01
LDL/HDL	0.31	0.10	<0.01
Age	0.25	0.06	0.02
ApoB	0.24	0.06	0.03
Lp(a)	0.23	0.05	0.04
LDL	0.17	0.03	0.12
ApoB/ApoA1	0.14	0.02	0.21
ApoA1	0.11	0.01	0.31
HDL	–0.12	0.01	0.27

ApoA1, Apolipoprotein A1; ApoB, apolipoprotein B; HDL, high-density lipoprotein; LDL, low-density lipoprotein; Lp(a), lipoprotein(a); TC, total cholesterol

Moreover, thrombolytic therapy was administered to 39% of STEMI patients (*n* = 32).

Correlation Analysis

Lipid ratios and Gensini scores exhibited a nonnormal distribution. Correlation analysis was done by comparing the Gensini score with each lipid parameter. Spearman correlation coefficients are given in Table 2 in descending order, and corresponding scatter plots are depicted in Figure 2. TC/HDL, LDL/HDL, age, ApoB, and Lp(a) had a statistically significant positive correlation with Gensini score, whereas other markers, like LDL, HDL, ApoA1, and the ApoB/ApoA1 ratio, did not show a significant correlation.

Multiple Linear Regression

Age, the LDL/HDL ratio, and ApoB levels showed statistically significant positive correlations with the Gensini score (r = 0.25, 0.31, and 0.24, respectively). Based on these findings, two multiple linear regression analysis models were proposed. In the first model, age and the LDL/HDL ratio were included as independent variables, with the Gensini score as the dependent variable. The second model included age and ApoB as covariates, with the Gensini score as the outcome variable. The regression coefficients with their confidence intervals are listed in Tables 3 and 4. Both models produced the same adjusted R-squared value of 0.14 (p < 0.01).

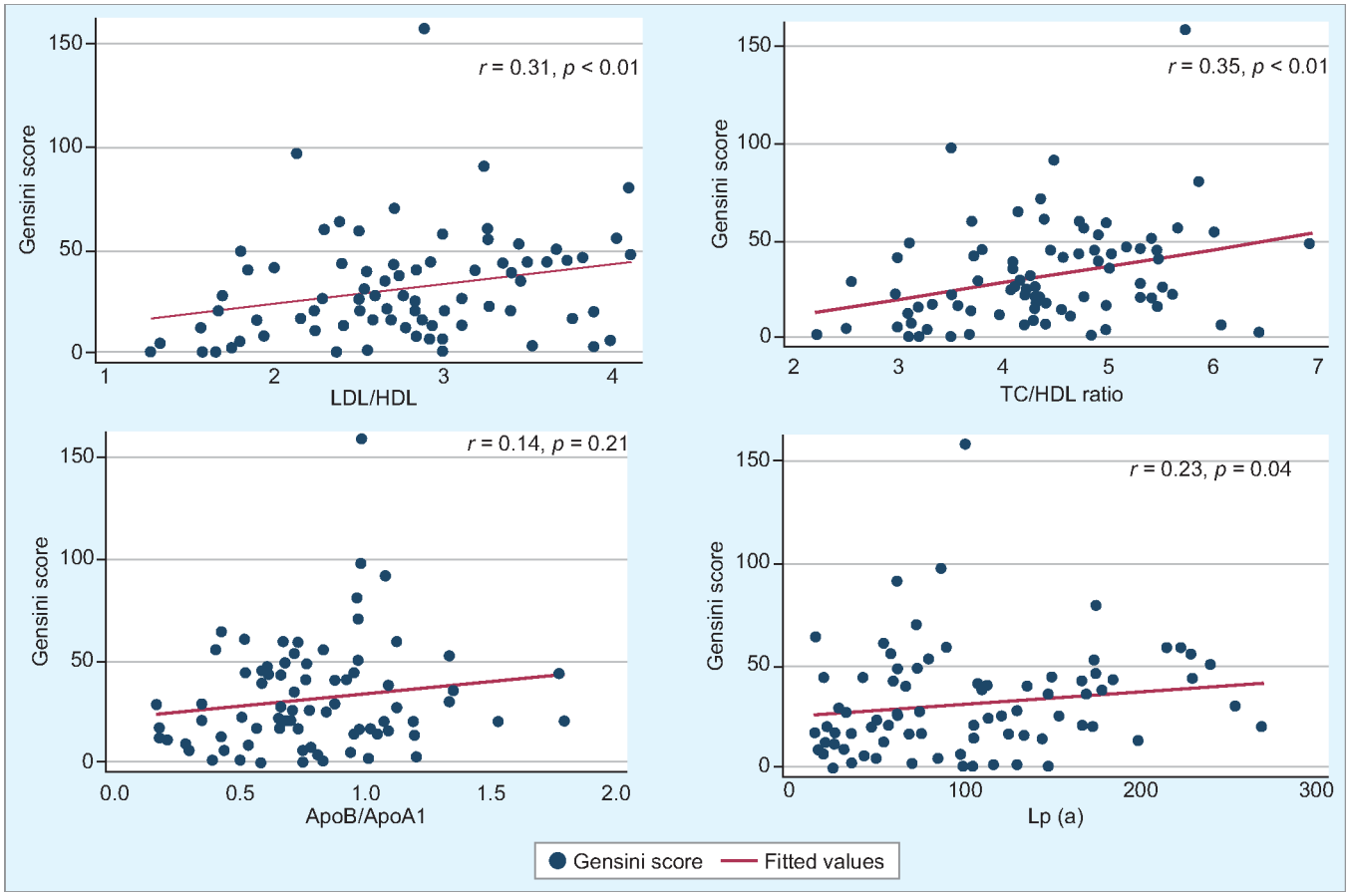

Fig. 2: Correlation of Gensini score with LDL/HDL, TC/HDL, ApoB/ApoA1, and Lp(a); ApoB, apolipoprotein B; HDL, high-density lipoprotein; LDL, low-density lipoprotein; Lp(a), lipoprotein(a); TC, total cholesterol

Table 3: Multiple regression analysis of Gensini score with age and LDL/HDL ratio (adjusted R squared = 0.14, $p < 0.01$)

Variable	*Regression coefficient*	*95% confidence interval*	*p-value*
Age	0.91	0.28, 1.55	<0.01
LDL/HDL ratio	10.43	2.66, 18.21	<0.01

HDL, high-density lipoprotein; LDL, low-density lipoprotein

Table 4: Multiple regression analysis of Gensini score with age and ApoB (adjusted R squared = 0.14, $p < 0.01$)

Variable	*Regression coefficient*	*95% confidence interval*	*p-value*
Age	1.03	0.39, 1.68	<0.01
ApoB	0.32	0.09, 0.56	<0.01

ApoB, apolipoprotein B

Clinical Outcome

Thirteen participants (16%) experienced a MACE either during their hospital stay or within the 28-day follow-up period. The observed MACE included cardiogenic shock (6%), acute decompensated heart failure (4%), recurrent MI (5%), and cardiac death (1.2%).

Discussion

This study found that TC/HDL and LDL/HDL ratios had a better correlation with the Gensini score than ApoB. In contrast, LDL and the ApoB/ApoA1 ratio showed very weak correlations. These results suggest that conventional lipid markers alone can predict the severity of ACS. However, ApoB also predicts the severity of ACS and has the advantage of being a single, reliable lipid parameter for atherogenesis. ApoA1 did not correlate with the Gensini score, and it does not seem to have a role in cardiovascular risk assessment (CRA), consistent with findings from previous studies.[6,15,16]

About 25% of the general Indian population have Lp(a) levels >50 mg/dL, and these levels are genetically determined.[8] Lp(a) level above 180 mg/dL is considered to carry a cardiovascular risk equivalent to that of heterozygous familial hypercholesterolemia.[7] In this study, 74% of the participants had Lp(a) levels >50 mg/dL and 12% had levels >180 mg/dL. A positive correlation was also observed between Lp(a) levels and the Gensini score ($r = 0.23, p < 0.05$). This means that the prevalence of elevated Lp(a) is higher among ACS patients. In a case-control study involving 208 participants at a tertiary care center in South India,

Table 5: Comparison of correlation coefficients of lipid parameters with Gensini score

Variables	*Our study, India, 2024*	*Mashayekhi et al., Iran, 2014*[18]	*Du et al., China, 2016*[19]	*Yaseen et al., Egypt, 2021*[15]	*Siallagan et al., Indonesia, 2023*[20]
Sample size	81	160	380	90	76
LDL/HDL	0.31*	–	0.11	–	–
TC/HDL	0.35*	–	0.10	–	–
LDL	0.17	–	–	–	0.27*
ApoB	0.24*	0.13*	–	0.32*	0.29*
ApoA1	0.11	0.02	–	–0.14	–
ApoB/ApoA1	0.14	–	0.18*	0.73*	–

**p*-value was <0.05; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; HDL, high density lipoprotein; LDL, low density lipoprotein; TC, total cholesterol

Geethanjali et al. reported elevated Lp(a) levels in patients with angiographically confirmed coronary artery disease compared to healthy controls.[17] This could be a reason for the higher prevalence of atherosclerotic heart diseases observed in younger people of South Indian ethnicity.

To the best of our knowledge, this is the first study from India to quantitatively examine the association between lipid profile components and coronary plaque burden using the Gensini scoring system. Correlation coefficients from different studies done in different populations are summarized in Table 5. ApoB consistently showed a positive correlation with the Gensini score. Notably, a single study from Egypt reported a strong correlation for the ApoB/ApoA1 ratio ($r = 0.73$, $p < 0.05$).[15]

The buildup of lipid particles in the arterial wall plays a central role in the development of atherosclerosis. Two mechanisms of lipid deposition have been proposed based on the cholesterol model and the ApoB particle model.[21] According to the cholesterol model, the amount of cholesterol carried within LDL and VLDL determines atherosclerosis. In contrast, the ApoB particle model proposes that the development of atherosclerosis is primarily influenced by the quantity of ApoB-containing lipoprotein particles. As these particles increase in size and cholesterol content, their ability to penetrate the arterial wall diminishes. On the contrary, smaller lipoprotein particles with less cholesterol can easily penetrate the vessel wall. The size of the plaque is determined by the number of ApoB-containing lipoprotein particles rather than the amount of cholesterol. The size of the plaque also increases with age because the greater the age, the longer the duration of dyslipidemia.

In this study, LDL/HDL and TC/HDL ratios among STEMI patients were significantly higher compared to NSTEMI and unstable angina groups. But ApoB, ApoA1, and ApoB/ApoA1 ratio did not have this association. STEMI is an occlusive form of ACS. A Mendelian randomization analysis based on the database obtained from the UK Biobank in 2022 also found that non-HDL cholesterol had a better causal relationship than ApoB concentration.[22] This means that cholesterol content rather than the number of lipoprotein particles is associated with a more vulnerable plaque. This is in favor of the cholesterol model of atherosclerosis.

Two multiple linear regression models were proposed for better comparison and are shown in Tables 3 and 4. The initial regression model explored the association between the Gensini score, age, and LDL/HDL ratio, reflecting the cholesterol-based framework of atherosclerosis. The second model assessed the relationship between the Gensini score, age, and ApoB levels, aligning with the ApoB particle hypothesis. In both these models, the adjusted R-squared was 0.14 ($p < 0.01$), which shows that both these models are equivalent and one is not superior to the other.

Statin therapy requires time to stabilize plaque and to increase lumen size, but it decreases LDL and ApoB levels in a shorter time. As a result, previous lipid-lowering therapy can cause a confounding effect on lipid levels. A key strength of this study was the exclusion of individuals with prior statin use. However, all participants received statin therapy after recruitment as part of the standard treatment of ACS. Statins are very effective and significantly improve cardiovascular outcomes. This could be the reason why lipid parameters did not show a significant association with MACE in this study.

In this study, dyslipidemia was the fourth most common modifiable risk factor, following smoking, obesity, and diabetes. This data is comparable to the data from the ICMR-INDIAB-17 study.[23] However, only 30% of the study participants had dyslipidemia, while the remaining patients presented with other risk factors like smoking, diabetes, and hypertension. These risk factors could have caused confounding effects. Also, severely ill patients were not included as they did not undergo CAG due to logistical reasons. Future study should consider including severely ill patients and excluding patients with risk factors other than dyslipidemia. Such study designs will better assess the predictive value of lipid parameters.

The mean ApoB and ApoA1 levels were lower in this study compared to those reported in previous studies.[6] Most earlier studies used the immunoturbidimetry method to estimate ApoB and ApoA1 levels.[20,24,25] The Quebec cardiovascular study used the rocket immunoelectrophoresis method.[26] In contrast, we used the sandwich ELISA method, which offers greater sensitivity and specificity.[27] Thus, we could speculate that the difference in measurement technique may account for the variation in values. Further research is needed to support these methods to predict consistent results among ACS patients.

This study also had a few limitations, including being a single-center study with a short follow-up period and the absence of a control group.

Conclusion

Conventional lipid indices, particularly the TC/HDL and LDL/HDL ratios, demonstrated predictive ability comparable to that of ApoB in assessing the severity of ACS. These easily accessible parameters remain clinically relevant for CRA and guiding the management of dyslipidemia. However, larger, prospective studies are needed to further validate the utility of extended lipid profiles, including ApoB, in evaluating plaque burden and improving risk stratification in ACS patients.

Author Contributions

Authors	*Mughilan Periasamy*	*Ramu Ramadoss*	*Avinash Anantharaj*	*Balasubramaniyan Vairappan*
Conceptualization	✓	✓	✓	✓
Presentation in Scientific Committee and Ethics Committee meeting	✓	✓	–	–
Data collection	✓	✓	✓	✓
Storage and processing of blood samples	✓	–	–	✓
Data analysis	✓	✓	–	–
Writing-original draft	✓	✓	–	–
Editing	✓	✓	✓	✓
Final approval	✓	✓	✓	✓
Agreement to be accountable for work	✓	✓	✓	✓

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Orcid

Mughilan Periasamy https://orcid.org/0009-0009-0296-6697
Ramu Ramadoss https://orcid.org/0000-0002-2913-4649
Avinash Anantharaj https://orcid.org/0000-0003-2426-8813
Balasubramaniyan Vairappan https://orcid.org/0000-0003-1708-4864

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