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Prevalence of Dyslipidemia and the Role of ApoB and hsCRP in Acute Myocardial Infarction: A Comprehensive Analysis

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ABSTRACT

Dyslipidemia significantly contributes to AMI, with ApoB and hsCRP offering potential for improved risk prediction. **Objective:** To determine the prevalence of dyslipidemia and the role of ApoB and hsCRP in acute myocardial infarction in patients presenting to a tertiary care hospital in Lodhran, Punjab. **Methods:** A cross-sectional study was conducted at the Department of Medicine, Shahida Islam Medical College, Lodhran, from May 2023 to November 2023. A total of 187 AMI patients aged 30–90 years were included using non-probability consecutive sampling. Data were collected using structured clinical history forms and laboratory analysis of lipid profiles, ApoB, and hsCRP levels. Dyslipidemia was defined using standard lipid cutoff values. Statistical analysis was performed using SPSS version 25.0, employing Chi-square and logistic regression to explore associations and predictors of dyslipidemia, with a significance level of $p < 0.05$. **Results:** Dyslipidemia was highly prevalent, affecting 74.9% of patients. Hypertension was significantly associated with dyslipidemia (OR = 2.049, p = 0.042), indicating a potential need for combined management strategies. ApoB and hsCRP levels did not show significant differences between dyslipidemic and non-dyslipidemic patients, though total cholesterol and LDL levels were significantly higher in the dyslipidemic group ($p < 0.001$). **Conclusions:** This study revealed a high prevalence of dyslipidemia in AMI patients, with hypertension as a key predictor. While ApoB and hsCRP were not significant discriminators, their roles in cardiovascular risk assessment may complement traditional lipid profiles, supporting personalized management strategies to reduce cardiovascular risk.

INTRODUCTION

Dyslipidemia is a major modifiable risk factor for Cardiovascular Disease (CVD), a leading cause of morbidity and mortality worldwide. This lipid imbalance, significantly elevated Low-Density Lipoprotein Cholesterol (LDL-C) and Apolipoprotein B (ApoB), promotes atherosclerosis, a primary precipitant of Acute Myocardial Infarction (AMI) [1]. The accumulation of ApoB-containing lipoproteins in arterial walls triggers an inflammatory response, driving lesion progression and plaque formation [2]. Recent studies have established ApoB as a crucial marker of cardiovascular disease risk, as it reflects the count of atherogenic particles undetectable by standard lipid profiles [3]. The pathophysiology of AMI includes inflammation, which often coexists with lipid abnormalities. High-Sensitivity C-Reactive Protein (hsCRP), an inflammation biomarker, is independently associated with adverse cardiovascular outcomes [4]. Elevated hsCRP levels indicate systemic inflammation, suggesting that the formation and rupture of atheromatous plaques correlate with AMI events [5]. The integration of hsCRP measurement into cardiovascular risk assessment enhances the evaluation of the inflammatory component of atherosclerosis [6]. ApoB and hsCRP together provide a comprehensive picture of

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cardiovascular risk through their respective lipid and inflammatory mechanisms. This dual assessment offers insights into reducing the incidence of adverse cardiac events in high-risk populations [7]. Lipid-lowering therapies targeting ApoB-rich lipoproteins can dramatically reduce cardiovascular risk in ApoB-rich patients [8]. Similarly, anti-inflammatory interventions targeting hsCRP may serve as promising adjunctive therapies in managing cardiovascular disease [9]. Further research is needed to clarify the significance of ApoB and hsCRP in AMI, particularly in refining risk stratification models and optimizing treatment protocols based on individual risk profiles. These biomarkers could provide novel insights into the contributions of lipid abnormalities and inflammation to atherosclerotic disease, thereby improving clinical outcomes [10]. Although traditional lipid profiles are valuable, they may not fully capture the cardiovascular risk burden in AMI patients. Emerging evidence highlighted the complementary roles of ApoB, as a measure of atherogenic particles, and hsCRP, as an inflammatory marker, in risk stratification. In resource-limited settings, conventional risk stratification tools may not have effectively applied to AMI patients. Assessing these biomarkers alongside conventional lipid levels could enhance risk stratification and guide tailored interventions.

The purpose of this study was to determine the prevalence of dyslipidemia and evaluate the significance of ApoB and hsCRP levels in AMI patients, aiming to improve management strategies for cardiovascular complications in this high-risk population.

METHODS

This cross-sectional study was conducted at the Department of Medicine, Shahida Islam Medical College, Lodhran, between May 2023 and November 2023. A total of 187 patients with AMI, aged 30–90 years, either male or female, were included. The sample size was calculated using OpenEpi, an online epidemiological tool, based on a reported prevalence of dyslipidemia of 60.83%, a 95% confidence level, and a precision of 7% [11]. This method ensured an adequate sample size to detect meaningful associations in the study population. A non-probability consecutive sampling technique was employed. Patients taking lipid-lowering treatments before the study or those with incomplete medical records were excluded. Informed consent was obtained from all patients, and the ethical committee approved the study SIMC/ET.C./10010/23. Demographic data of all the patients were recorded. A comprehensive clinical history including BMI, smoking status, and comorbidities like hypertension, diabetes mellitus, and family history of cardiovascular disease were taken. Blood samples were taken within the first 24 hours of admission, before starting lipid-lowering treatments, and were sent to a laboratory for lipid profile, ApoB, and hsCRP levels. The lipid profile measured total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides, while ApoB levels indicated atherogenic lipoprotein particles, and hsCRP was measured as a marker of systemic inflammation. Each variable was carefully recorded to ensure data integrity and accuracy. Collected data were entered and analyzed using SPSS version 25.0. Mean and SD were calculated for age, total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, ApoB, and hsCRP. For categorical variables like dyslipidemia status (Yes/No), gender (Male/Female), obesity status (Obese/Non-obese), smoking status (Smoker/Non-smoker), hypertension status (Hypertensive/Normotensive), diabetes mellitus status (Diabetic/Non-diabetic), and family history of cardiovascular disease (Present/Absent) were present as frequencies and percentages. The Chi-square test was employed to assess associations between categorical variables. Logistic regression analysis was performed to adjust for potential confounding variables, including ApoB and hsCRP, with a significance level set at $p < 0.05$

RESULTS

A total of 187 AMI patients were included in the study. Smoking status showed a high prevalence of dyslipidemia in both smokers and non-smokers, with 76 out of 102 smokers (74.5%) and 64 out of 85 non-smokers (75.3%) identified as dyslipidemic. The p-value of 0.902 indicated no significant association, suggesting that smoking status may not notably influence dyslipidemia prevalence in this cohort. For hypertension status, dyslipidemia was observed in 76 out of 94 hypertensive patients (80.9%) and 64 out of 93 normotensive patients (68.8%), yielding a p-value of 0.058. Although this result did not reach statistical significance, it suggests a potential trend where hypertensive AMI patients might have a higher predisposition to dyslipidemia. Similarly, diabetes status did not show a significant association with dyslipidemia. Among diabetic patients, 69 out of 90 (76.7%) were dyslipidemic, compared to 71 out of 97 (73.2%) non-diabetic patients ($p = 0.585$), implying no notable difference in dyslipidemia prevalence based on diabetes status. Regarding family history of cardiovascular disease (CVD), dyslipidemia occurred in 79 out of 100 patients (79.0%) with a family history of CVD and 61 out of 87 patients (70.1%) without such history, resulting in a p-value of 0.162. This indicates no significant relationship between a family history of CVD and dyslipidemia. Analysis of gender showed that 66 out of 93 male patients (71.0%) and 74 out of 94 female patients (78.7%) were dyslipidemic, with a p-value of 0.222, suggesting no significant gender influence on dyslipidemia status among AMI patients in this study (Table 1).

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Table 1: Association between Dyslipidemia and Key Clinical Variables in Patients with Acute Myocardial Infarction (n = 187)

Variable Names	Status	Dyslipidemia N (%)	Dyslipidemia N (%)	Total	p-value
Smoking Status	Smokers	26 (25.5%)	76 (74.5%)	102	0.902
	Non-Smokers	21 (24.7%)	64 (75.3%)	85	
Hypertension Status	Hypertensive	18 (19.1%)	76 (80.9%)	94	0.058
	Normotensive	29 (31.2%)	64 (68.8%)	93	
Diabetes Status	Diabetic	21 (23.3%)	69 (76.7%)	90	0.585
	Non-Diabetic	26 (26.8%)	71 (73.2%)	97	
Family History of CVD	Present	21 (21.0%)	79 (79.0%)	100	0.162
	Absent	26 (29.9%)	61 (70.1%)	87	
Gender	Male	27 (29.0%)	66 (71.0%)	93	0.222
	Female	20 (21.3%)	74 (78.7%)	94	
ApoB Status	Normal	45 (25.4%)	132 (74.6%)	x	0.829
	Elevated	2 (22.2%)	7 (77.8%)	9	
hsCRP Status	Normal	13 (19.1%)	55 (80.9%)	68	0.152
	Elevated	34 (28.6%)	85 (71.4%)	119	

Novel markers ApoB and hsCRP were also examined. Among patients with normal ApoB levels, 132 out of 177 (74.6%) were dyslipidemic, while 7 out of 9 (77.8%) with elevated ApoB levels had dyslipidemia, resulting in a p-value of 0.829. For hsCRP, 55 out of 68 patients (80.9%) with normal hsCRP levels and 85 out of 119 (71.4%) with elevated hsCRP levels were dyslipidemic, with a p-value of 0.152. These findings indicate no statistically significant association between dyslipidemia status and either ApoB or hsCRP levels in this AMI cohort, suggesting that while ApoB and hsCRP are relevant cardiovascular markers, they may function independently of dyslipidemia in the context of AMI. Several key differences in clinical and metabolic parameters were noted in comparing patients with and without dyslipidemia. Total cholesterol and LDL cholesterol levels were significantly elevated in the dyslipidemic group, with mean values of 217.91 mg/dL (SD 40.921) for total cholesterol and 141.54 mg/dL (SD 31.376) for LDL, compared to 164.67 mg/dL (SD 27.457) and 110.81 mg/dL (SD 15.027) in the non-dyslipidemic group (both $p < 0.001$). These differences emphasize the substantially higher levels of atherogenic lipids in dyslipidemic patients, aligning with established roles of elevated total and LDL cholesterol in increasing cardiovascular risk and contributing to atherosclerosis. HDL cholesterol levels did not significantly differ between groups, with a mean of 46.53 mg/dL (SD 10.547) in dyslipidemic patients compared to 44.20 mg/dL (SD 11.391) in non-dyslipidemic patients ($p = 0.200$). ApoB and hsCRP levels also did not show significant differences between the groups: ApoB levels were 99.97 mg/dL (SD 19.449) in dyslipidemic patients and 99.27 mg/dL (SD 18.163) in non-dyslipidemic patients ($p = 0.827$), while hsCRP levels were 2.92 mg/L (SD 1.360) in dyslipidemic patients and 3.11 mg/L (SD 1.357) in non-dyslipidemic patients ($p = 0.414$). These findings suggest that while ApoB and hsCRP are valuable indicators of cardiovascular risk, they may act as independent risk factors rather than correlating directly with dyslipidemia status in AMI patients (Table 2).

Table 2: Comparison of Clinical and Metabolic Parameters between Patients with and without Dyslipidemia in Acute Myocardial Infarction

Variable	No Dyslipidemia (Mean ± SD)	Dyslipidemia (Mean ± SD)	p-Value
Age	58.45 ± 17.151	60.04 ± 18.320	0.600
BMI	24.69 ± 3.870	24.87 ± 4.613	0.812
Total Cholesterol	164.67 ± 27.457	217.91 ± 40.921	0.000
LDL	110.81 ± 15.027	141.54 ± 31.376	0.000
HDL	44.20 ± 11.391	46.53 ± 10.547	0.200
Triglycerides	160.91 ± 51.358	146.21 ± 50.981	0.089
ApoB	99.27 ± 18.163	99.97 ± 19.449	0.827
hs-CRP	3.11 ± 1.357	2.92 ± 1.360	0.414

Regression analysis identified hypertension as the only significant predictor of dyslipidemia among AMI patients, with hypertensive patients being twice as likely to have dyslipidemia (OR = 2.049, 95% CI: 1.027–4.086, p = 0.042). This aligns with established evidence linking hypertension to lipid abnormalities, highlighting the importance of managing both conditions to mitigate cardiovascular risk. Other variables, including age, BMI, smoking status, diabetes status, family history of CVD, ApoB, and hsCRP, did not show statistically significant associations with dyslipidemia in this cohort. The model fit was acceptable, with a Hosmer and Lemeshow test p-value of 0.720 and an overall classification accuracy of 74.9%, supporting the reliability of hypertension as an independent predictor in this population (Table 3).

Table 3: Logistic Regression Analysis for Predictors of Dyslipidemia in AMI Patients

Variables	B	S.E.	Wald	df	Significant (p-Value)	Exp (B) (Odds Ratio)	95% CI for Exp (B)
Age	0.007	0.010	0.476	1	0.490	1.007	0.987 – 1.027
BMI	-0.001	0.040	0.000	1	0.987	0.999	0.923 – 1.082
Smoking Status (1)	-0.067	0.353	0.036	1	0.850	0.936	0.469 – 1.868
Diabetes Status (1)	0.102	0.354	0.082	1	0.774	1.107	0.551 – 2.225
Family History of CVD (1)	0.578	0.360	2.570	1	0.109	1.782	0.882 – 3.601
ApoB	0.000	0.009	0.002	1	0.965	1.000	0.982 – 1.018
hs-CRP	-0.100	0.130	0.594	1	0.441	0.905	0.697 – 1.174
Constant	0.324	1.540	0.044	1	0.833	1.383	-

Notes: (1) Indicates the reference category for binary variables. Adjusted Odds Ratios (ORs) are provided with 95% Confidence Intervals (CI).

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DISCUSSION

The comprehensive analysis of dyslipidemia in patients presenting with Acute Myocardial Infarction (AMI) revealed significant findings that both align with and expand upon existing literature. The high prevalence of dyslipidemia observed in the cohort underscores the critical role of lipid abnormalities in the pathogenesis of AMI. The inclusion of biomarkers such as Apolipoprotein B (ApoB) and high-sensitivity C-reactive Protein (hsCRP) aimed to provide novel insights into cardiovascular risk profiles in dyslipidemic AMI patients. Although ApoB levels did not differ significantly between dyslipidemic and non-dyslipidemic patients, ApoB remains a well-recognized marker reflecting the total number of atherogenic particles, as highlighted by Kayani T et al [11]. The absence of significant differences in ApoB levels may suggest that while cholesterol levels are elevated, the atherogenic particle count itself does not vary notably, or that post-AMI metabolic changes influence these results. Similarly, hsCRP, a key inflammatory biomarker, showed no significant differences between groups. Nevertheless, hsCRP's role in cardiovascular risk is well-established, with Khan HA et al., reporting an inverse relationship between hsCRP and HDL cholesterol [12]. This relationship underscores inflammation's role as an independent contributor to cardiovascular risk, rather than solely through lipid dysregulation. Compared with Ali SN et al., who reported a 60.83% prevalence of dyslipidemia in young AMI patients, the higher rate may have suggested an upward trend in dyslipidemia or demographic differences in lipid profiles [13]. However, smoking status did not significantly impact dyslipidemia prevalence in the cohort, contrasting with findings by Iqbal MZ et al [14]. Differences in smoking intensity, genetic predispositions, or population characteristics may contribute to these varied results, indicating that smoking's impact on lipid levels may differ across populations. Morofuji Y et al., have also noted the influence of lifestyle-related factors, which may affect lipid levels independently of smoking in certain cohorts [15]. Hypertension was the only predictor that reached significance in the regression analysis and hypertensive patients were twice as likely to be dyslipidemic (OR = 2.049, p = 0.042) [16]. This association, in turn, underscores the importance of coordinating the treatment of hypertension and lipid abnormalities in AMI patients to contain cardiovascular risk. By comparing these findings of elevated total and LDL cholesterol in dyslipidemic patients with well-described atherogenic patterns associated with greater risk for cardiovascular disease, reflected the well-documented alterations. These findings emphasize the need to monitor these lipid parameters to better control cardiovascular health in AMI patients. In this study, ApoB and hsCRP be statistically significant predictors of dyslipidemia, but remain important risk assessment biomarkers independent of cardiovascular risk. As Kayani T et al., suggested, ApoB's predictive value for cardiovascular events may be stronger in larger studies [11]. Furthermore, hsCRP's functionality as an index of atherosclerotic inflammation lends it as one more tool enabling assessment of AMI risk that warrants further exploration. However, dyslipidemia and its cardiovascular risk in AMI patients can be further unraveled by incorporating genetic markers, as discussed by Pham-Thi NN et al [17]. Finding polymorphisms related to cardiovascular risk will make it possible to improve risk prediction and to provide personalized therapeutic strategies. The results of this study underline the importance of conducting comprehensive lipid profiling in AMI patients together with ApoB and hsCRP in the risk assessment of cardiovascular diseases. Considered together with hypertension, both dyslipidemia and its treatment were consistent with aggressive lipid-lowering and blood pressure control strategies, given the high prevalence of dyslipidemia. More often, gaps in dyslipidemia management are suggested by Talviste G et al., to be addressed in order to reduce recurrent cardiovascular events and improve the outcomes of patients with AMI [18]. Emerging research highlights the role of lipoprotein (a) [Lp (a)] as an independent cardiovascular risk factor, particularly in AMI patients with dyslipidemia. Elevated Lp (a) levels are associated with increased residual cardiovascular risk despite optimal LDL-C management, as demonstrated by Tsimikas S et al [19]. Moreover, recent advances in lipidomics have provided insights into specific lipid species contributing to cardiovascular events. Rhee EJ et al., reported that certain ceramide and sphingolipid profiles may serve as novel biomarkers for AMI risk stratification, offering new therapeutic targets for dyslipidemia management [20].

CONCLUSIONS

This study highlighted the high prevalence of dyslipidemia in AMI patients with its important role in cardiovascular risk. This powerful association of hypertension with dyslipidemia supports the idea that integrated hypertensive patient management is helpful for AMI in reducing cardiovascular risk by addressing both blood pressure and lipid abnormalities. Although novel biomarkers ApoB and hs-CRP did not significantly identify dyslipidemic versus non-dyslipidemic patients, they are important cardiovascular risk assessments and may also function independently of conventional lipid measures. Additionally, the reduction of the prevalence of dyslipidemia, among which approximately half of the population who do not achieve Lp (a) LDL goals, is a reflection of statin therapy's central role in the

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management of cardiovascular risk. Future research should be done with a larger, more diverse population exploring potential genetic, inflammatory, and lifestyle influences on dyslipidemia in AMI patients, which will support more personalized therapeutic approaches.

Authors Contribution

Conceptualization: AF
Methodology: ZI
Formal analysis: MAQ
Writing, review and editing: MAQ, BH, SAF, NA

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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