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Original Research Article

COMPARATIVE EFFECTIVENESS OF HIGH-INTENSITY VERSUS MODERATE-INTENSITY STATIN THERAPY IN PATIENTS WITH ACUTE CORONARY SYNDROME

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ABSTRACT

Background: After acute coronary syndrome (ACS), statin therapy is essential for secondary cardiovascular prevention. However, differences in the effectiveness and tolerability of high-intensity and moderate-intensity statin regimens require further evaluation. The objective is to compare the effectiveness of high-intensity versus moderate-intensity statin therapy in patients with ACS.

Materials and Methods: A prospective observational comparative study was designed at Al-Aleem Medical College/Gulab Devi Hospital, Lahore, between December 1st, 2025, and 31st May, 2026. The patients were randomly allocated into two groups of 60 with high and moderate statin intensity. Lipid profiles were performed at baseline and at 4 weeks; LDL-C target achievement, adherence to medication, adverse effects, and cardiovascular events were evaluated. The level of $p<0.05$ was taken as a criterion of statistical significance.

Results: High-intensity statin therapy had a significantly larger mean LDL-C reduction than moderate-intensity statin therapy (53.1% vs. 35.9%, $p < 0.001$). An LDL-C reduction of at least 50% was achieved in 70.0% and 26.7% of patients, respectively ($p = 0.001$). LDL-C below 55 mg/dL was achieved in 40.0% versus 13.3% ($p = 0.020$). No statistically significant differences were observed in adherence, adverse effects, or cardiovascular events.

Conclusion: High-intensity statin therapy demonstrated greater lipid-lowering efficacy in the short term, but with no evidence of a difference in short-term safety or cardiovascular outcomes.

Keywords: Acute coronary syndrome; high-intensity statins; moderate-intensity statins; LDL-C.

INTRODUCTION

Cardiovascular disease (CVD) is the main cause of morbidity and mortality in the world and also plays a significant role in burdening the health care systems in low- and middle-income countries.[1] In 2022, it was reported that globally around 19.8 million people died from cardiovascular diseases, which represent nearly 32% of all deaths, with 85% being attributed to heart attacks and strokes.[2] Acute coronary syndrome (ACS) is one of the most important clinical presentations for coronary artery disease, including the ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA).[2] Although the early diagnosis, reperfusion, and pharmacological therapy of ACS have improved, survivors are still at a high risk for repeat cardiovascular events.[3] Recent guidelines suggest that the risk of cardiovascular death, myocardial infarction, or ischemic stroke during the first year after hospitalization for ACS is about 10% to 15%, highlighting the necessity for effective secondary prevention measures.[4]

Lipid-lowering therapy, especially statins, is an important component of secondary cardiovascular prevention after ACS.[5] Statins block 3-HMG-CoA reductase, which decreases cholesterol production in the liver and low-density lipoprotein (LDL) cholesterol levels in the blood, and have positive effects on plaque stabilization and vascular inflammation.[6] The usual range of LDL-C reduction with high-intensity statin therapy (atorvastatin 40-80 mg/daily or rosuvastatin 20-40 mg/daily) is at least 50% reduction, and moderate-intensity statin therapy typically yields a reduction of 30-49%.[7] Based on the guidelines of the European Society of Cardiology (ESC) 2023, high-intensity statins should be started as early as possible following ACS, aiming for an LDL-C level below 55 mg/dL and at least 50% reduction from baseline.[8] Likewise, the 2025 American College of Cardiology/American Heart Association guidelines suggest that high-intensity statins should be used in patients with ACS to reduce subsequent major adverse cardiovascular events.[9]

There has been a recent increase in available evidence supporting the rationale for intensive lipid-lowering therapy after ACS.[10] The 2025 ACC/AHA guidelines highlight evidence from randomized trials that show that high-intensity statin therapy has greater benefit with respect to major cardiovascular events than moderate-intensity therapy.[11] New research, however, has been conducted on the potential efficacy of personalized LDL-C goals and other lipid-lowering therapies.[12] Additionally, the 2025 ACS guidelines highlight findings from various studies that have shown that high-intensity statins help prevent major vascular events by around 15% compared to moderate-intensity statins in patients with coronary artery disease.[13] However, the results of the LODESTAR randomized trial of 4,400 patients with CAD showed similar three-year clinical outcomes for the LDL-C treat-to-target strategy as compared with fixed high-intensity statin therapy (8.1% vs 8.7%).[14] These results indicate that intensive lipid lowering has proven cardiovascular advantages, but the best therapy might be individualized based on patients' characteristics and LDL-C levels.

Although guidelines have endorsed the use of high-intensity statins following ACS, key clinical issues remain: What is the extent of their extra benefits compared with moderate-intensity statins in the real world? Variations in baseline cardiovascular risk, comorbidities, treatment adherence, lipid response, and tolerability to the drugs used may affect treatment outcomes, especially in resource-constrained environments where access to follow-up may be limited. Furthermore, current evidence often does not directly compare high-intensity statins with combination lipid-lowering therapy, but rather compares high-intensity statins with any combination of statins and ezetimibe, which leaves an important question regarding the relative effectiveness of high-intensity statins and combination lipid-lowering therapy in ACS patients. So, the purpose of the current study is to determine whether high-intensity statin therapy is more effective than moderate-intensity statin therapy for lowering LDL-C levels in patients with acute coronary syndrome, with the secondary objectives being to measure the timing of major adverse cardiovascular events and adverse effects of the therapy, which are available with follow-up data. The results could offer clinically relevant information that could guide the selection of statins for individual patients, optimize secondary cardiovascular prevention, and guide therapeutic decisions among patients with ACS.

MATERIALS AND METHODS

A hospital-based, prospective observational comparative study was conducted in the Department of Cardiology, Al-Aleem Medical College/Gulab Devi Hospital, Lahore, Pakistan. Acute coronary syndrome patients with the inclusion criteria were recruited from the cardiology ward of the hospital and then evaluated during outpatient follow-up visits. This study was a 6-month study, which involved patient recruitment, clinical assessment, follow-up, data collection, and statistical analysis from 1st December 2025 to 31st May 2026.

The number of samples was calculated using the OpenEpi sample size calculator for two independent proportions, where the 95% confidence level, 80% statistical power, and allocation ratio of 1:1 were used for the treatment groups. The calculation was based on an analysis by Mehmood et al. (2026) on the 50% LDL-C reductions one could achieve at 3 months of treatment with high-intensity statins (46.8%) and moderate-intensity statins (16.8%).[15] These proportions led to a minimum sample size of around 22 patients in each group according to the Fleiss formula. Continuity correction resulted in the necessary sample size of about 27 patients per group. The proposed sample size was 60 patients, with 30 patients in each group, which included an anticipated 10% loss to follow-up.

A non-probability consecutive sampling technique was employed. Patients who were admitted to the hospital with an ACS during the recruitment period and were eligible for the study were screened for enrollment. Consecutive sampling was performed for the selected patients who gave written informed consent until the sample size was reached. The patients were divided into two groups based on the intensity of statin therapy. Patients in the high-intensity statin therapy group were assigned to group A, and patients in the moderate-intensity statin therapy group were assigned to group B.

The patients were aged 18 years or older, both males and females, and had been diagnosed with acute coronary syndrome (ACS), which encompassed ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), or unstable angina. The diagnosis was made based on clinical presentation, electrocardiographic findings, and cardiac

biomarkers, if available. Patients who were prescribed high-intensity or moderate-intensity statin therapy during the hospital stay, had a baseline lipid profile measurement, and consented to follow-up evaluation were included.

Patients who were known to be statin-sensitive or intolerant, who had active liver disease, with unexplained increases in hepatic transaminases more than three times the upper limits of normal, severe renal impairment, or a previous history of rhabdomyolysis were excluded. Patients who were using other lipid-lowering drugs simultaneously, such as fibrates, PCSK9 inhibitors, and ezetimibe, were also not included, so as to avoid the impact of those other drugs on LDL-C. Pregnant or breastfeeding women, patients with terminal diseases, and patients who did not complete the necessary follow-up evaluation were not included in the final analysis. Those who had not undergone a lipid profile before the study were not included.

Once approved by the institutional ethics committee, the patients with ACS were screened in the cardiology department for eligibility. Eligible participants were recruited after obtaining informed written consent. The data collection form was structured, and demographic data such as age, gender, BMI, smoking history, and medical history were collected. Clinical data were obtained, such as the type of ACS, hypertension, diabetes mellitus, a history of cardiovascular disease, and baseline lipid profile data. Patients were randomized to two groups by the statin therapy prescribed by their treating cardiologists. Adherence to the high-intensity statin group was defined as taking atorvastatin 40–80 mg/day or rosuvastatin 20–40 mg/day, while the moderate-intensity statin group was defined as patients taking atorvastatin 10–20 mg/day or rosuvastatin 5–10 mg/day, according to established intensity categories.

At enrollment, baseline LDL-C, total cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglyceride levels were measured. Patients were maintained on the assigned statins by their treating physicians. Approximately four weeks after the start of treatment, lipid profile measurements were repeated to assess the biochemical response. The percentage reduction in LDL-C was determined by calculating the difference between the follow-up LDL-C and baseline LDL-C, dividing by baseline LDL-C, and multiplying by 100.

The main outcome was the percentage of patients with at least a 50% decrease in LDL-C from baseline. A secondary outcome was the achievement of an LDL-C level < 55 mg/dL. Medication adherence, muscle symptoms, adverse effects reported, and any documented events of the cardiovascular system were obtained via interviews with the patient and medical records during follow-up. All data collected were recorded on a standardized data collection form and checked for completeness prior to statistical analysis. Data collected were analyzed using IBM Statistical Package for the Social Sciences (SPSS) version 26.0.

For continuous variables, such as age, BMI, baseline LDL-C, follow-up LDL-C, and percentage change in LDL-C, these were reported as mean and standard deviation for normally distributed variables or median and interquartile range for variables that were not normally distributed. The categorical variables gender, ACS type, hypertension, diabetes mellitus, smoking history, LDL-C target achievement, and adverse effects) were presented as frequencies and percentages. Normality of the continuous variables was checked by the Shapiro–Wilk test. Independent-samples t-tests. Categorical outcomes were compared using chi-square tests and Fisher's exact tests. To determine the association between statin intensity and LDL-C target achievement, after adjusting for important baseline clinical factors, multivariable logistic regression analysis was planned, subject to the number of outcomes observed being sufficient for reliable estimation. A two-sided p-value of less than 0.05 was considered statistically significant.

RESULTS

The study involved 60 patients with acute coronary syndrome (ACS), 30 in each treatment group. The average age was 56.4 ± 9.8 years for the high-intensity group and 57.2 ± 10.3 years for the moderate-intensity group. In both groups, there were more male patients (70.0% and 66.7%). There were no statistically significant differences in demographic characteristics: age, gender, BMI, or smoking status. [Table 1]

ST-segment elevation myocardial infarction (STEMI) accounted for the largest proportion of ACS cases, at 46.7% in the high-intensity group and 43.3% in the moderate-intensity group. The most common comorbidities were hypertension, diabetes mellitus, and prior cardiovascular disease. The distribution of ACS types and comorbidities was comparable between both treatment groups, with no statistically significant difference ($p>0.05$). [Table 2]

Atorvastatin was the most commonly used statin in both groups. Among the high-intensity group, atorvastatin 40 mg/day (50.0%) was the most common treatment, followed by rosuvastatin 20 mg/day (23.3%). In the moderate-intensity group, atorvastatin 20 mg/day was the most commonly prescribed (43.3%), followed by atorvastatin 10 mg/day (30.0%). [Table 3]

The high-intensity statin group had a significantly larger LDL-C reduction than the moderate-intensity statin group ($p < 0.001$). Follow-up LDL-C levels were significantly lower in the high-intensity group ($p < 0.001$). The total cholesterol levels at follow-up also differed significantly between groups ($p = 0.002$); however, differences in the change in HDL-C and triglyceride levels were not statistically significant. [Table 4]

The proportion of patients who achieved the pre-defined LDL-C treatment targets was significantly higher among those who were prescribed high-

intensity statins. A reduction of LDL-C was achieved in 70.0% of patients in the high-intensity group vs 26.7% in the moderate-intensity group (p = 0.001). Likewise, 40.0% and 13.3% of patients had an LDL-C level < 55 mg/dL, respectively (p = 0.020). [Table 5]

No statistically significant difference was observed in the level of adherence to medication between the treatment groups (p = 1.000). Muscle symptoms were more common in the high-intensity group (16.7% vs. 6.7%), but this difference was not statistically significant (p = 0.424). Documented cardiovascular events were rare, with a prevalence of 3.3% and 6.7% of patients in the two groups, respectively, with no significant difference between these groups (p = 1.000). [Table 6]

Table 1: Demographic Characteristics of Study Participants (n = 60)

Variables	High-Intensity (n=30)	Moderate-Intensity (n=30)	p-value
Age (years), mean ± SD	56.4 ± 9.8	57.2 ± 10.3	0.759
BMI (kg/m²), mean ± SD	27.3 ± 3.6	27.8 ± 3.9	0.608
Gender			
Male	21 (70.0%)	20 (66.7%)	0.781
Female	9 (30.0%)	10 (33.3%)	
Smoking status			
Smoker	12 (40.0%)	11 (36.7%)	0.791
Non-smoker	18 (60.0%)	19 (63.3%)	

Table 2: Baseline Clinical Characteristics of Patients (n = 60)

Variables	High-Intensity (n=30)	Moderate-Intensity (n=30)	p-value
Type of ACS			
STEMI	14 (46.7%)	13 (43.3%)	0.966
NSTEMI	11 (36.7%)	12 (40.0%)	
Unstable angina	5 (16.7%)	5 (16.7%)	
Comorbidities			
Hypertension	18 (60.0%)	17 (56.7%)	0.793
Diabetes mellitus	12 (40.0%)	11 (36.7%)	0.791
Previous cardiovascular disease	7 (23.3%)	6 (20.0%)	0.754

Table 3: Distribution of Statin Therapy and Prescribed Doses (n = 60)

Statin regimen	High-Intensity (n=30)	Moderate-Intensity (n=30)
Atorvastatin 10 mg/day	0	9 (30.0%)
	0	13 (43.3%)
	15 (50.0%)	0
	6 (20.0%)	0
Rosuvastatin 5 mg/day	0	5 (16.7%)
	0	3 (10.0%)
	7 (23.3%)	0
	2 (6.7%)	0
Total	30 (100%)	30 (100%)

Table 4: Comparison of Baseline and Follow-Up Lipid Profiles between Treatment Groups

Lipid parameters	High-Intensity (n=30)	Moderate-Intensity (n=30)	p-value
Baseline LDL-C (mg/dL)	130.0 ± 22.4	128.0 ± 23.1	0.734
Follow-up LDL-C (mg/dL)	61.0 ± 16.2	82.0 ± 19.5	<0.001
LDL-C reduction (mg/dL)	69.0 ± 19.1	46.0 ± 18.2	<0.001
LDL-C reduction (%)	53.1 ± 11.6	35.9 ± 12.4	<0.001
Baseline total cholesterol (mg/dL)	211.5 ± 31.2	209.3 ± 29.6	0.780
Follow-up total cholesterol (mg/dL)	143.2 ± 23.4	163.5 ± 25.7	0.002
Baseline HDL-C (mg/dL)	39.6 ± 7.1	40.1 ± 7.5	0.792
Follow-up HDL-C (mg/dL)	42.8 ± 7.3	42.2 ± 7.6	0.756
Baseline triglycerides (mg/dL)	178.4 ± 41.3	175.6 ± 39.8	0.790
Follow-up triglycerides (mg/dL)	146.5 ± 33.2	153.8 ± 35.4	0.413

Table 5: Comparison of LDL-C Target Achievement and Treatment Response (n = 60)

Outcome variables	High-Intensity (n=30)	Moderate-Intensity (n=30)	p-value
LDL-C reduction ≥50%			
Achieved	21 (70.0%)	8 (26.7%)	0.001
Not achieved	9 (30.0%)	22 (73.3%)	
LDL-C below 55 mg/dL			
Achieved	12 (40.0%)	4 (13.3%)	0.020
Not achieved	18 (60.0%)	26 (86.7%)	

Table 6: Comparison of Medication Adherence, Adverse Effects, and Cardiovascular Events (n = 60)

Variables	High-Intensity (n=30)	Moderate-Intensity (n=30)	p-value
Medication adherence			
Adherent	26 (86.7%)	27 (90.0%)	1.000

Non-adherent	4 (13.3%)	3 (10.0%)	
Treatment-related adverse effects			
Muscle-related symptoms	5 (16.7%)	2 (6.7%)	0.424
Other reported adverse effects	1 (3.3%)	1 (3.3%)	1.000
Any adverse effect	6 (20.0%)	3 (10.0%)	0.472
No adverse effect	24 (80.0%)	27 (90.0%)	
Cardiovascular events during follow-up			
Any documented cardiovascular event	1 (3.3%)	2 (6.7%)	1.000
No documented cardiovascular event	29 (96.7%)	28 (93.3%)	

DISCUSSION

In the present study, there was a greater reduction in low-density lipoprotein cholesterol (LDL-C) among patients who received high-intensity statin therapy than in those who received moderate-intensity statin therapy after acute coronary syndrome (ACS). The difference in mean percentage reduction of LDL-C between the high-intensity group and moderate-intensity group was statistically significant and amounted to 53.1% versus 35.9% ($p < 0.001$). These results were consistent with the known dose-dependent lipid-lowering action of statins in a direction that was consistent with that effect. In a 2021 nationwide cohort study of patients with AMI, there were no significant differences in MACE between high-intensity statins and moderate-intensity statins plus ezetimibe.[16] Likewise, another cohort study in 2021 of coronary revascularization patients found similar CV outcomes with these methods. The importance of effective lipid reduction was confirmed in these studies, but they are not directly comparable with the groups in the present study because their moderate-intensity statin therapy included ezetimibe.[17]

A significantly higher percentage of patients in the high-intensity group (70.0%) achieved at least a 50% drop in LDL-C levels than did the moderate-intensity group (26.7%). Likewise, high-intensity treatment (40.0% versus 13.3%) was more likely to obtain an LDL-C concentration below 55 mg/dL. The results of these findings indicated a difference in the short-term biochemical responses of the treatment intensities. In the RACING randomized trial, Kim et al. (2022) showed that moderate-intensity rosuvastatin plus ezetimibe was not inferior to high-intensity rosuvastatin alone in terms of major cardiovascular outcomes after 3 years (9.1% vs. 9.9%).[18] Similarly, Hong et al. (2023) found similar three-year cardiovascular events between treat-to-target and a fixed high-intensity statin therapy in the LODESTAR randomized trial of 4,400 patients with coronary artery disease (8.1% vs. 8.7%).[17] These trials suggest that LDL-C-lowering strategies other than statins can lead to similar longer-term results, but neither compared the same statin with each other nor followed a similar regimen of short-term follow-up in the present trial.

The incremental benefit of higher-intensity therapy on reducing LDL-C was also taken into account based on the current observational evidence. A recent 2024 study of a nationwide cohort of patients with ACS who underwent PCI showed a reduced risk of adverse cardiovascular outcomes in patients taking moderate doses of statins plus ezetimibe rather than high-intensity statins alone. The additional lipid-lowering effect of ezetimibe, however, and the observational design prevented direct comparisons with present findings.[19] In another 2024 study on early intensive lipid-lowering therapy after ACS, LDL-C decreased from 107±38 mg/dL at baseline to 78±25 mg/dL at 2 months, with only 40.2% of patients having an LDL-C <70 mg/dL.[20] The overall results of the analyses were similar to the known fact that significant LDL lowering was often not associated with targeted LDL reduction in all individuals. Some variations between studies might be due to the different baseline lipid concentrations, treatments, and follow-up periods.

In terms of treatment safety, 16.7% of those receiving high-intensity therapy reported muscle-related side effects, while 6.7% reported muscle-related side effects with moderate-intensity therapy, but there was no significant difference. There was no difference between the two groups in adherence to medication. The number of patients who developed new-onset diabetes after myocardial infarction was greater in those taking high-intensity statin therapy than in those taking moderate-intensity statin therapy in a 2024 nationwide cohort study.[21] There were also fewer major adverse cardiovascular events in the high-intensity group, which was not significant after adjustment. Contrary to the study above, the present study focused on short-term adverse events, rather than incident diabetes, and did not make long-term conclusions about metabolic safety. In a 2025 prospective cohort study conducted in Switzerland, statin intensity and physical activity after ACS were also investigated, highlighting the importance of treatment intensity and patient-related factors in the recovery phase.[22]

Overall, patients with ACS receiving high-intensity statin therapy had a greater short-term reduction in LDL-C and more patients reached preselected lipid targets than those receiving moderate-intensity statin monotherapy. No statistically significant difference was shown in adherence to treatment, side effects, or reported cardiovascular events, however. The small sample size, single-centre observational design, short follow-up period, and the possibility of confounding due to non-randomised allocation of treatment limited the findings. Furthermore, no multivariable adjustment has been done to determine if the observed differences were independent of baseline clinical characteristics. These results, therefore, must be considered preliminary biochemical comparisons

and are not a sign of greater long-term clinical effectiveness. Further, larger, prospective studies with longer follow-up periods that can adjust for potential confounding factors would be needed to more reliably evaluate cardiovascular outcomes and treatment safety.

Limitations: There were some limitations in the study. The findings were only generalizable to 60 patients from one centre. Second, the observational design and non-randomized treatment allocation posed the risk of selection bias and residual confounding. Third, the lack of long-term follow-up for the cardiovascular outcomes, medication adherence, and adverse effects limited the evaluation of these parameters, which are typically assessed over a longer period. In addition, there could have been treatment differences due to baseline clinical features and personal responses to statins as well as to unrecorded confounding factors. Lastly, the number of cardiovascular events was too small to enable a proper comparison of clinical effectiveness between the two treatment groups.

CONCLUSION

The high-intensity statin therapy achieved significantly greater LDL-C reduction, and more individuals achieved the recommendation of LDL targets in the study population with acute coronary syndrome (ACS) compared with moderate-intensity statin therapy. But there were no statistical differences in adverse effects or with respect to cardiovascular outcomes in the short term, or in medication adherence. These results indicated that high-intensity statins better achieved short-term lipid control, but this did not translate to an improvement in cardiovascular outcomes. Larger multicentre studies with longer follow-up periods are needed to evaluate long-term effectiveness and safety.

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