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COMPARISON OF HIGH AND MODERATE INTENSITY STATINS IN ACHIEVING THE TARGET LDL-C LEVEL AFTER ACUTE CORONARY SYNDROME

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ABSTRACT

To compare the effectiveness of high intensity and moderate intensity statin therapy in achieving target low density lipoprotein cholesterol level in patients after acute coronary syndrome.

Study Design

Randomized controlled trial.

Duration and Place of Study

This study was conducted from 1 February 2025 to 15 November 2025 at the Cardiology Department of KTH Peshawar.

Methodology

In this clinical study 190 patients with acute coronary syndrome were selected. These patients were divided into two groups based on the method and dose of statins prescribed to them. In one group, the prescribed dose was moderate, and for the remaining group, the prescribed dose was higher. The criterion for the efficacy of the treatments was established to take into account the lowering of LDL-C levels by at least 50%. Analysis of all the gathered data was done using SPSS software. Version 25 was used. Chi-square and Fisher's Exact tests were used to compare the efficacy levels of the two treatments. An established criterion for significance was a p-value equal to and less than 0.05.

Results

Mean age was 58.52 ± 10.77 years in moderate intensity group and 60.22 ± 10.08 years in high intensity group. After three months target LDL cholesterol level was achieved in 16.8% patients in moderate intensity group and 56.8% patients in high intensity group.

Conclusion

High intensity statin therapy was more effective than moderate intensity statin therapy in achieving target LDL cholesterol level after acute coronary syndrome.

Keywords
Acute coronary syndrome, Cholesterol LDL, Hydroxymethylglutaryl-CoA reductase inhibitors, Hyperlipidemias

INTRODUCTION

The Acute Coronary Syndrome includes a group of clinical entities that occur because of sudden diminished or complete occlusion of myocardial blood flow, which is primarily caused by rupture of an atherosclerotic plaque with subsequent formation of thrombus in the coronary arteries.[1] The Acute Coronary Syndrome primarily includes unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI) based on increased severity despite having an identical underlying mechanism.[2] Clinically, patients with Acute Coronary Syndrome present with chest pain, shortness of breath, diaphoresis, nausea, as well as anxiety, which require urgent medical assessment.[3] The Acute Coronary Syndrome is considered to be one of the leading reasons for morbidity and mortality worldwide, and thus its early diagnosis based on electrocardiographic changes, blood biomarkers, as well as detailed clinical assessment, is also paramount.[4] Subsequent to Acute Coronary Syndrome, patients are also at high risk of recurrent events, which require implementation of secondary prevention measures to improve outcomes.[5]

Statins are recognized as playing a pivotal role in lowering low-density lipoprotein cholesterol (LDL-C) and serve as first-line management in dyslipidemias, especially in patients with existing cardiovascular disease.[6] LDL-C is known to have a strong correlation with the emergence and development of atherosclerosis, while its reduction is also correlated with low plaque formation and enhancement in endothelial dysfunction.[7] Statins work by inhibiting HMG-CoA reductase in the hepatic tissue, which also increases LDL-R expression in the blood cells, leading to increased uptake of LDL-C.[8] Early initiation of statins in patients with acute coronary syndromes has also demonstrated its efficacy in both recurrent ischemic events and in improved survival rates.[9] Apart from its potential in lipid lowering, it also has pleiotropic properties with anti-inflammatory responses and improvement in endothelial functions.[10]

High and moderate intensity statins are used to achieve the LDL-C goals recommended in patients after being exposed to acute coronary syndrome, with an individual risk profile, tolerability and the available clinical guidance.[11] High intensity statins can reduce LDL-C by more than 50 percent and are thus normally recommended to most post-ACS patients with the aim of achieving rigorous lipid targets.[12] Moderate statins, including moderate-intensity statins, reduce LDL-C by 30-49 percent and can be used in people unable to tolerate high doses, or with contraindications.[13] Achievement of target LDL-C levels has been found to have clinical importance as lower concentrations have been linked with reduced recurrence of myocardial infarction and cardiovascular death.[14]

There is strong need to do this study in Peshawar because acute coronary syndrome cases are increasing and cardiovascular disease is becoming a major health problem in this region. Many patients after acute coronary syndrome do not achieve target LDL C levels due to limited awareness and late follow up and differences in treatment practices. Data related to use of high and moderate intensity statins and their effectiveness in achieving LDL C targets is very limited in Peshawar population. Local lifestyle factors and dietary habits and access

to healthcare may also affect statin response. Therefore, this study is important to provide local evidence and help improve treatment strategies and patient outcomes in Peshawar.

METHODOLOGY

This randomized controlled trial was carried out in the Cardiology Department of MTI Khyber Teaching Hospital Peshawar from 1 February 2025 to 15 November 2025. Ethical approval was obtained from the Ethical Review Board of the hospital and the research department of CPSP Karachi before initiation of the study, and the trial was registered with the Clinical Trial Unit of Khyber Medical University, Peshawar (KMU/DIR/CTU/2024/013). The sample size was calculated by using OpenEpi calculator with confidence level of 95% and power of 80% taking expected proportion of patients achieving target LDL-C as 30% in moderate intensity statin group and 50% in high intensity statin group.[15] The total calculated sample was 190 patients, with 95 patients in each group. Patients of age ranging from 25 to 85 years of both genders, diagnosed with acute coronary syndrome were included in the study. Patients having previous history of adverse reaction to statins such as hypersensitivity, myopathy or acute renal failure, acute liver disease, pregnant or breastfeeding women and those who were already taking statin regularly were excluded. Acute coronary syndrome was considered when patients presented with features of ST-segment elevation myocardial infarction, non-ST-segment elevation myocardial infarction or unstable angina based on clinical presentation, electrocardiographic findings and cardiac biomarkers. ST-segment elevation myocardial infarction was considered when patients had chest or epigastric pain with score of 4 or more on visual analogue scale along with electrocardiographic ST-segment elevation at J-point persisting for more than 20 minutes in at least 2 contiguous leads and elevated high sensitivity troponin levels above 16 ng/l in females and 34 ng/l in males. Non-ST-segment elevation myocardial infarction was considered when symptoms and troponin elevation were present without ST-segment elevation on ECG. Unstable angina was considered when ischemic symptoms and ECG changes were present without troponin elevation.

Written informed consent was taken from all patients prior to data collection after explaining the purpose and procedure of the study. On admission, detailed history was taken regarding chest pain, associated symptoms and past medical illness. Physical examination was performed with focus on cardiovascular system. All patients received standard management for acute coronary syndrome according to ACC guidelines. Baseline investigations including lipid profile, liver function tests and creatine phosphokinase were performed. Patients were then allocated into two groups by blocked randomization based on age and gender to maintain balance between groups. The randomization was done in six blocks with distribution according to predefined age ranges of 25–44 years, 45–64 years and 65–85 years and equal gender representation within each block. Group-A received moderate intensity statin, while Group-B received high intensity statin. Same trade name of rosuvastatin was used for all patients. High intensity statin was atorvastatin 40 mg or rosuvastatin 20 mg. Moderate intensity statin was atorvastatin 20 mg or rosuvastatin 10 mg. Patients were monitored during hospital stay for any adverse effects related to statins. Patients and family members were educated regarding dosage, possible side effects, and the need for compliance. They were instructed to record any missed doses. Lack of compliance with statins was considered to be lack of a dose for more than 7 days in any given month. A follow-up visit was planned at 3 months, where lipids, liver function tests, and creatine phosphokinase levels were again estimated. Patients who could not be contacted for 3 months were considered lost to follow-up while patients

who withdrew from the study were considered for exclusion from analysis. Response was considered to be at least 50% lowering of LDL-C levels at 3 months.

All statistical analysis was carried out using IBM SPSS version 27. Continuous data including age, body mass index and LDL-C levels were summarized using mean values along with their standard deviations. Categorical information was described in terms of counts and percentages. Efficacy of moderate dose versus high-dose statin therapy was compared using the Chi-square test or Fisher's exact test with a significance level ≤ 0.05. Factors such as age, gender, place of residence, socioeconomic background, smoking status, educational level and occupation stratification were performed by post-stratification analysis. Results were considered significant when the p-value ≤ 0.05.

RESULTS

The mean age of patients in Group A was found 58.52 ± 10.77 years while in Group B it was 60.22 ± 10.08 years. The average weight was recorded as 75.00 ± 12.26 kg in Group A and 79.22 ± 10.99 kg in Group B. Height measurements was showing 170.20 ± 7.19 cm for Group A patients and 169.03 ± 7.05 cm for Group B patients. Body mass index was calculated as 25.90 ± 4.76 in Group A compared to 27.83 ± 4.45 in Group B. The baseline LDL-C level was measured 113.95 ± 39.19 mg/dL in Group A which was higher than Group B showing 99.16 ± 36.92 mg/dL. After 3 months of treatment, LDL-C levels was reduced to 82.05 ± 28.72 mg/dL in Group A and 58.72 ± 28.68 mg/dL in Group B. Regarding gender distribution, male patients were comprising 53 (55.8%) in Group A and 64 (67.4%) in Group B, while female patients were 42 (44.2%) and 31 (32.6%) respectively. Most of the patients was residing in urban areas with 68 (71.6%) in Group A and 67 (70.5%) in Group B, whereas rural residence was reported in 27 (28.4%) and 28 (29.5%) patients respectively. Smoking status was revealing that 17 (17.9%) patients in Group A and 21 (22.1%) in Group B was smokers, while non-smokers was 78 (82.1%) and 74 (77.9%) respectively. Education level distribution was showing that majority was uneducated with 71 (74.7%) in Group A and 62 (65.3%) in Group B. Primary education was found in 5 (5.3%) patients of Group A only, secondary education was present in 15 (15.8%) and 25 (26.3%) patients, and higher education was achieved by 4 (4.2%) and 8 (8.4%) patients in Group A and B respectively. The type of acute coronary syndrome was distributed differently between groups where NSTEMI was diagnosed in 44 (46.3%) patients of Group A and 38 (40.0%) of Group B. STEMI was more frequent in Group B with 57 (60.0%) cases compared to 37 (38.9%) in Group A. Unstable Angina was found only in Group A with 12 (12.6%) cases, and Anterior Wall MI was also limited to Group A with 2 (2.1%) cases. Comorbidity pattern was showing that 25 (26.3%) patients in Group A and 12 (12.6%) in Group B was having no comorbidities. Diabetes Mellitus alone was present in 19 (20.0%) and 11 (11.6%) patients, Hypertension alone was found in 25 (26.3%) and 43 (45.3%) patients, while combination of Diabetes and Hypertension was observed in 26 (27.4%) and 29 (30.5%) patients of Group A and B respectively as shown in Table-I.

Table I: Patients Demographics in both groups

Variables	Group A (n=95) Mean ± SD	Group B (n=95) Mean ± SD
Age	58.52 ± 10.77	60.22 ± 10.08
Weight (kg)	75.00 ± 12.26	79.22 ± 10.99
Height (cm)	170.20 ± 7.19	169.03 ± 7.05
BMI	25.90 ± 4.76	27.83 ± 4.45
Baseline LDL-C	113.95 ± 39.19	99.16 ± 36.92

After 3 months LDL-C	82.05 ± 28.72	58.72 ± 28.68
Gender	**n** (%)	**n** (%)
Male	53 (55.8%)	64 (67.4%)
Female	42 (44.2%)	31 (32.6%)
Residence	**n** (%)	**n** (%)
Rural	27 (28.4%)	28 (29.5%)
Urban	68 (71.6%)	67 (70.5%)
Smoking Status	**n** (%)	**n** (%)
Yes	17 (17.9%)	21 (22.1%)
No	78 (82.1%)	74 (77.9%)
Education Level	**n** (%)	**n** (%)
Uneducated	71 (74.7%)	62 (65.3%)
Primary	5 (5.3%)	–
Secondary	15 (15.8%)	25 (26.3%)
Higher	4 (4.2%)	8 (8.4%)
Type of ACS	**n** (%)	**n** (%)
NSTEMI	44 (46.3%)	38 (40.0%)
STEMI	37 (38.9%)	57 (60.0%)
Unstable Angina	12 (12.6%)	–
Anterior Wall MI	2 (2.1%)	–
Comorbidity	**n** (%)	**n** (%)
None	25 (26.3%)	12 (12.6%)
Diabetes Mellitus	19 (20.0%)	11 (11.6%)
Hypertension	25 (26.3%)	43 (45.3%)
Diabetes + Hypertension	26 (27.4%)	29 (30.5%)

The efficacy comparison between two groups was demonstrating significant differences where achieving target LDL-C level was successful in only 16 (16.8%) patients of Group A compared to 54 (56.8%) patients in Group B. Failure to achieve target was observed in 79 (83.2%) patients of Group A and 41 (43.2%) patients of Group B. This difference was statistically highly significant with p-value <0.001 as shown in Table-II.

Table- II: Comparison of efficacy between the two groups

Efficacy	Group A (n=95) n (%)	Group B (n=95) n (%)	P value
Yes	16 (16.8%)	54 (56.8%)	
No	79 (83.2%)	41 (43.2%)	<0.001
Total	95 (100%)	95 (100%)	

The association of efficacy with demographic variables was analyzed through stratified analyses showing various significant relationships. For age stratification, in patients ≤50 years, efficacy was achieved in 5 (22.7%) of Group A versus 9 (47.4%) of Group B with p=0.019, while in patients >50 years, efficacy was 11 (15.1%) versus 45 (59.2%) with p=0.004. Gender stratification was revealing that among males, efficacy was 10 (18.9%) in Group A and 39 (60.9%) in Group B (p=0.187), whereas in females it was 6 (14.3%) and 15 (48.4%) respectively (p=0.142). Residence-based analysis was

showing that rural patients was achieving efficacy in 4 (14.8%) of Group A and 14 (50.0%) of Group B (p=0.043), while urban patients was showing 12 (17.6%) and 40 (59.7%) respectively (p=0.001). Smoking status stratification was demonstrating that among smokers, only 1 (5.9%) of Group A achieved efficacy compared to 16 (76.2%) of Group B (p=0.021), and in non-smokers, efficacy was 15 (19.2%) versus 38 (51.4%) with p=0.037. Education level analysis was showing that uneducated patients was achieving efficacy in 11 (15.5%) of Group A and 38 (61.3%) of Group B (p=0.008). Primary educated patients were showing 2 (40.0%) efficacy in Group A only (p=0.146), secondary educated patients were 2 (13.3%) and 11 (44.0%) (p=0.126), and higher educated patients was 1 (25.0%) and 5 (62.5%) respectively (p=0.687). Type of ACS stratification was revealing that NSTEMI patients was achieving efficacy in 11 (25.0%) of Group A versus 25 (65.8%) of Group B (p=0.026), while STEMI patients was showing 5 (13.5%) and 29 (50.9%) respectively (p=0.003). Unstable Angina patients in Group A was showing 0 (0.0%) efficacy, and Anterior Wall MI patients was also showing 0 (0.0%) efficacy. Comorbidity stratification was demonstrating that patients with no comorbidities was achieving efficacy in 1 (4.0%) of Group A compared to 8 (66.7%) of Group B (p<0.001). Diabetic patients were showing 7 (36.8%) and 7 (63.6%) efficacy (p=0.851), hypertensive patients were 4 (16.0%) and 28 (65.1%) (p<0.001), while patients with both Diabetes and Hypertension was showing 4 (15.4%) and 11 (37.9%) efficacy with p=0.028 as shown in Table-III.

Table- III: Association of Efficacy with Demographic Variables

Demographic Variables	Group	Efficacy Yes, n (%)	Efficacy No, n (%)	P-value
Age Group (years)				
≤50	A	5 (22.7%)	17 (77.3%)	0.019
	B	9 (47.4%)	10 (52.6%)	
>50	A	11 (15.1%)	62 (84.9%)	0.004
	B	45 (59.2%)	31 (40.8%)	
Gender				
Male	A	10 (18.9%)	43 (81.1%)	0.187
	B	39 (60.9%)	25 (39.1%)	
Female	A	6 (14.3%)	36 (85.7%)	0.142
	B	15 (48.4%)	16 (51.6%)	
Residence				
Rural	A	4 (14.8%)	23 (85.2%)	0.043
	B	14 (50.0%)	14 (50.0%)	
Urban	A	12 (17.6%)	56 (82.4%)	0.001
	B	40 (59.7%)	27 (40.3%)	
Smoking Status				
Yes	A	1 (5.9%)	16 (94.1%)	0.021*
	B	16 (76.2%)	5 (23.8%)	
No	A	15 (19.2%)	63 (80.8%)	0.037
	B	38 (51.4%)	36 (48.6%)	
Education Level				
Uneducated	A	11 (15.5%)	60 (84.5%)	0.008
	B	38 (61.3%)	24 (38.7%)	
Primary	A	2 (40.0%)	3 (60.0%)	0.146*

	B	–	–	
Secondary	A	2 (13.3%)	13 (86.7%)	0.126
	B	11 (44.0%)	14 (56.0%)	
Higher	A	1 (25.0%)	3 (75.0%)	0.687*
	B	5 (62.5%)	3 (37.5%)	
Type of ACS				
NSTEMI	A	11 (25.0%)	33 (75.0%)	0.026
	B	25 (65.8%)	13 (34.2%)	
STEMI	A	5 (13.5%)	32 (86.5%)	0.003
	B	29 (50.9%)	28 (49.1%)	
Unstable Angina	A	0 (0.0%)	12 (100.0%)	–*
	B	–	–	
Anterior Wall MI	A	0 (0.0%)	2 (100.0%)	–*
	B	–	–	
Comorbidity				
None	A	1 (4.0%)	24 (96.0%)	<0.001*
	B	8 (66.7%)	4 (33.3%)	
Diabetes Mellitus	A	7 (36.8%)	12 (63.2%)	0.851
	B	7 (63.6%)	4 (36.4%)	
Hypertension	A	4 (16.0%)	21 (84.0%)	<0.001
	B	28 (65.1%)	15 (34.9%)	
Diabetes + Hypertension	A	4 (15.4%)	22 (84.6%)	0.028
	B	11 (37.9%)	18 (62.1%)	

*Fischer Exact Test

Discussion:

The overall efficacy analysis was showing that 54 (56.8%) patients in high intensity group achieved target LDL-C compared to only 16 (16.8%) in moderate intensity group with p<0.001. This mark difference can be explained by pharmacological mechanism where high intensity statins was providing greater HMG-CoA reductase inhibition leading to more pronounced reduction in hepatic cholesterol synthesis. The increased potency of high dose statins was resulting in enhanced upregulation of LDL receptors on hepatocyte surface which facilitates greater LDL-C clearance from circulation. The baseline LDL-C was higher in moderate intensity group at 113.95 ± 39.19 mg/dL compared to 99.16 ± 36.92 mg/dL in high intensity group, yet after 3 months treatment, high intensity group achieved lower levels of 58.72 ± 28.68 mg/dL versus 82.05 ± 28.72 mg/dL. This superior reduction in high intensity group was occurring despite lower baseline values which demonstrates dose-dependent lipid lowering effect of statins. Higher doses were providing approximately 6% additional LDL-C reduction for each doubling of statin dose. The comorbidity analysis was showing that hypertensive patients on high intensity statins achieved 28 (65.1%) efficacy versus 4 (16.0%) on moderate intensity with p<0.001. Hypertension and hyperlipidemia were having synergistic effect on endothelial dysfunction and atherosclerosis progression. The combination was accelerating plaque formation through multiple pathways including increased oxidative stress and inflammation, thus requiring more aggressive lipid lowering for cardiovascular protection. Urban residents were showing better efficacy with 40 (59.7%) in high intensity group compared to 12 (17.6%) in moderate intensity group with p=0.001. Urban

population was having better access to healthcare facilities ensuring regular medication adherence and follow-up. Moreover, urban patients were more educated about cardiovascular risk factor and importance of lipid control leading to better compliance with prescribed therapy.

The current study findings were demonstrating that high intensity statins achieved significantly better efficacy with 54 (56.8%) patients reaching target LDL-C compared to only 16 (16.8%) in moderate intensity group (p<0.001). This result was showing similarity with Dai YY *et al.* [16] where intensive statin group achieved 67.9% target attainment compared to 46.9% in moderate-intensity group (p=0.047), though their overall success rates was higher possibly due to shorter follow-up period of 1 month versus 3 months in present study. The longer duration in current study was allowing more time for lipid metabolism adaptation and potential adherence issues which might be explaining lower achievement rates. However, current findings were contrasting sharply with Chinwong D *et al.* [17] who reported no significant difference between high potency (25%) and low potency statins (23%) with p=0.363. This discrepancy can be attributed to different patient population characteristics and statin dosing protocols in Thailand where genetic variations in cytochrome P450 metabolism was affecting statin bioavailability differently in Asian populations. The baseline LDL-C reduction from 113.95 ± 39.19 mg/dL to 82.05 ± 28.72 mg/dL in moderate intensity group and from 99.16 ± 36.92 mg/dL to 58.72 ± 28.68 mg/dL in high intensity group was comparable to Lee J *et al.* [18] who demonstrated 31.0-41.0% additional LDL-C reductions with enhanced therapy achieving 88.3-91.1% target success. The magnitude of reduction in present study was smaller because Lee J *et al.* [18] used combination therapy with ezetimibe which was providing dual mechanism of action through both hepatic synthesis inhibition and intestinal absorption blockade, whereas current study used statin monotherapy only. The synergistic effect of combination therapy was naturally producing superior lipid lowering compared to dose escalation alone.

Current study showed particularly poor outcomes in moderate intensity group with 79 (83.2%) patients failing to achieve targets which was consistent with alarming findings of Al Shibli ES *et al.* [19] where 85% patients failed reaching LDL-C <1.4mmol/L at 1-year follow-up, and Toth S *et al.*[20] reporting that over 90% of very high-risk patients was not achieving target levels. These consistently poor results across different populations was indicating systematic problem in lipid management rather than regional variation. The widespread failure was suggesting that moderate intensity approach was fundamentally inadequate for post-ACS patients who require aggressive risk reduction, and that current guideline recommendations was not being implemented effectively in clinical practice. The age-stratified analysis in present study revealed that patients >50 years achieved better efficacy in high intensity group at 45 (59.2%) versus 11 (15.1%) with p=0.004, which was aligning with mean age of 60.5 years in Tungsubutra W *et al.* [21] where only 30.1% achieved targets due to suboptimal dosing. The elderly population was requiring more aggressive therapy because age-related metabolic changes including decreased hepatic function and altered drug metabolism was necessitating higher doses, yet many clinicians was hesitating to prescribe intensive therapy in elderly due to unfounded concerns about adverse effects. Smoking status analysis in current study was showing dramatic difference where smokers on high intensity statins achieved 16 (76.2%) efficacy versus only 1 (5.9%) on moderate intensity (p=0.021). This finding was not specifically addressed in comparative studies but Hong SJ *et al.* [22] demonstrated that treat-to-target strategy was noninferior to high-intensity approach with 8.1% versus 8.7% composite

endpoints, suggesting that achieving specific LDL-C goals regardless of statin intensity was more important than fixed high-dose approach. However, in smokers specifically, the increased metabolic clearance and oxidative stress was making fixed moderate doses completely inadequate as demonstrated by present study's poor outcomes. The comorbidity analysis showing hypertensive patients achieving 28 (65.1%) efficacy on high intensity versus 4 (16.0%) on moderate intensity ($p<0.001$) was supporting findings of Almaramihi WS *et al.* [23] where 83% patients achieved LDL-C goals when 91% received high-potency statins. The Saudi study was demonstrating better overall success rates possibly because their definition of goal achievement was less stringent at 70mg/dL, and their high utilization of atorvastatin 40mg was providing consistent therapeutic effect. The presence of multiple comorbidities was creating compounding cardiovascular risk requiring more aggressive intervention, and moderate intensity therapy was simply insufficient for managing this complex pathophysiology.

This study also has some limitations that must be considered. To begin with, this is a single center study, which means that its findings cannot be generalize to larger groups. The study also used only 190 patients, which is considered to be a small size. The duration of this study is only three months. During this time, long-term outcomes such as cardiovascular events and deaths have not been considered. Finally, medication adherence is also not monitored during this period, which is considered to be a confounding variable.

Conclusion:

The present study has concluded that high intensity statin therapy was significantly more effective than moderate intensity statin therapy in achieving target LDL-C level among patient with acute coronary syndrome. The superiority of high intensity statin was consistently observed across various demographic subgroup including different age group, smoking status, comorbidity patterns and residential locations.

Disclaimer:

No disclaimer is applied.

References:

1. Theofilis P, Oikonomou E, Chasikidis C, Tsioufis K, Tousoulis D. Pathophysiology of acute coronary syndromes—diagnostic and treatment considerations. *Life (Basel).* 2023;**13(7)**:1543. DOI: https://doi.org/10.3390/life13071543
2. Bergmark BA, Mathenge N, Merlini PA, Lawrence-Wright MB, Giugliano RP. Acute coronary syndromes. *Lancet.* 2022;**399(10332)**:1347-1358. DOI: https://doi.org/10.1016/S0140-6736(21)02391-6
3. Dorjey Y, Tshering U, Watts MR. Clinical characteristics and outcomes of acute coronary syndrome patients in a PCI-limited setting: a prospective study from Bhutan. *BMC Cardiovasc Disord.* 2025;**25**:324. DOI: https://doi.org/10.1186/s12872-025-04782-w
4. Huang X, Bai S, Luo Y. Advances in research on biomarkers associated with acute myocardial infarction: A review. *Medicine (Baltimore).* 2024;**103(15)**:e37793. doi: 10.1097/MD.0000000000037793
5. Wang Q, Liang C. Role of Lipid-Lowering Therapy in Low-Density Lipoprotein Cholesterol Goal Attainment: Focus on Patients With Acute Coronary Syndrome. *J Cardiovasc Pharmacol.* 2020 Dec;**76(6):**658-670. DOI: https://doi.org/10.1097/FJC.0000000000000914.
6. Ray S. Role of statins in the management of dyslipidaemia. *Indian Heart J.* 2024 Mar;**76 Suppl 1(Suppl 1)**:S33-S37. DOI: https://doi.org/10.1016/j.ihj.2023.11.267.
7. Mhaimeed O, Burney ZA, Schott SL, Kohli P, Marvel FA, Martin SS. The importance of LDL-C lowering in

atherosclerotic cardiovascular disease prevention: lower for longer is better. *Am J Prev Cardiol*. 2024;**18**:100649. DOI: https://doi.org/10.1016/j.ajpc.2024.100649

9. Bakour HA, Timraz JH, Bin Saddiq BW, Alghamdi NA, Thalib HI, Alyarimi M, *et al.* Familial hypercholesterolemia: a comprehensive review of advances in treatment strategies and the role of patient beliefs. *Cureus*. 2025;**17(1)**:e78032. DOI: https://doi.org/10.7759/cureus.78032
10. Arnold N, Koenig W. Lipid lowering drugs in acute coronary syndromes (ACS). *Curr Atheroscler Rep*. 2023;**25(12)**:939-946. DOI: https://doi.org/10.1007/s11883-023-01163-6
11. Khatiwada N, Hong Z. Potential benefits and risks associated with the use of statins. *Pharmaceutics*. 2024;**16(2)**:214. DOI: https://doi.org/10.3390/pharmaceutics16020214
12. Pradhan A, Sharma P, Prajapathi S, Aracri M, Iellamo F, Perrone MA. Intensive lipid-lowering therapy following acute coronary syndrome: the earlier the better. *J Cardiovasc Dev Dis*. 2025;**12(8)**:300. DOI: https://doi.org/10.3390/jcdd12080300
13. Ch IA, Nasir K, Majeed U, Chaudhry A, Abdullah M, Haider A, *et al.* Effect of statin intensity on cardiovascular outcomes and survival following coronary artery bypass grafting. *Clin Cardiol*. 2025;**48(7)**:e70170. DOI: https://doi.org/10.1002/clc.70170
14. Kim AR, Han JY, Kim M, Lee H, Baek YH, Ahn I, *et al.* Cardiovascular outcomes of early LDL-C goal achievement in patients with very-high-risk ASCVD. *Cardiol Ther*. 2025;**14(1)**:101-115. DOI: https://doi.org/10.1007/s40119-025-00397-6
15. Choo EH, Kim CJ, Hwang BH, Lee KY, Oh GC, Lim S, *et al.* High-intensity statin versus upfront equivalent-dose combination of moderate-intensity statin with ezetimibe following acute myocardial infarction (ROSUZET-AMI): protocol of a multicentre, open-label, randomised non-inferiority trial. *BMJ Open*. 2025;**15(11)**:e104127. DOI: https://doi.org/10.1136/bmjopen-2025-104127
16. Thongtang N, Sitthananun C, Sriussadaporn S, Nitiyanant W. Efficacy of low- and moderate-intensity statins for achieving low-density lipoprotein cholesterol targets in Thai type 2 diabetic patients. *J Diabetes Metab Disord*. 2017;**16**:6. DOI: https://doi.org/10.1186/s40200-017-0290-x
17. Dai YY, Zhang HS, Zhang XG, *et al.* Statin-ezetimibe versus statin lipid-lowering therapy in patients with acute coronary syndromes undergoing percutaneous coronary intervention. *J Thorac Dis*. 2017;**9(5)**:1345-1352. DOI: https://doi.org/10.21037/jtd.2017.05.46
18. Chinwong D, Patumanond J, Chinwong S, *et al.* Statin therapy in patients with acute coronary syndrome: low-density lipoprotein cholesterol goal attainment and effect of statin potency. *Ther Clin Risk Manag*. 2015;**11**:127-136. DOI: https://doi.org/10.2147/TCRM.75608
19. Lee J, Lee SH, Kim H, *et al.* Low-density lipoprotein cholesterol reduction and target achievement after switching from statin monotherapy to statin/ezetimibe combination therapy: real-world evidence. *J Clin Pharm Ther*. 2021 Feb;**46(1)**:134-142. doi: 10.1111/jcpt.13271.
20. Al Shibli ES, Prashanth P. Low-density lipoprotein cholesterol level on admission and after one year with clinical outcomes in patients admitted with acute coronary syndrome undergoing percutaneous coronary

intervention. *Heart Views*. 2025;**26(2)**:80-87. DOI: https://doi.org/10.4108/journal.hv.2025.00013

21. Toth S, Turek M, Pella D. Success in achieving LDL-C target values in a high-risk population in Slovakia: the SlovakLipid retrospective study. *Arch Med Sci*. 2025;**21(3)**:738-746. DOI: https://doi.org/10.5114/aoms/170961
22. Tungsubutra W, Phongtuntakul B. Achievement of LDL-cholesterol goal with statins after an ST segment elevation myocardial infarction. *J Med Assoc Thai*. 2015;**98(2)**:129-136.
23. Hong SJ, Lee YJ, Lee SJ, *et al.* Treat-to-target or high-intensity statin in patients with coronary artery disease: a randomized clinical trial. *JAMA*. 2023;**329(13)**:1078–1087. DOI: https://doi.org/10.1001/jama.2023.2487
24. Almaramihi WS, Zahrani M, Kamfar R. Lipid lowering agent prescription and LDL target achievement in ACS patients in Jeddah, Saudi Arabia. *Int J Med Dev Ctries*. 2023;**7(11)**:1436-1445. https://doi.org/10.24911/IJMDC.51-1685365843