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ORIGINAL PAPER

LOW-DENSITY LIPOPROTEIN CHOLESTEROL TARGET ATTAINMENT AND ASSOCIATED FACTORS AFTER PERCUTANEOUS CORONARY INTERVENTION FOR NON–ST-ELEVATION ACUTE CORONARY SYNDROME: A REAL-WORLD STUDY

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Abstract

Introduction. Despite advances in lipid-lowering therapy (LLT), low-density lipoprotein cholesterol (LDL-C) control remains suboptimal after non–ST-elevation acute coronary syndrome (NSTE-ACS), particularly following percutaneous coronary intervention (PCI). Real-world data on LDL-C target attainment according to contemporary guidelines remain limited.
The objective of the study was to evaluate real-world LDL-C target attainment according to the 2019 European Society of Cardiology (ESC)/ European Atherosclerosis Society (EAS) guidelines and to identify factors associated with LDL-C target non-achievement in patients with NSTE-ACS treated with PCI.
Material and methods. This single-center observational study included 144 consecutive patients with NSTE-ACS (mean age 67.8 years; 61.8% male) who underwent PCI. Lipid profiles were measured at admission and during follow-up (median 4 months). Failure to achieve the LDL-C target was defined as LDL-C

Résumé

Atteinte des objectifs de cholestérol LDL et facteurs associés après une intervention coronarienne percutanée pour un syndrome coronarien aigu sans sus-décalage du ST: une étude en conditions réelles

Introduction. Malgré les progrès des traitements hypolipémiants (LLT), le contrôle du cholestérol à lipoprotéines de basse densité (LDL-C) demeure insuffisant après un syndrome coronarien aigu sans sus-décalage du ST (SCA NST), en particulier après une intervention coronarienne percutanée (ICP). Les données en vie réelle sur l'atteinte des objectifs de LDL-C selon les recommandations actuelles restent limitées.
L'objectif de cette étude était d'évaluer en conditions réelles l'atteinte des objectifs de LDL-C selon les recommandations ESC/EAS 2019 et d'identifier les facteurs associés à la non-atteinte de ces objectifs chez des patients présentant un SCA NST traités par ICP.

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≥1.4 mmol/L at follow-up. Post-discharge LLT changes were recorded. Independent factors associated with LDL-C target non-achievement were assessed using multivariable logistic regression.
Results. At baseline, 8.3% of patients had LDL-C <1.4 mmol/L. At follow-up, 18.1% achieved LDL-C <1.4 mmol/L, and 6.3% achieved both LDL-C <1.4 mmol/L and a ≥50% reduction from baseline. LLT was modified in 33.3% of patients. Higher baseline LDL-C (odds ratio [OR] 3.12; 95% confidence interval [CI] 1.64–5.96; p=0.001) and higher body mass index (OR 1.30; 95% CI 1.06–1.61; p=0.013) were independently associated with LDL-C target non-achievement, whereas high-intensity statin therapy at discharge (OR 0.15; 95% CI 0.03–0.82; p=0.029) and reduced renal function (estimated glomerular filtration rate <60 mL/min/1.73 m^2; OR 0.32; 95% CI 0.12–0.91; p=0.032) were associated with lower odds of failing to achieve LDL-C targets.
Conclusions. In patients with NSTE-ACS undergoing PCI, LDL-C target attainment according to the guidelines remains low. Maintaining sufficiently intensive LLT after discharge may help improve LDL-C target attainment in this high-risk population.

Keywords: low-density lipoprotein cholesterol, lipid-lowering therapy, non–ST-elevation acute coronary syndrome, percutaneous coronary intervention, statin.

List of abbreviations:
ACS: acute coronary syndrome
BMI: body mass index
CAD: coronary artery disease
CI: confidence interval
CKD: chronic kidney disease
EAS: European Atherosclerosis Society
eGFR: estimated glomerular filtration rate
ESC: European Society of Cardiology
hs-TnT: high-sensitivity cardiac troponin T
IQR: interquartile range
LDL-C: low-density lipoprotein cholesterol
LLT: lipid-lowering therapy
NSTE-ACS: non–ST-elevation acute coronary syndrome
OR: odds ratio
PCI: percutaneous coronary intervention
PCSK9: proprotein convertase subtilisin/kexin type 9
SD: standard deviation

Matériel et méthodes. Cette étude observationnelle monocentrique a inclus 144 patients consécutifs atteints de SCA NST (âge moyen 67,8 ans; 61,8% d'hommes) ayant bénéficié d'une ICP. Les profils lipidiques ont été mesurés à l'admission puis au cours du suivi (médiane 4 mois). L'absence d'atteinte de la cible de LDL-C était définie par LDL-C ≥1,4 mmol/L au suivi. Les modifications de la LLT après la sortie hospitalière ont été recensées. Les facteurs indépendants associés à la non-atteinte de la cible de LDL-C ont été évalués par régression logistique multivariée.
Résultats. À l'inclusion, 8,3% des patients présentaient LDL-C <1,4 mmol/L. Au suivi, 18,1% ont atteint LDL-C <1,4 mmol/L et 6,3% ont obtenu à la fois LDL-C <1,4 mmol/L et une réduction ≥50% par rapport à la valeur initiale. La LLT a été modifiée chez 33,3% des patients. Un LDL-C de base plus élevé (odds ratio [OR] 3,12 ; intervalle de confiance [IC] 95% : 1,64–5,96 ; p=0,001) et un indice de masse corporelle plus important (OR 1,30 ; IC 95% : 1,06–1,61 ; p=0,013) étaient indépendamment associés à la non-atteinte de la cible, tandis qu'un traitement par statine à forte intensité à la sortie (OR 0,15 ; IC 95% : 0,03–0,82 ; p=0,029) et une fonction rénale diminuée (débit de filtration glomérulaire estimé <60 mL/min/1,73 m^2 ; OR 0,32 ; IC 95% : 0,12–0,91 ; p=0,032) étaient associés à une moindre probabilité de non-atteinte des objectifs de LDL-C.
Conclusions. Chez les patients présentant un SCA NST traités par ICP, l'atteinte des objectifs de LDL-C selon les recommandations ESC/EAS 2019 reste faible en pratique courante. Le maintien d'une LLT suffisamment intensive après la sortie pourrait contribuer à améliorer l'atteinte des objectifs de LDL-C dans cette population à haut risque.

Mots-clés: cholestérol à lipoprotéines de basse densité, traitement hypolipémiant, syndrome coronarien aigu sans élévation du ST, intervention coronarienne percutanée, statine.

Introduction

Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide, with acute coronary syndrome (ACS) representing one of its most severe clinical manifestations.[1] Among patients with ACS, those presenting with non–ST-elevation acute coronary syndrome (NSTE-ACS) constitute a heterogeneous population characterized by high short- and long-term risks of recurrent ischemic events despite advances in revascularization strategies and pharmacological therapy.[2–5]

Low-density lipoprotein cholesterol (LDL-C) plays a central role in the pathogenesis and

progression of atherosclerotic cardiovascular disease. Robust evidence from randomized controlled trials and meta-analyses has consistently demonstrated that intensive LDL-C lowering significantly reduces the risk of major adverse cardiovascular events in both primary and secondary prevention settings.[6] In patients with ACS, early and sustained LDL-C reduction has been shown to improve plaque stability, reduce recurrent ischemic events, and improve long-term prognosis.[6]

In recognition of this evidence, the 2019 European Society of Cardiology (ESC)/European Atherosclerosis Society (EAS) guidelines for the management of dyslipidemias classified all patients with ACS as being at very high cardiovascular risk.[6] These guidelines recommend a stringent LDL-C target of <1.4 mmol/L (<55 mg/dL), along with at least a 50% reduction from baseline levels. Achieving these goals often requires early initiation of high-intensity statin therapy, timely treatment intensification, and, in selected patients, combination therapy with non-statin lipid-lowering agents. Despite clear guideline recommendations, multiple real-world studies have shown that attainment of LDL-C targets after ACS remains suboptimal. Large international registries have reported low rates of LDL-C goal achievement, even in high-income countries with well-established healthcare systems.[7,8] Factors contributing to this gap between guidelines and clinical practice include insufficient statin intensity, delayed treatment intensification, limited use of combination lipid-lowering therapy (LLT), comorbid conditions, and suboptimal long-term follow-up.

Data from low- and middle-income countries are particularly limited.[9–12] In Southeast Asia, including Vietnam, the burden of CAD is increasing rapidly, while access to comprehensive secondary prevention strategies remains variable. Previous studies conducted in Vietnam[13,14] and neighboring countries have reported modest rates of LDL-C target achievement, often based on older guideline thresholds and heterogeneous patient populations.[9–12] Moreover, few studies have specifically focused on patients with NSTE-ACS undergoing percutaneous coronary intervention (PCI), a subgroup in whom optimal lipid control is crucial for preventing recurrent events.[10,15] In addition, the determinants of LDL-C target non-achievement in real-world practice remain incompletely understood.[7] While baseline LDL-C levels and statin intensity are recognized predictors, the roles of clinical characteristics are less consistent across studies.[7,8,10] Understanding these factors is essential to identify high-risk patients who may benefit from early and more aggressive lipid-lowering strategies.

The objective of the study was to evaluate real-world LDL-C target attainment according to the 2019 ESC/EAS guidelines and to identify factors associated with LDL-C target non-achievement in patients with NSTE-ACS treated with PCI.

Materials and methods

Study Design and participants

This was a single-center ambispective observational cohort study conducted at Tam Duc Cardiology Hospital, a specialized cardiology hospital in Vietnam. Adult patients aged 18 years or older were eligible for inclusion if they were diagnosed with NSTE-ACS and underwent PCI during the index hospitalization and had at least one documented LDL-C measurement obtained during follow-up after hospital discharge. NSTE-ACS was diagnosed according to the 2023 ESC guidelines for the management of ACS.[16] Patients with ST-elevation myocardial infarction, those who did not undergo PCI, patients who died during the index hospitalization, or those without follow-up lipid profile data were excluded.

Consecutive patients meeting these criteria were identified retrospectively, and LDL-C levels were prospectively monitored after discharge. Clinical, laboratory, angiographic, and treatment-related data were obtained from electronic medical records and standardized outpatient follow-up visits. The study was conducted in accordance with the principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of Tam Duc Cardiology Hospital (approval no. 23.24/GCN-BVTĐ, October 4, 2024).

Clinical and Laboratory Variables

Baseline demographic characteristics included age and sex. Cardiovascular risk factors and comorbidities were defined based on documented medical history and standard clinical criteria, including hypertension, diabetes mellitus, dyslipidemia, prior myocardial infarction, and smoking status. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2) and categorized according to Asia–Pacific criteria: normal weight (18.5–22.9 kg/m^2), overweight (23.0–24.9 kg/m^2), and obesity (≥25.0 kg/m^2).[17] Baseline laboratory measurements included fasting lipid profiles, glucose indices, cardiac biomarkers, and renal function parameters. High-sensitivity cardiac troponin T (hs-TnT) was measured at presentation and repeated after 3 hours when indicated, using an automated immunoassay on the ARCHITECT system (Abbott Diagnostics, USA), with a 99th percentile upper reference limit of 14 pg/mL.

Baseline LDL-C was defined as the first available LDL-C measurement obtained during index hospitalization or prior to initiation of intensive LLT. Renal function was assessed using estimated glomerular filtration rate (eGFR), calculated with the CKD-EPI equation. Coronary angiography findings were reviewed by experienced interventional cardiologists. Multivessel CAD was defined as the presence of ≥70% stenosis in two or more major epicardial coronary arteries or ≥50% stenosis of the left main coronary artery.

Lipid-Lowering Therapy and Treatment Modification

LLT at hospital discharge and during follow-up was classified according to statin intensity and the use of combination therapy. High-intensity statin therapy was defined as atorvastatin 40–80 mg/day or rosuvastatin 20–40 mg/day. Moderate-intensity statin therapy included atorvastatin 10–20 mg/day, rosuvastatin 5–10 mg/day, simvastatin 20–40 mg/day, pravastatin 40–80 mg/day, lovastatin 40 mg/day, fluvastatin extended-release 80 mg/day or 40 mg twice daily, and pitavastatin 2–4 mg/day. Low-intensity statin therapy comprised simvastatin 10 mg/day, pravastatin 10–20 mg/day, lovastatin 20 mg/day, fluvastatin 20–40 mg/day, and pitavastatin 1 mg/day.[18] In the present study, LLT consisted predominantly of atorvastatin and rosuvastatin; use of other statins was infrequent. Combination therapy was defined as statin treatment in combination with ezetimibe.

Changes in LLT during follow-up were categorized into three predefined strategies. Treatment intensification was defined as an increase in statin intensity from moderate- to high-intensity statin therapy and/or the addition of ezetimibe to any statin regimen. Treatment de-escalation was defined as a reduction in statin intensity from high-intensity statin therapy or combination therapy to moderate-intensity statin therapy. Patients whose lipid-lowering regimen remained unchanged from hospital discharge to the end of follow-up were classified as having no treatment modification.

Follow-Up and Outcome Definition

Patients were followed during routine outpatient visits. Follow-up duration was defined as the interval from hospital discharge to the most recent lipid profile measurement and varied across patients. The primary outcome was attainment of LDL-C <1.4 mmol/L at the end of follow-up, in accordance with the 2019 ESC/EAS guidelines for patients at very high cardiovascular risk.[6] Additional lipid parameters, including ≥50% LDL-C reduction from baseline and combined attainment of LDL-C <1.4 mmol/L with ≥50% reduction, were recorded descriptively and reported in the results but were not used as outcomes in regression analyses.

Statistical Analysis

Continuous variables were assessed for normality and are presented as mean ± standard deviation or median with interquartile range, as appropriate. Categorical variables are presented as counts and percentages. Comparisons between patients who achieved and did not achieve the LDL-C target were performed using Student's t-test or Mann–Whitney U test for continuous variables and chi-square test or Fisher's exact test for categorical variables. Univariable logistic regression was used to identify factors associated with failure to achieve LDL-C <1.4 mmol/L. Variables with a p-value <0.20 in univariable analysis and/or clinical relevance were entered into a multivariable logistic regression model. To minimize multicollinearity, correlated lipid variables were not entered simultaneously. Multicollinearity was assessed using variance inflation factors. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported. A two-sided p-value <0.05 was considered statistically significant. Statistical analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA).

Results

Study population and baseline characteristics

A total of 144 patients were included. The mean age was 67.8±12.5 years, and 61.8% were male. Hypertension (63.9%), dyslipidemia (46.5%), diabetes mellitus (37.5%), and obesity (35.4%) were common. Multivessel CAD was present in 61.1% of patients (Table 1).

Table 1. Baseline clinical characteristics, lipid profiles, and lipid-lowering therapy of the study population

Variable	*Total cohort (n = 144)*
Age, years (mean ± SD)	67.8 ± 12.5
Male sex, n (%)	89 (61.8)
BMI, kg/m² (mean ± SD)	23.6 ± 3.2
BMI category, n (%)	
Normal weight	40 (27.8)
Overweight	53 (36.8)
Obesity	51 (35.4)

Variable	*Total cohort (n = 144)*
Hypertension, n (%)	92 (63.9)
Diabetes mellitus, n (%)	54 (37.5)
Dyslipidemia, n (%)	67 (46.5)
Prior myocardial infarction, n (%)	36 (25.0)
Prior stroke, n (%)	6 (4.2)
Prior PCI, n (%)	23 (16)
Heart failure, n (%)	21 (14.6)
Atrial fibrillation, n (%)	7 (4.9)
Current smoking, n (%)	43 (29.9)
GRACE score, median (IQR)	132 (118–178)
Killip class ≥ II, n (%)	59 (41.0)
LVEF, % (median, IQR)	50 (35–63)
Single-vessel disease, n (%)	45 (31.3)
Multivessel disease, n (%)	88 (61.1)
Laboratory findings at baseline	
Fasting glucose, mmol/L (median, IQR)	6.38 (5.50–8.15)
HbA1c, % (median, IQR)	7.20 (5.50–8.60)
NT-proBNP, pg/mL (median, IQR)	905.7 (240–3150)
hs-TnT (0h), pg/mL (median, IQR)	193 (42.2–695.5)
hs-TnT (3h), pg/mL (median, IQR)	230 (84 – 833)
Creatinine, µmol/L (median, IQR)	92.5 (78–111.8)
eGFR, mL/min/1.73 m² (median, IQR)	58 (45–90)
eGFR <60 mL/min/1.73 m², n (%)	77 (53.5)
ALT, U/L (median, IQR)	27 (23–38.3)
AST, U/L (median, IQR)	30 (24–41)
Lipid profiles at baseline	
Total cholesterol, mmol/L (median, IQR)	4.30 (3.46–5.41)
HDL-C, mmol/L (median, IQR)	1.17 (1.03–1.38)
Triglycerides, mmol/L (median, IQR)	1.76 (1.25–2.51)
LDL-C, mmol/L (median, IQR)	2.60 (1.93–3.30)
LDL-C target attainment (<1.4 mmol/L), n (%)	12 (8.3)
Lipid profiles at follow-up	
Total cholesterol, mmol/L (median, IQR)	3.72 (3.02–4.45)
HDL-C, mmol/L (median, IQR)	1.19 (1.02–1.35)
Triglycerides, mmol/L (median, IQR)	1.74 (1.22–2.25)
LDL-C, mmol/L (median, IQR)	1.9 (1.4–2.3)
LDL-C target attainment (<1.4 mmol/L), n (%)	26 (18.1)
≥50% LDL-C reduction from baseline, n (%)	26 (18.1)
Both LDL-C <1.4 mmol/L and ≥50% reduction	9 (6.3)
Lipid-lowering therapy at discharge	
High-intensity statin	113 (78.5)
Moderate-intensity statin	22 (15.3)
Moderate statin + ezetimibe	4 (2.8)
High-intensity statin + ezetimibe	5 (3.5)
Fenofibrate	0
Lipid-lowering therapy at follow-up	
High-intensity statin	81 (56.3)
Moderate-intensity statin	35 (24.3)
Moderate statin + ezetimibe	14 (9.7)
High-intensity statin + ezetimibe	13 (9.0)
Fenofibrate	1 (0.6)

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; eGFR, estimated glomerular filtration rate; GRACE, Global Registry of Acute Coronary Events; HDL-C, high-density lipoprotein cholesterol; hs-TnT, high-sensitivity cardiac troponin; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro–B-type natriuretic peptide; PCI, percutaneous coronary intervention; SD, standard deviation.

Table 2. Treatment modification during follow-up

Variable	*Total cohort (n = 144)*
Treatment modification strategy	
No change from discharge therapy, n (%)	96 (66.7)
Treatment intensification*, n (%)	24 (16.7)
Treatment de-escalation†, n (%)	23 (16.0)
Switch to fenofibrate, n (%)	1 (0.6)

*Treatment intensification was defined as escalation from moderate- to high-intensity statin therapy and/or addition of ezetimibe. †Treatment de-escalation was defined as reduction from high-intensity statin therapy or combination therapy to moderate-intensity statin therapy.

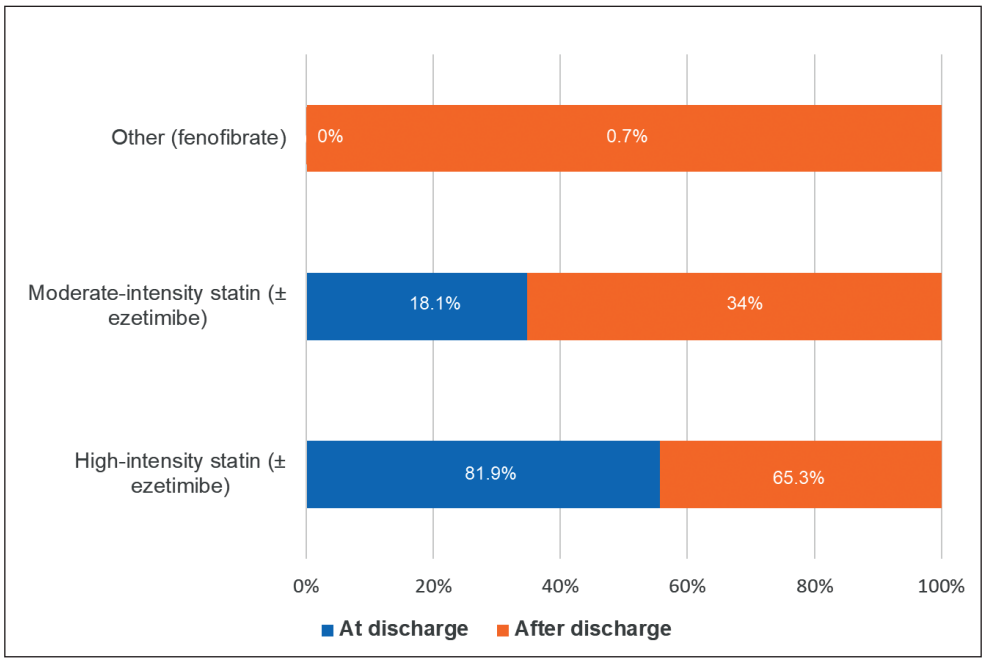


Figure 1. Changes in lipid-lowering therapy intensity from hospital discharge to follow-up. The distribution of lipid-lowering therapy shifted over time after discharge. The proportion of patients receiving high-intensity statin–based regimens decreased from 81.9% at discharge to 65.3% during follow-up, whereas use of moderate-intensity statins increased from 18.1% to 34.0%. Combination therapy with ezetimibe increased after discharge, while use of non-statin therapy (fenofibrate) remained minimal.

Median follow-up for lipid assessment was 4 months (IQR 3–7; range 1–24 months). At baseline, 8.3% of patients had LDL-C <1.4 mmol/L. At follow-up, 18.1% achieved LDL-C <1.4 mmol/L, 18.1% achieved a ≥50% LDL-C reduction, and 6.3% met both recommended targets.

Lipid-lowering therapy and treatment modification

All patients received statin therapy during hospitalization. At discharge, 81.9% were treated with high-intensity statin–based regimens, while 6.3% received combination therapy with ezetimibe.

During follow-up, substantial treatment changes were observed. The proportion of patients on high-intensity statins decreased from 81.9% at discharge to 65.3%, whereas combination therapy with ezetimibe increased from 6.3% to 18.7%. Overall, LLT was modified in one-third of patients: treatment intensification occurred in 16.7%, de-escalation in 16.0%, and no change in 66.7% (Table 2, Figure 1).

Factors associated with failure to achieve LDL-C targets

In univariate logistic regression analysis, higher BMI, hypertension, higher baseline LDL-C and total cholesterol and impaired renal function were associated with failure to achieve LDL-C <1.4 mmol/L.

In multivariate logistic regression analysis, baseline LDL-C (OR 3.12; 95% CI 1.64–5.96) and BMI (OR 1.30; 95% CI 1.06–1.61) were independent predictors of LDL-C target non-achievement. In contrast, high-intensity statin therapy at discharge (OR 0.15; 95% CI 0.03–0.82) and eGFR <60 mL/min/1.73 m^2 (OR 0.32; 95% CI 0.12–0.91) were independently

Table 3. Factors associated with failure to achieve LDL-C target (<1.4 mmol/L) in univariate and multivariate logistic regression

Variable	*Univariate analysis*		*Multivariate analysis*	
	OR (95% CI)	*p value*	*OR (95% CI)*	*p-value*
Age (per year)	1.01 (0.98–1.04)	0.618	–	–
Male sex	1.01 (0.42–2.43)	0.975	–	–
Hypertension	2.46 (1.04–5.82)	0.041	–	–
BMI (per kg/m²)	1.32 (1.08–1.61)	0.006	1.30 (1.06–1.61)	**0.013**
Baseline LDL-C (per mmol/L)	2.88 (1.58–5.18)	<0.001	3.12 (1.64–5.96)	**0.001**
Total cholesterol (per mmol/L)	2.01 (1.32–3.05)	0.001	–	–
eGFR (per mL/min/1.73 m²)	0.97 (0.96–0.99)	0.008	–	–
eGFR <60 mL/min/1.73 m²	2.57 (1.06–6.24)	0.037	0.32 (0.12–0.91)	**0.032**
High-intensity statin at discharge	3.06 (0.68–13.88)	0.146	0.15 (0.03–0.82)	**0.029**

Abbreviations: BMI, body mass index; CI, confidence interval; eGFR, estimated glomerular filtration rate; LDL-C, low-density lipoprotein cholesterol; OR, odds ratio.

associated with a lower likelihood of LDL-C target non-achievement (Table 3).

Discussion

To the best of our knowledge, this is one of the first real-world studies from Vietnam to evaluate LDL-C target attainment according to the 2019 ESC/EAS guidelines in patients with NSTE-ACS undergoing PCI. The principal finding of this study is that LDL-C target attainment remained low, with fewer than one in five patients achieving LDL-C <1.4 mmol/L during follow-up, despite widespread use of statin-based lipid-lowering therapy. In addition, we identified several clinical and treatment-related factors associated with LDL-C target non-achievement, including higher baseline LDL-C levels and higher BMI, whereas high-intensity statin therapy at discharge and reduced renal function were associated with a lower likelihood of failing to achieve LDL-C goals. Patterns of post-discharge modification of LLT, including both treatment intensification and de-escalation, were also observed but were not the primary focus of the multivariable analysis.

Our results are broadly consistent with prior international registries reporting suboptimal LDL-C control following ACS and PCI. In the DYSIS (*Dyslipidaemia International Study*) II,[11] only 18.9% of patients with ACS achieved LDL-C <1.8 mmol/L after approximately four months, despite high rates of statin use and a less stringent target than current ESC/EAS recommendations. Similarly, Harris et al. reported that only 23.4% of patients undergoing PCI achieved LDL-C <1.4 mmol/L within one year, despite more than half receiving high-intensity statin therapy.[7] Together, these studies indicate that inadequate LDL-C control remains a global challenge, even in healthcare systems with well-established secondary prevention strategies.

Studies from East Asia provide additional context. Zhang et al. reported a 48% attainment rate of LDL-C <1.8 mmol/L after one year in Chinese patients with ACS undergoing PCI,[10] while the DYSIS II–China study documented a 41.2% attainment rate at six months.[11] These higher rates are likely attributable to less aggressive LDL-C targets and longer follow-up durations, highlighting the increasing difficulty of achieving the more stringent ESC/EAS 2019 goals in routine clinical practice. More recent European registries suggest that substantially higher attainment rates are achievable when early combination lipid-lowering strategies are adopted. In a multicenter Italian registry, more than half of patients achieved LDL-C <1.4 mmol/L within three months, driven by widespread use of ezetimibe and proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors at discharge.[15] In contrast, the limited use of combination therapy in our cohort, similar to patterns observed in other Asian country settings,[9] likely contributed to the modest LDL-C control observed.

Vietnamese data further reinforce the consistency of our findings. Previous studies have reported LDL-C target attainment rates ranging from 14% to 36.8%, depending on follow-up duration and patient population, even when applying contemporary guideline thresholds.[13,14,19] Collectively, these data indicate that low LDL-C target attainment after ACS is not unique to our cohort but reflects a broader and persistent implementation gap.

A novel and clinically relevant observation of our study is the high frequency of post-discharge modification in LLT. Although most patients were discharged on high-intensity statin–based regimens, one-third experienced treatment changes during

follow-up, with treatment intensification and de-escalation occurring at similar rates. This dynamic pattern of LLT has been insufficiently characterized in previous studies. Most large registries and observational studies have primarily reported LDL-C target attainment at predefined follow-up time points, without systematically examining longitudinal changes in treatment intensity after hospital discharge. For example, DYSIS II and EUROASPIRE (*European Action on Secondary and Primary Prevention by Intervention to Reduce Events*) V surveys documented statin use and LDL-C levels during follow-up but did not explicitly analyze treatment escalation or de-escalation over time.[11,20] Similarly, observational data from real-world cohorts suggest that clinical inertia, delayed intensification, and premature statin down titration are common after ACS, yet these patterns have rarely been quantified in detail.[21]

Our findings indicate that early prescription of high-intensity statins does not necessarily translate into sustained intensive lipid lowering in real-world practice. Treatment de-escalation occurred as frequently as intensification, suggesting that post-discharge lipid management is highly dynamic and influenced by factors such as tolerability, clinician judgement, and perceived risk. Similar patterns of statin down titration after ACS have been indirectly suggested in national registries, including SWEDEHEART (*Swedish Web-system for Enhancement and Development of Evidence-based care in Heart disease Evaluated According to Recommended Therapies*) and other real-world cohorts, but without detailed quantification.[7,20] Taken together, these findings suggest that LDL-C target non-achievement is driven not only by inadequate treatment initiation but also by failure to sustain intensive lipid-lowering strategies after discharge. Structured follow-up, early LDL-C reassessment, and protocol-driven treatment escalation may be critical to prevent premature de-escalation and improve long-term lipid goal attainment in patients with NSTE-ACS undergoing PCI.[6,22] Importantly, de-escalation from high-intensity statin therapy may attenuate cumulative LDL-C reduction during the early post-PCI period, a phase characterized by particularly high residual cardiovascular risk.[7] This phenomenon may partly explain the persistently low rate of LDL-C target attainment observed in our cohort despite widespread statin initiation at discharge.

Determinants of LDL-C target non-achievement

In the present study, baseline LDL-C level emerged as the strongest independent predictor of failure to achieve LDL-C <1.4 mmol/L during follow-up. This finding is consistent with prior observational studies and registry data demonstrating that patients with higher initial LDL-C face greater difficulty in reaching stringent lipid targets, even when treated with high-intensity statins. Zhang et al. reported that lower baseline LDL-C was the most powerful predictor of LDL-C target attainment one year after PCI in patients with ACS (OR 4.81; 95% CI 3.46–6.70).[10] Similarly, analyses from DYSIS II showed that baseline lipid burden substantially influenced goal attainment across both stable CAD and ACS populations.[11]

Higher BMI was another independent predictor of LDL-C target non-achievement in our cohort. Obesity is known to be associated with adverse lipid profiles, increased hepatic cholesterol synthesis, insulin resistance, and chronic low-grade inflammation, all of which may attenuate the lipid-lowering response to statin therapy.[23] Previous studies have reported inconsistent associations between obesity and LDL-C target attainment. In DYSIS II, obesity paradoxically appeared to be associated with higher odds of LDL-C target achievement, possibly reflecting more aggressive treatment or closer follow-up in obese patients.[11] In contrast, our findings suggest that higher BMI remains a barrier to optimal lipid control, particularly when combination LLT is underutilized.

Renal dysfunction demonstrated a complex relationship with LDL-C target attainment in our study. In univariable analyses, reduced renal function (lower eGFR and eGFR <60 mL/min/1.73 m^2) was associated with failure to achieve LDL-C targets, consistent with prior reports indicating that chronic kidney disease (CKD) is linked to dyslipidemia, altered lipoprotein metabolism, and poorer response to LLT.[11,24] Indeed, DYSIS II identified CKD as an independent factor associated with lower odds of achieving LDL-C targets.[11] Interestingly, after multivariable adjustment, eGFR <60 mL/min/1.73 m^2 was independently associated with a lower likelihood of LDL-C target non-achievement in our cohort. This apparent paradox may reflect treatment-related confounding. Patients with CKD are generally classified as being at very high cardiovascular risk and may therefore receive more intensive LLT, closer follow-up, and stricter adherence to guideline-recommended targets.[24] Similar reverse associations have been observed in other real-world studies, suggesting that clinician behavior and risk stratification can modify the impact of renal dysfunction on lipid outcomes.[24] Our findings highlight the importance of considering treatment intensity and clinical context when interpreting associations between renal function and lipid control.

High-intensity statin therapy at discharge was independently associated with a significantly lower likelihood of LDL-C target non-achievement in

multivariable analysis, despite not reaching statistical significance in univariable analysis. This likely reflects confounding by indication, as patients prescribed high-intensity statins tended to have higher baseline LDL-C and greater overall cardiovascular risk, which masked the beneficial effect of intensive therapy in unadjusted analyses. After accounting for these factors, high-intensity statin therapy was independently associated with a lower likelihood of LDL-C target non-achievement. Large randomized trials and meta-analyses have consistently shown that high-intensity statin therapy initiated early after ACS reduces recurrent ischemic events and promotes plaque stabilization [25]. However, our study also revealed that the proportion of patients receiving high-intensity statins decreased over time, while the use of combination therapy with ezetimibe, although increased during follow-up, remained relatively low. This pattern mirrors findings from other registries in which treatment de-escalation and delayed intensification contribute to suboptimal LDL-C control.[7,12] In contrast, contemporary European cohorts with early combination therapy, including ezetimibe and PCSK9 inhibitors, have reported LDL-C 1.4 mmol/L attainment rates exceeding 50% within three months.[15] These observations suggest that reliance on statin monotherapy may be insufficient to achieve current guideline-recommended targets in a substantial proportion of high-risk patients.

Clinical implications

Our findings emphasize that LDL-C target non-achievement after NSTE-ACS and PCI is driven by a combination of high baseline lipid burden, an unfavorable metabolic profile, and suboptimal treatment intensification. While patient-related factors such as BMI and baseline LDL-C are not modifiable in the acute setting, treatment-related factors, including early initiation of high-intensity statins, timely addition of ezetimibe, and structured follow-up, represent actionable targets for improving lipid control. In resource-limited settings such as Vietnam, optimizing statin intensity and expanding the appropriate use of combination therapy may offer a pragmatic approach to narrowing the gap between guideline recommendations and real-world practice.

Study limitations. This study has several limitations. First, its observational design precludes causal inference. Second, follow-up duration was not uniform, reflecting real-world clinical practice, and may have influenced the timing of lipid measurements. Third, information on medication adherence and lifestyle modification was limited and primarily derived from medical records, which may have resulted in residual confounding. Finally, the relatively small sample size may have reduced statistical power and the precision of effect estimates, particularly in multivariable analyses.

Conclusions

In patients with NSTE-ACS undergoing PCI, LDL-C target attainment according to the 2019 ESC/EAS guidelines remains suboptimal in routine clinical practice. Less than one in five patients achieved LDL-C <1.4 mmol/L during follow-up, and baseline LDL-C, BMI, renal function, and the use of high-intensity statin therapy at discharge were independently associated with LDL-C target non-achievement. Although post-discharge modification of lipid-lowering therapy was common, particularly treatment de-escalation, our findings suggest that sustaining sufficiently intensive LLT after discharge, together with early LDL-C reassessment and timely treatment optimization, may help improve LDL-C target attainment in this high-risk population.

Author's Contribution:

Conceptualization, T.H.H., K.H.L.; methodology, T.H.H., H.D.D.; software, T.H.H., K.H.L.; validation, T.H.H., K.H.L.; formal analysis, T.H.H., K.H.L.; investigation, T.H.H., K.H.L., H.D.D.; resources, T.H.H., H.D.D.; data curation, T.H.H., K.H.L., H.D.D.; writing – original draft, T.H.H.; writing – review and editing, T.H.H., K.H.L., H.D.D., visualization, T.H.H., K.H.L., H.D.D.; supervision: K.H.L., H.D.D.; project administration T.H.H., K.H.L. All the authors have read and agreed with the final version of the article.

Compliance with Ethics Requirements:

"The authors declare no conflict of interest regarding this article"

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from all the patients included in the study"

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