Research Square

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LDL-C target attainment after PCI: the role of treatment intensity and real-world treatment gaps

Tuan Anh Ho
dr.hatuan@gmail.com

Tam Anh Hospital

Nga Thi Hang Pham
Thu Duc City Hospital

My Nu Tra Ton
Vinmec International Hospital

Chinh Van Nguyen
University of Medicine and Pharmacy

Thanh Tri Vu
Thu Duc City Hospital

Thien Hoang Tran
Thu Duc City Hospital

Anh Thai Nguyen
Thu Duc City Hospital

Minh Duc Tran
Tam Anh Hospital

Vinh Xuan Nguyen
Tam Anh Hospital

Duong Van Nguyen
Tam Anh Hospital

Tien Anh Hoang
Hue University of Medicine and Pharmacy

Nhan Thanh Vo
Tam Anh Hospital

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LDL-C target attainment after PCI: the role of treatment intensity and real-world treatment gaps

Highlights

- Only 12.4% of patients achieved the combined guideline-recommended LDL-C target after PCI in real-world practice.
- Baseline LDL-C and LDL-C reduction were independent determinants of target attainment.
- Patients with baseline LDL-C of 100–150 mg/dL demonstrated higher target attainment than those with lower or higher baseline LDL-C levels.
- Statin plus ezetimibe was associated with a 4.29-fold increase in the odds of achieving the combined LDL-C target compared with low-/moderate-intensity statin therapy, $p = 0.01$.
- Patients achieving the combined LDL-C target tended to have higher event-free survival than those who did not achieve the target.

Abstract

Background

Achieving guideline-recommended low-density lipoprotein cholesterol (LDL-C) targets is essential for secondary prevention after percutaneous coronary intervention (PCI). However, real-world attainment remains suboptimal, particularly in resource-limited settings.

Methods

We conducted a retrospective observational study of consecutive patients undergoing PCI at a district-level hospital in Vietnam between January 2023 and December 2024. The primary outcome was achievement of the combined LDL-C target (<55 mg/dL and ≥50% reduction from baseline) at 8–12 weeks. Multivariable logistic regression was used to identify independent determinants. Secondary outcomes included LDL-C reduction across treatment strategies and associations with major adverse cardiovascular events (MACE).

Results

Among 186 patients (median age 59 years; 61.3% male), 22.6% achieved LDL-C <55 mg/dL and 12.4% achieved the combined target. Median LDL-C decreased from 129 mg/dL to 71.4 mg/dL (−30.6%).

Baseline LDL-C (OR 0.72 per 10 mg/dL increase; 95% CI 0.65–0.80; $p < 0.001$) and LDL-C reduction (OR 1.37 per 10% increase; 95% CI 1.26–1.49; $p < 0.001$) were independent determinants of target attainment.
LDL-C reduction increased with treatment intensity (18.6%, 34.0%, and 44.0% for low/moderate, high-intensity, and combination therapy, respectively). Statin plus ezetimibe was associated with significantly higher odds of target attainment (OR 4.29; 95% CI 1.41–13.02). Patients achieving LDL-C targets showed a lower incidence of MACE, although statistical significance was not reached.

Conclusions

LDL-C target attainment after PCI remained markedly suboptimal. Baseline LDL-C levels and the magnitude of LDL-C reduction were independent determinants of treatment success. Combination therapy with statin plus ezetimibe was associated with substantially higher odds of achieving the combined LDL-C target. These findings support earlier and more intensive lipid-lowering strategies, particularly upfront combination therapy, in very-high-risk patients undergoing PCI in resource-limited settings.

Keywords: LDL-C; Percutaneous coronary intervention; Secondary prevention; Dyslipidemia; Statin; Ezetimibe; Therapeutic inertia

1. Introduction

Percutaneous coronary intervention (PCI) has significantly improved outcomes in patients with coronary artery disease; however, residual cardiovascular risk remains substantial. Elevated low-density lipoprotein cholesterol (LDL-C) continues to be a major modifiable contributor to recurrent events. Current guidelines recommend aggressive lipid lowering in very-high-risk patients, including those undergoing PCI, targeting LDL-C <55 mg/dL and ≥50% reduction from baseline [3,6].

Despite these recommendations, real-world studies consistently demonstrate suboptimal LDL-C target attainment, even in high-income settings. This gap is likely more pronounced in low- and middle-income countries, where access to high-intensity statins, combination therapy, and structured follow-up may be limited.

Vietnam is experiencing a growing burden of coronary artery disease, with increasing PCI volumes across all healthcare levels. However, data on lipid management after PCI, particularly in district-level hospitals, remain scarce.

This study aimed to determine the rate of LDL-C target attainment after PCI and to identify independent determinants of treatment success in a real-world district-level cohort. We also compared LDL-C reduction tailored to treatment strategies and its associations with major adverse cardiovascular events (MACE).

2. Methods

2.1. Study design and population

This retrospective observational study was conducted at Thu Duc City General Hospital, a district-level hospital in Ho Chi Minh City, Vietnam. Consecutive adults between January 2023 and December 2024 were included if they had undergone PCI for either acute or chronic coronary syndromes and had available lipid profile measurements both before PCI (baseline) and during follow-up. Patients were excluded if they had prior coronary artery bypass grafting, previous PCI, severe hepatic dysfunction (transaminases >3 times the upper limit of normal), advanced chronic kidney disease (estimated glomerular filtration rate <30 mL/min/1.73 m^2), documented intolerance to statins, or incomplete lipid data.

The study was conducted in accordance with the Declaration of Helsinki and all relevant guidelines and regulations. The study protocol was reviewed and approved by the Institutional Review Board/Ethics Committee of Thu Duc City General Hospital. The study was carried out under a scientific research project officially approved by the Ho Chi Minh City Department of Health (Decision No. 1084/QĐ-SYT). Due to the retrospective design of the study and the use of anonymized clinical data, the requirement for informed consent to participate was waived by the Institutional Review Board/Ethics Committee.

2.2. Data collection and definitions

Demographic characteristics, cardiovascular risk factors, clinical presentation, angiographic findings, and pharmacological treatments were extracted from electronic medical records. Baseline LDL-C was defined as the most recent measurement obtained prior to PCI. Follow-up lipid measurements were typically obtained during routine outpatient visits approximately 8–12 weeks after PCI, in accordance with standard clinical practice and guideline recommendations

[3,6]. Patients subsequently continued routine outpatient follow-up according to standard clinical practice.

2.3. Lipid-lowering therapy

Lipid-lowering therapy was prescribed at the discretion of the treating physician in accordance with local clinical practice. Statin intensity was categorized as low-, moderate- or high-intensity based on guideline definitions. Combination therapy with ezetimibe was recorded when applicable. Use of PCSK9 inhibitors was not observed during the study period due to limited availability and cost constraints.

2.4. Statistical analysis

Continuous variables were presented as median (interquartile range) due to non-normal distribution and compared using the Mann–Whitney U test. Categorical variables were presented as counts and percentages and compared using the Chi-square test or Fisher's exact test, as appropriate.

Multivariable logistic regression analysis was performed to identify independent determinants of achievement of the combined LDL-C target. Variables with $p < 0.10$ in univariable analyses and those considered clinically relevant were entered into the multivariable model. Collinearity was assessed prior to model construction. To improve clinical interpretability, continuous variables were rescaled to clinically meaningful increments (10 mg/dL and 10%) to facilitate interpretation of odds ratios. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs) and reflected the change in odds associated with these increments. A two-sided p-value < 0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 27.0. Kaplan–Meier analysis was performed to compare event-free survival stratified according to achievement of LDL-C target.

3. Results

3.1. Patient characteristics

A total of 186 patients who underwent PCI during the study period were included in the analysis (Figure 1). The median age was 59 years (interquartile range [IQR] 53–69), and 61.3% were male. Nearly half of the cohort had diabetes mellitus, and most procedures were performed for acute coronary syndromes.

Baseline clinical characteristics stratified by achievement of the combined LDL-C target are presented in Table 1. Patients achieving the combined target (n = 23, 12.4%) had significantly

lower baseline LDL-C levels than those who did not achieve the target (median 108 vs 134 mg/dL, $p < 0.001$). Other baseline characteristics were generally similar between groups.

3.2. Lipid-lowering therapy patterns

Lipid-lowering therapy patterns before and after PCI are illustrated in Figure 2. The proportion of patients receiving high-intensity statins increased from 31.2% before PCI to 50.5% after PCI, whereas use of low- to moderate-intensity statins decreased from 46.2% to 34.9%. Combination therapy with statin and ezetimibe remained relatively uncommon.

3.3. Changes in lipid parameters

Changes in lipid parameters are summarized in Table 2. Median LDL-C decreased from 129 mg/dL (IQR 110–152) at baseline to 71.4 mg/dL (IQR 58–92) at follow-up, corresponding to a median relative reduction of 30.6%. Non–HDL-C levels also decreased from 164 mg/dL (IQR 140–192) to 101 mg/dL (IQR 85–124).

3.4. LDL-C target attainment

Overall attainment of the guideline-recommended LDL-C target remained low (Figure 3). Although 22.6% of patients achieved LDL-C <55 mg/dL, only 12.4% achieved the combined target of LDL-C <55 mg/dL together with a ≥50% reduction from baseline. Meanwhile, 18.3% achieved a ≥50% LDL-C reduction alone, whereas 59.1% met neither criterion.

3.5. Treatment adequacy relative to baseline LDL-C

Individual-level analysis demonstrated that most patients failed to achieve the magnitude of LDL-C reduction required relative to their baseline LDL-C levels (Figure 4). The treatment gap became progressively larger with increasing baseline LDL-C, suggesting insufficient treatment intensity among patients at highest lipid-related risk.

Interestingly, treatment adequacy was not linear across baseline LDL-C strata. Patients with baseline LDL-C levels between approximately 100 and 150 mg/dL were more likely to approach the required LDL-C reduction, whereas those with higher baseline LDL-C exhibited substantially larger treatment gaps.

3.6. LDL-C reduction and target attainment according to treatment strategy

Figure 5 illustrates the theoretical LDL-C reduction required to achieve the guideline-recommended target of LDL-C <55 mg/dL according to baseline LDL-C levels. Patients with higher baseline LDL-C required progressively greater percentage reductions, frequently exceeding the LDL-C lowering typically achieved with statin monotherapy. Nevertheless, the

performance of real-world statin therapies was substantially lower. Median percentage LDL-C reduction was 18.6% (IQR: -10.3 to 43.7%) in the low- to moderate-intensity statin group, 34.0% (IQR: 8.8 to 53.8%) in the high-intensity statin group, and 44.0% (IQR: 23.7 to 57.6%) in the statin plus ezetimibe group (Figure 6).

Similarly, attainment of the combined LDL-C target increased stepwise across treatment strategies. The proportion of patients achieving the target was 7.3% in the low- to moderate-intensity statin group, 12.1% in the high-intensity statin group, and 23.8% in the statin plus ezetimibe group (Table 3).

In adjusted logistic regression analysis, statin plus ezetimibe therapy was associated with significantly higher odds of target attainment compared with low- to moderate-intensity statin therapy (OR 4.29; 95% CI 1.41–13.02; $p = 0.010$), whereas high-intensity statin therapy alone was not (Figure 7).

3.7. Determinants of LDL-C target attainment

Multivariable logistic regression identified baseline LDL-C and magnitude of LDL-C reduction as independent determinants of achieving the combined LDL-C target (Table 4).

Each 10 mg/dL increase in baseline LDL-C was associated with a 28% lower likelihood of target attainment (OR 0.72; 95% CI 0.65–0.80; $p < 0.001$). In contrast, each 10% increase in LDL-C reduction was associated with a 37% higher likelihood of success (OR 1.37; 95% CI 1.26–1.49; $p < 0.001$).

3.8. Clinical outcomes

During follow-up, patients achieving the combined LDL-C target showed a lower incidence of MACE compared with those who did not achieve the target (8.7% vs 19.6%). Kaplan–Meier analysis demonstrated a consistent direction of effect toward improved event-free survival, although the difference did not reach statistical significance (Figure 8).

No cases of rhabdomyolysis or severe statin intolerance requiring treatment discontinuation were observed during follow-up.

4. Discussion

In this real-world study conducted at a district-level hospital in Vietnam, we observed a markedly low rate of LDL-C target attainment after PCI. Although approximately one in five patients achieved LDL-C <55 mg/dL, only 12.4% achieved the combined guideline-recommended target of LDL-C <55 mg/dL together with a ≥50% reduction from baseline. These findings highlight a

substantial gap between contemporary guideline recommendations and clinical lipid management in resource-limited settings.

The low rate of LDL-C target attainment observed in our cohort is consistent with previous international studies. Farmakis et al. reported that only 12.7% of patients with acute coronary syndromes achieved the combined LDL-C target recommended by the 2019 ESC/EAS guidelines, while Park et al. reported a dual-target attainment rate of 17.2% among patients undergoing PCI in Korea [1,2]. In Vietnam, the DYSIS II registry previously demonstrated low attainment even for the older LDL-C target of <70 mg/dL [7]. Together, these findings suggest that achieving current guideline-directed LDL-C targets remains challenging even in contemporary clinical practice.

A major finding of the present study is that treatment success appeared to depend not only on baseline LDL-C levels but also on the magnitude of LDL-C reduction achieved during therapy. Each 10% increase in LDL-C reduction increased the likelihood of target attainment by 37%, emphasizing the importance of achieving sufficient lipid lowering rather than relying solely on baseline LDL-C levels.

Importantly, the individual-level treatment adequacy analysis provides mechanistic insight into the observed treatment gap. Most patients failed to achieve the magnitude of LDL-C reduction required relative to their baseline LDL-C levels, suggesting undertreatment rather than unavoidable treatment failure. The treatment gap became progressively larger with increasing baseline LDL-C, indicating a mismatch between treatment intensity and patient risk profile. Interestingly, treatment adequacy was not linear across baseline LDL-C levels. Patients with moderate baseline LDL-C values (approximately 100–150 mg/dL) were more likely to approach adequate LDL-C reduction, whereas those with higher baseline LDL-C exhibited substantially larger treatment gaps. These findings suggest that patients with higher baseline LDL-C may require earlier and more intensive lipid-lowering strategies to achieve guideline-recommended targets.

Our findings also demonstrated a clear relationship between treatment intensity and LDL-C reduction. LDL-C reduction increased progressively from low- to moderate-intensity statin therapy to high-intensity statins and combination therapy with ezetimibe. Notably, combination therapy was associated with higher odds of achieving the combined LDL-C target after

multivariable adjustment, whereas high-intensity statin therapy alone was not significantly associated with target attainment after adjustment.

These findings suggest that the observed treatment gap may be driven less by baseline risk alone and more by insufficient treatment intensification. Despite guideline recommendations supporting early combination therapy in very-high-risk patients, ezetimibe use remained relatively uncommon in our cohort. Underutilization of combination therapy may therefore represent an important contributor to suboptimal lipid control in real-world practice.

Structural and economic barriers likely contribute to these findings in resource-limited environments. Although PCSK9 inhibitors provide substantial additional LDL-C reduction, their use remains restricted in many developing countries because of limited availability and high cost. Consequently, optimization of more accessible strategies—including earlier initiation of high-intensity statins and timely addition of ezetimibe—may represent a pragmatic approach to improving secondary prevention.

Although the Kaplan–Meier analysis did not demonstrate a statistically significant reduction in MACE among patients achieving the combined LDL-C target, the observed directional separation of survival curves was biologically and clinically consistent with the greater LDL-C reduction achieved in this group (Figure 8). The low number of clinical events in the target-attainment group (2 events among 23 patients) likely limited the statistical power to detect significant differences in outcomes. Nonetheless, the overall pattern of survival curves remained consistent with the treatment adequacy framework observed throughout the study, suggesting that more intensive lipid-lowering strategies and successful LDL-C target attainment may potentially translate into improved long-term cardiovascular outcomes. These findings are directionally consistent with evidence from large randomized trials demonstrating a log-linear relationship between LDL-C reduction and cardiovascular risk reduction [8].

Clinically, our findings support the concept that failure to achieve LDL-C targets after PCI may reflect undertreatment relative to baseline LDL-C rather than unavoidable therapeutic failure. Therefore, those with higher baseline LDL-C levels may benefit from earlier and more aggressive treatment intensification, including upfront combination therapy.

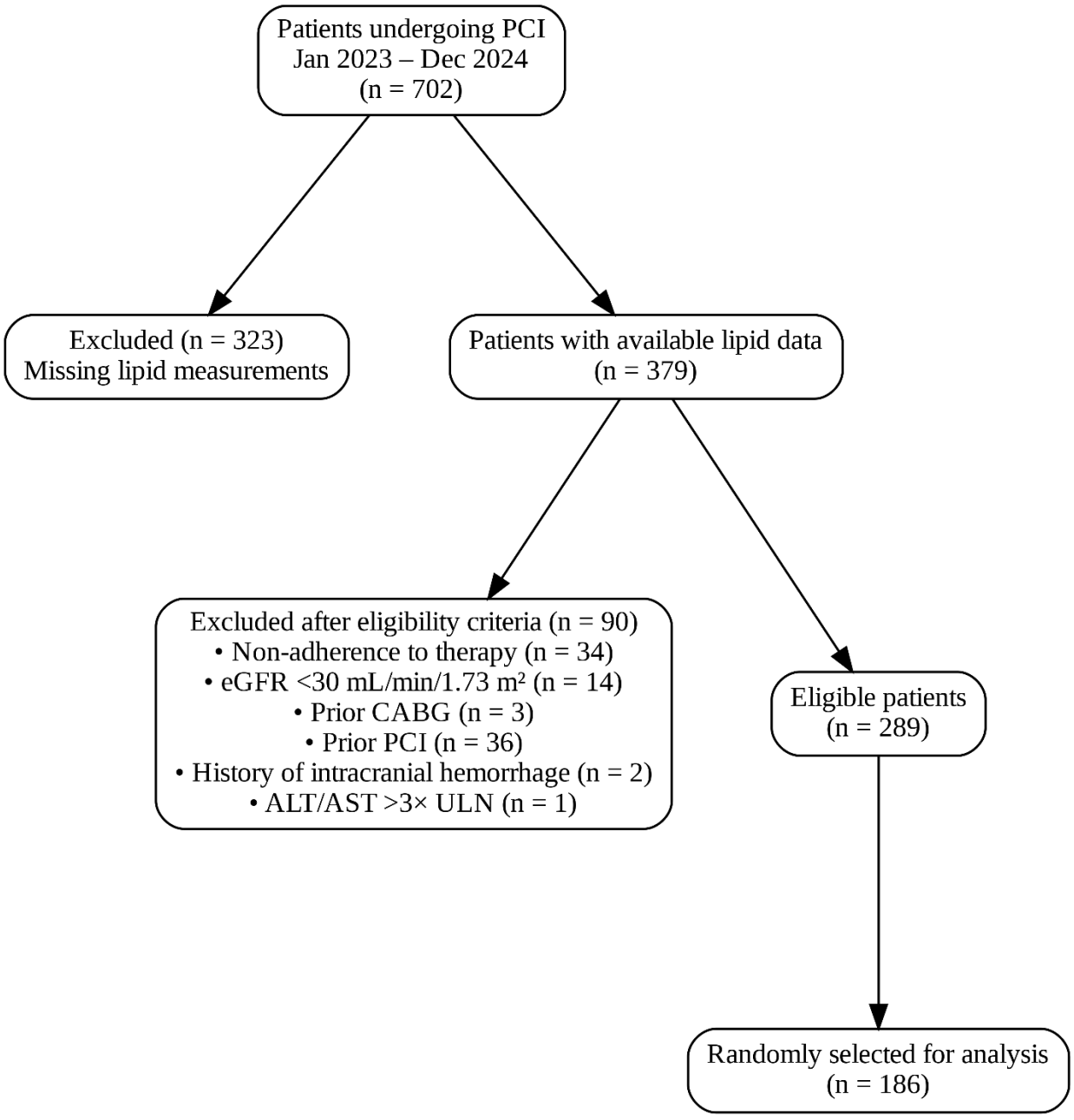
Several limitations should be acknowledged. First, this was a retrospective single-center study, which may limit generalizability. Second, the modest sample size reduced statistical power, particularly for clinical outcome analyses. Third, treatment strategies were not standardized and

reflected routine clinical practice, introducing potential treatment heterogeneity. Nevertheless, this pragmatic design also represents a major strength by providing insight into real-world lipid management in a district-level healthcare setting.

5. Conclusion

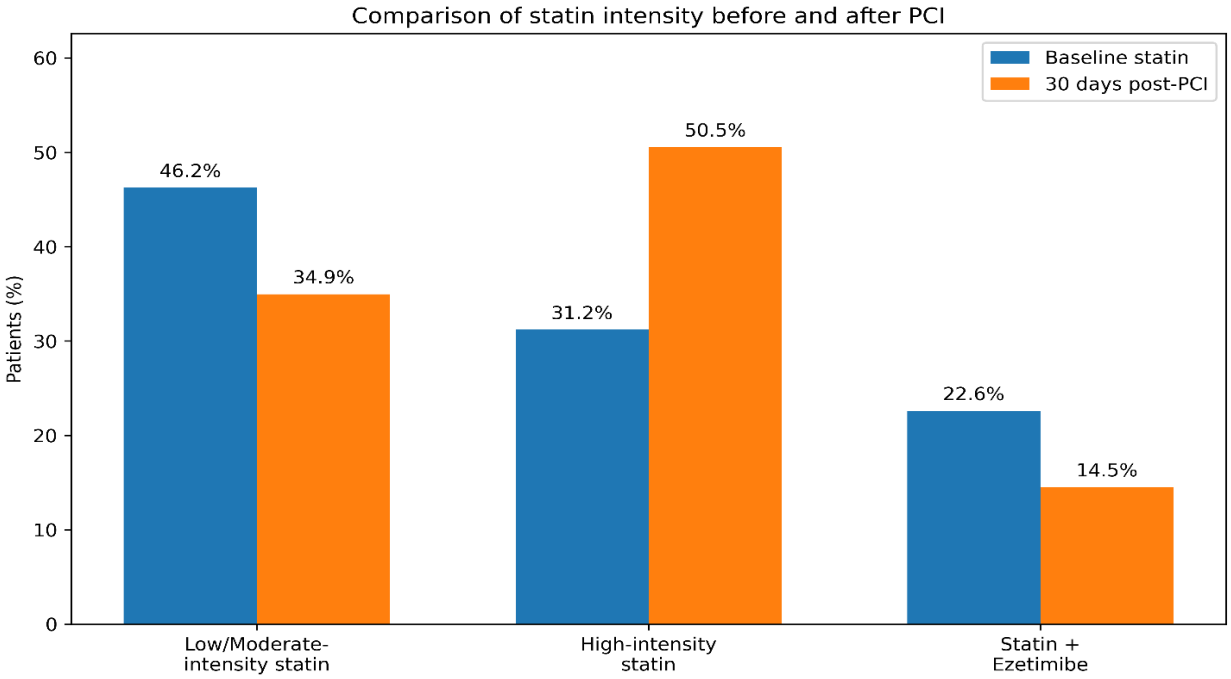
LDL-C target attainment after PCI remained markedly suboptimal in this real-world cohort. Baseline LDL-C levels and the magnitude of LDL-C reduction were independent determinants of treatment success, suggesting that failure to achieve guideline-recommended targets may largely reflect insufficient treatment intensification relative to baseline LDL-C. Combination therapy with statin plus ezetimibe was associated with substantially higher odds of achieving the combined LDL-C target. These findings support earlier and more intensive lipid-lowering strategies, particularly upfront combination therapy, in very-high-risk patients undergoing PCI in resource-limited settings.

Figure 1. Flowchart of patient selection



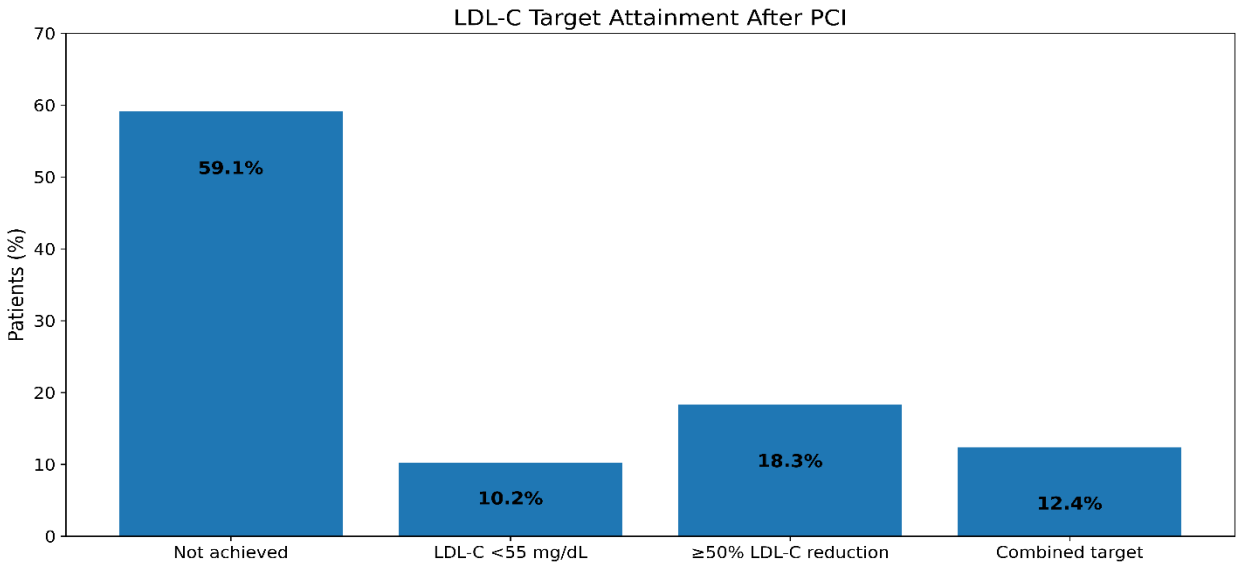
Flow diagram illustrating the inclusion and exclusion of patients in the study cohort.

Figure 2. Lipid-lowering therapy patterns before and after PCI



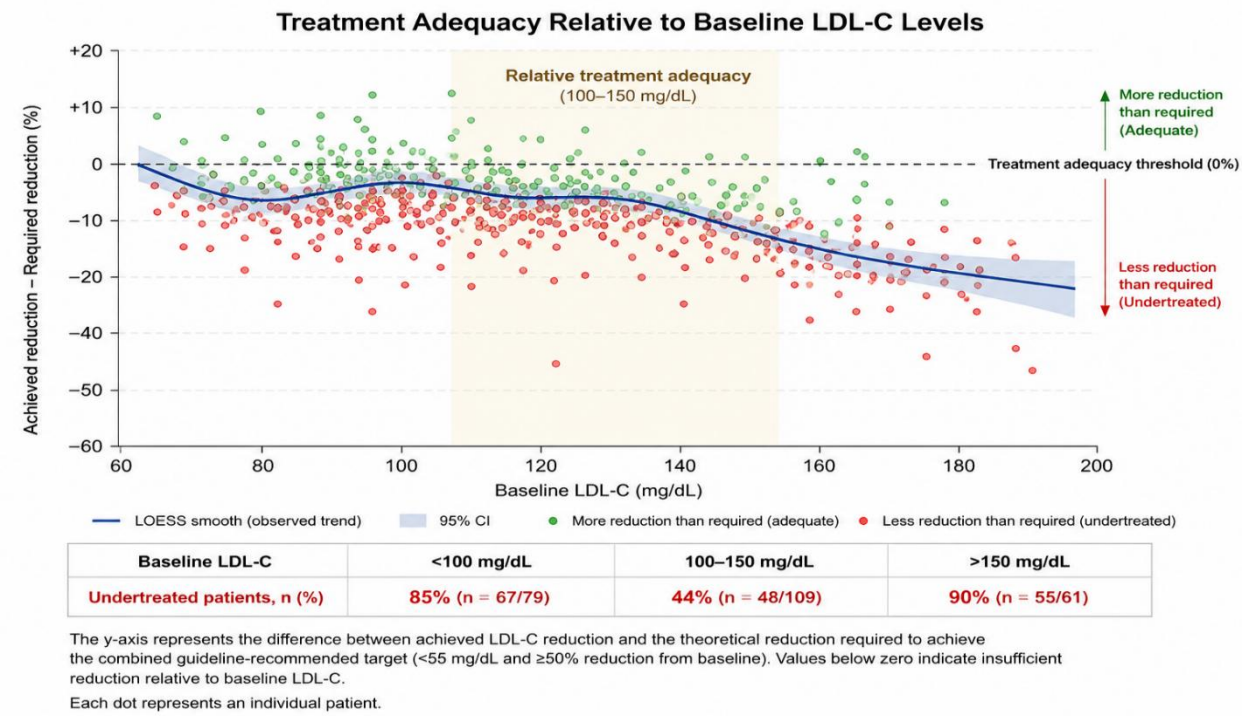
Distribution of lipid-lowering therapies prescribed within 60 days before baseline lipid assessment and within 30 days after PCI, demonstrating increased use of high-intensity statins but limited adoption of combination therapy.

Figure 3. LDL-C target attainment after PCI



Proportion of patients achieving individual LDL-C targets (<55 mg/dL or ≥50% reduction) and the combined guideline-recommended target (<55 mg/dL and ≥50% reduction from baseline), highlighting the low rate of attainment of the guideline-recommended combined target.

Figure 4. Treatment adequacy relative to baseline LDL-C (individual-level analysis)



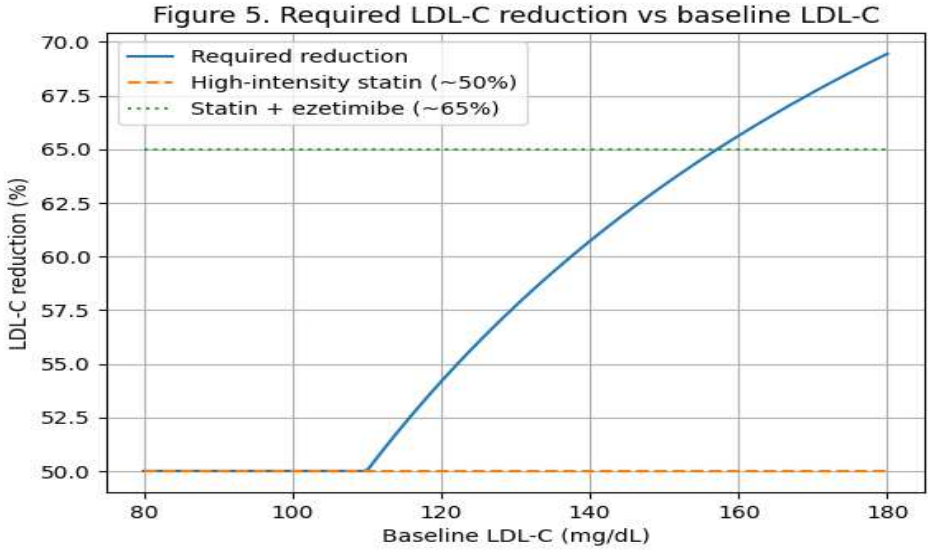
Baseline LDL-C	<100 mg/dL	100–150 mg/dL	>150 mg/dL
Undertreated patients, n (%)	85% (n = 67/79)	44% (n = 48/109)	90% (n = 55/61)

The y-axis represents the difference between achieved LDL-C reduction and the theoretical reduction required to achieve the combined guideline-recommended target (<55 mg/dL and ≥50% reduction from baseline). Values below zero indicate insufficient reduction relative to baseline LDL-C.

Each dot represents an individual patient.

Most patients exhibited a negative treatment gap, particularly at higher baseline LDL-C levels, suggesting that current treatment strategies were frequently insufficient to achieve guideline-recommended targets.

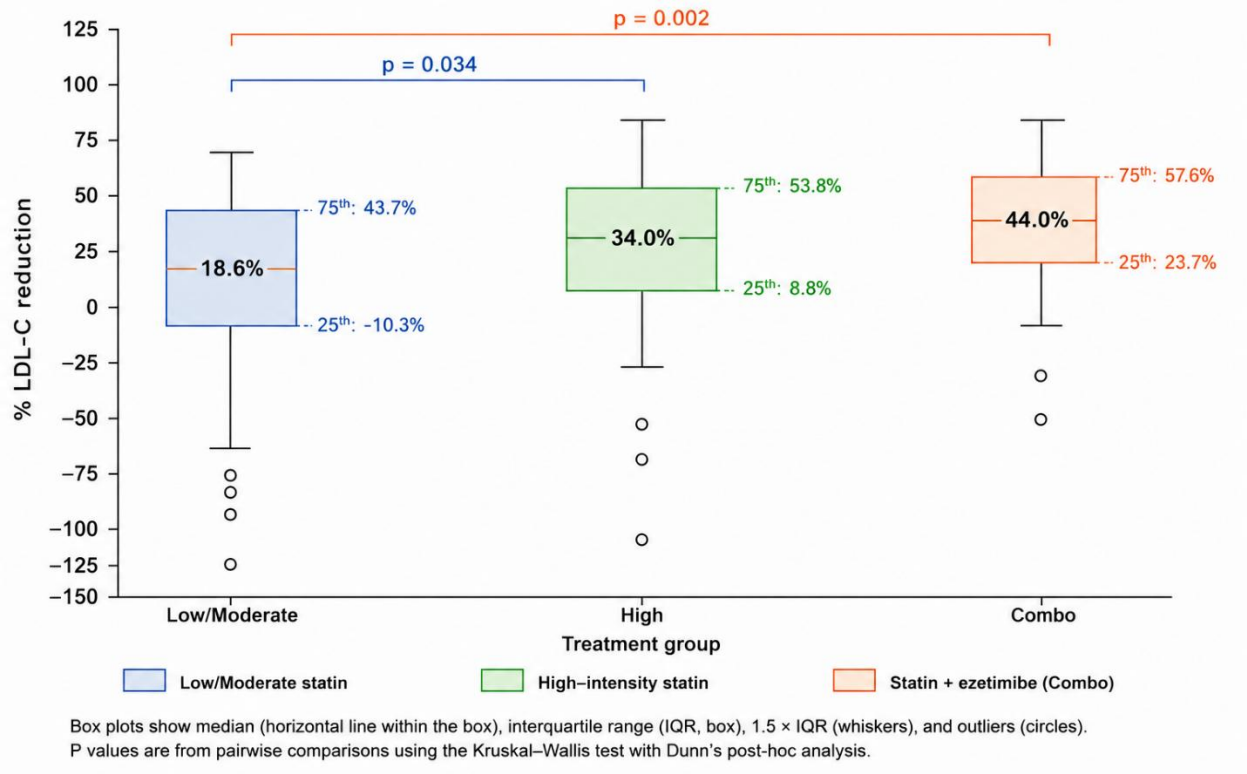
Figure 5. Required LDL-C reduction according to baseline LDL-C levels



The figure illustrates the theoretical percentage reduction in LDL-C required to achieve the

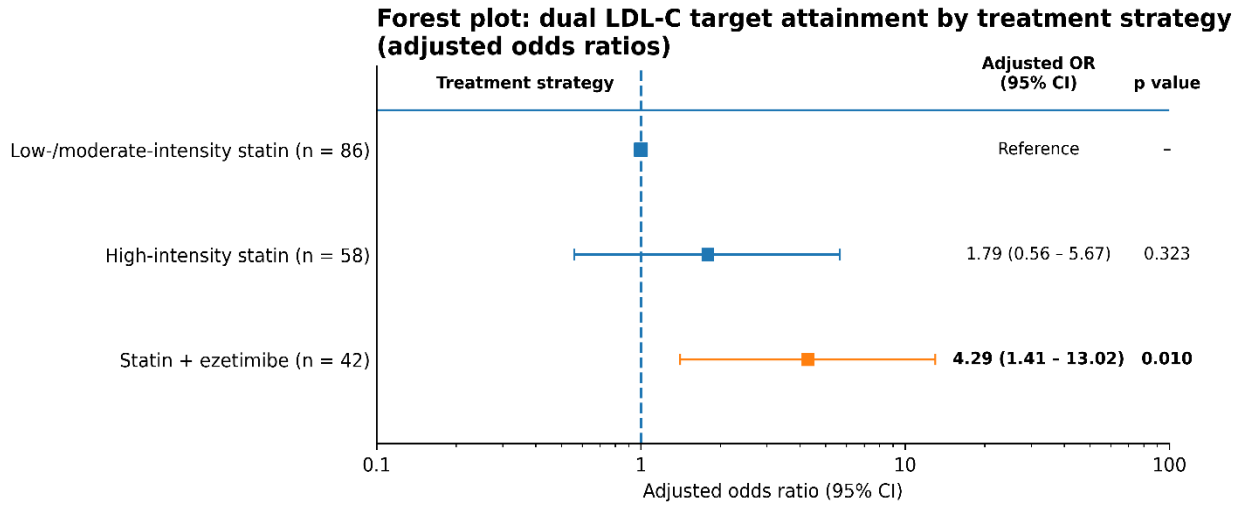
guideline-recommended target of LDL-C <55 mg/dL and ≥50% reduction from baseline. Patients with higher baseline LDL-C levels required substantially greater reductions, frequently exceeding the LDL-C lowering typically achieved with statin monotherapy. Expected LDL-C reductions associated with different lipid-lowering strategies were estimated according to contemporary guideline definitions and prior clinical trial data [3,6].

Figure 6. LDL-C reduction according to treatment strategy



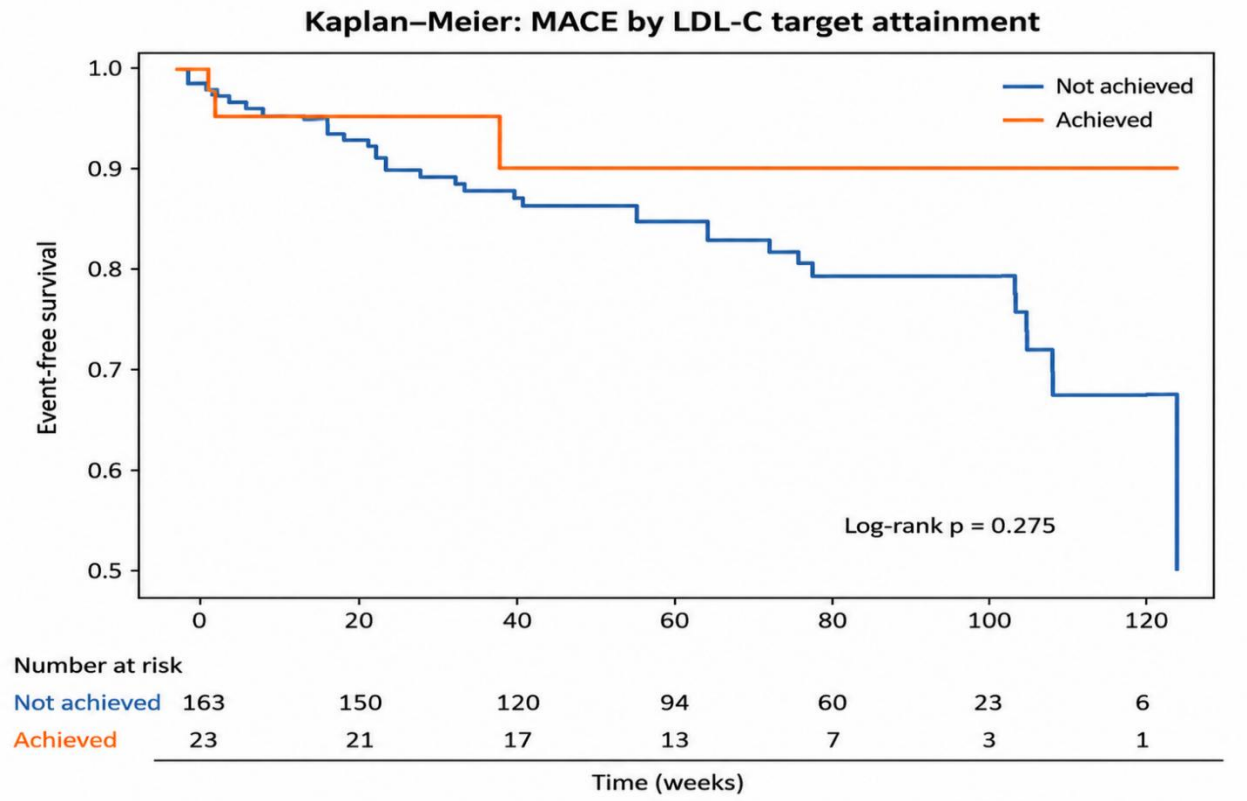
Median LDL-C reduction increased progressively with treatment intensity, with the greatest reduction observed in patients receiving statin plus ezetimibe combination therapy.

Figure 7. Forest plot: dual LDL-C target attainment by treatment strategy



Statin plus ezetimibe combination therapy was associated with significantly greater odds of achieving the combined LDL-C target compared with low-/moderate-intensity statin therapy (OR 4.29, 95% CI 1.41–13.02; p = 0.01), whereas high-intensity statin therapy alone was not significantly associated with target attainment.

Figure 8. Kaplan–Meier analysis of MACE-free survival according to achievement of the combined LDL-C target



Patients achieving the combined LDL-C target showed numerically higher event-free survival compared with those who did not achieve the target, although the difference did not reach statistical significance (log-rank p = 0.275).

Table 1. Baseline characteristics of the study population stratified by LDL-C target attainment

Variable	Total (n=186)	Achieved target (n=23)	Did not achieve target (n=163)	p-value
Age, years (median, IQR)	59 (53–69)	61 (55–70)	58 (52–69)	0.28
Male sex, n (%)	114 (61.3)	15 (65.2)	99 (60.7)	0.68
Diabetes mellitus, n (%)	92 (49.5)	11 (47.8)	81 (49.7)	0.86
Acute coronary syndrome, n (%)	130 (69.9)	16 (69.6)	114 (69.9)	0.98
Baseline LDL-C, mg/dL (median, IQR)	129 (110–152)	108 (96–120)	134 (118–158)	< 0.001
Gensini score (median, IQR)	21.8 (10–40)	18 (9–32)	23 (11–42)	0.19
High-intensity statin, n (%)	54 (29.0)	10 (43.5)	44 (27.0)	0.08

Table 2. Changes in lipid parameters before and after PCI

Parameter	Baseline	Follow-up	Relative change
LDL-C, mg/dL (median, IQR)	129 (110–152)	71.4 (58–92)	−30.6%
Non-HDL-C, mg/dL (median, IQR)	164 (140–192)	101 (85–124)	−29.9%

Table 3. Performance of treatment strategies

Comparison	Achieved (%)	Absolute difference (95% CI)	Adjusted OR (95% CI)	NNT (95% CI)
Low-/moderate-intensity statin	7.3	Reference	Reference	Reference
High-intensity statin vs Low/Moderate	12.1	+4.8% (−5.1 to 16.3)	1.79 (0.56–5.67)	21 (unstable estimate*)
Statin + ezetimibe vs Low/Moderate	23.8	+16.5% (3.6 to 31.7)	4.29 (1.41–13.02)	6 (3–28)

*Estimate unstable because the confidence interval crossed zero.

Table 4. Multivariable logistic regression analysis for determinants of combined LDL-C target attainment

Variable	Adjusted OR	95% CI	p-value	Clinical interpretation
Baseline LDL-C (per 10 mg/dL increase)	0.72	0.65–0.80	<0.001	~28% lower odds per 10 mg/dL increase
LDL-C reduction (per 10% increase)	1.37	1.26–1.49	<0.001	~37% higher odds per 10% increase

OR indicates odds ratio; CI, confidence interval.
Odds ratios are presented per 10-unit increments to facilitate clinical interpretation.
Estimates were derived from a multivariable logistic regression model.

Declarations

Abbreviations

ACS: Acute coronary syndrome

CI: Confidence interval

ESC/EAS: European Society of Cardiology/European Atherosclerosis Society

HDL-C: High-density lipoprotein cholesterol

IQR: Interquartile range

IRB: Institutional Review Board

LDL-C: Low-density lipoprotein cholesterol

MACE: Major adverse cardiovascular events

NNT: Number needed to treat

OR: Odds ratio

PCI: Percutaneous coronary intervention

PCSK9: Proprotein convertase subtilisin/kexin type 9

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki and all relevant guidelines and regulations. The study protocol was reviewed and approved by the Institutional Review Board/Ethics Committee of Thu Duc City General Hospital. The study was carried out under a scientific research project officially approved by the Ho Chi Minh City Department of Health (Decision No. 1084/QĐ-SYT). Due to the retrospective design of the study and the use of anonymized clinical data, the requirement for informed consent to participate was waived by the Institutional Review Board/Ethics Committee.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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The authors received no specific funding for this work.

Authors' contributions

Tuan Anh Ho conceived and designed the study, performed the statistical analyses, and drafted the manuscript.

Nga Thi Hang Pham collected the data, participated in the statistical analyses, and contributed to drafting the manuscript.

My Nu Tra Ton participated in the statistical analyses and contributed to drafting the manuscript.

Chinh Van Nguyen reviewed the statistical analyses and contributed to drafting the manuscript.

Thanh Tri Vu revised the Methods section, reviewed the statistical analyses, and contributed to drafting the manuscript.

Thien Hoang Tran collected the data.

Anh Thai Nguyen collected the data.

Minh Duc Tran participated in the statistical analyses and prepared the figures.

Vinh Xuan Nguyen contributed to writing and revising the Results section.

Duong Van Nguyen contributed to writing and revising the Results and Discussion sections.

Tien Anh Hoang revised the Methods section, reviewed the statistical analyses, and contributed to drafting the manuscript.

Nhan Thanh Vo revised the Methods section and contributed to drafting the manuscript.

All authors read and approved the final manuscript.

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