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Original Article

A PDCA cycle-based early intensive lipid-lowering strategy improves LDL-C goal achievement and clinical outcomes after PCI in acute coronary syndrome patients

Kun Zhang*, Qiuxian Liu*, Yanping Wang*, Xinjun Zhang, Hongyuan Wang

*Department of Cardiology, Jiangshan People's Hospital, Jiangshan 324100, Zhejiang, China. *Equal contributors and co-first authors.*

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Abstract: Aims: To evaluate whether a PDCA cycle-based early intensive lipid-lowering strategy could improve LDL-C goal attainment in patients with acute coronary syndrome undergoing PCI. Methods: This retrospective, single-center cohort study enrolled 203 consecutive ACS patients undergoing PCI between January 2022 and January 2025. Patients were assigned to a control group receiving standard lipid-lowering therapy (n=101) or a PDCA intervention group (n=102) managed through a Plan-Do-Check-Act cycle-based early intensive lipid-lowering protocol. The primary endpoint was LDL-C goal attainment at 12 weeks (<1.8 mmol/L or ≥50% reduction). Secondary endpoints included lipid dynamics, medication adherence, therapy intensification, safety outcomes, and lifestyle adherence. Laboratory, echocardiographic, and behavioral data were prospectively collected and analyzed by blinded investigators using standardized protocols. Results: The PDCA cycle-based strategy achieved greater and earlier reductions in LDL-C and total cholesterol, leading to higher LDL-C goal attainment (<1.8 mmol/L: 57.8% vs. 31.7%; ≥50% reduction: 61.7% vs. 28.7%; both P<0.001). High-intensity statin, ezetimibe, and PCSK9 inhibitor use were significantly higher, accompanied by improved medication adherence (94.38 ± 10.64% vs. 83.63 ± 11.56%, P<0.001) and lifestyle compliance. PDCA management enhanced LVEF improvement (from 53.84 ± 6.71 to 62.00 ± 6.09% vs. 52.91 ± 5.59 to 53.65 ± 6.09%; P<0.001) without increasing adverse events. Multivariate analysis confirmed PDCA intervention as an independent predictor of LDL-C goal achievement (OR 9.353, 95% CI 4.424-19.775; P<0.001). Conclusion: A PDCA cycle-based early intensive lipid-lowering strategy significantly improved LDL-C goal achievement, medication and lifestyle adherence, and cardiac function after PCI in ACS patients, without increasing adverse events.

Keywords: Acute coronary syndrome, percutaneous coronary intervention, PDCA cycle, intensive lipid-lowering therapy, LDL-C target achievement

Introduction

Low-density lipoprotein cholesterol (LDL-C) is a key, changeable goal of cardiovascular disease development [1], and atherosclerotic cardiovascular disease (ASCVD) has remained the major cause of morbidity and mortality globally [2]. Acute coronary syndrome (ACS) represents a particularly vulnerable phase of ASCVD, characterized by a markedly increased risk of recurrent ischemic events, heart failure, and cardiovascular death. In this context, secondary prevention strategies initiated immediately after the index event are critical determinants of both short- and long-term prognosis. Among these strategies, aggressive lipid management has emerged as a cornerstone of contemporary cardiovascular care.

Rapid management and maintenance of LDL-C reduction is important in patients with acute coronary syndrome (ACS) to prevent repeating ischemic experiences and long-term outcomes [3]. Numerous landmark trials - including IMPROVE-IT, FOURIER, and ODYSSEY OUTCOMES - have established a clear "lower is better" paradigm, with contemporary guidelines advocating an LDL-C target of <1.4 mmol/L (55 mg/dL)

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and at least a 50% reduction from baseline for very-high-risk patients following percutaneous coronary intervention (PCI) [4-6]. These recommendations underscore not only the magnitude of LDL-C lowering required, but also the importance of achieving targets as early as possible after PCI. Nevertheless, in spite of the evidence strength and the presence of strong lipid-lowering agents, in practice experiential evidence always indicates that a significant percentage of post-PCI ACS patients do not reach the recommended LDL-C levels.

Multifactorial issues are manifested in this ongoing evidence-practice gap. Resting placebo effect, prolonged rate of lipid-lowering therapy (LLT) amplification, and discontinuous manner of follow-up procedures usually detract timely maximization of LDL-C levels [7]. In addition, the current care pathways would focus on acute revascularization and dual antiplatelet therapy with lipid control being delayed to outpatient rehabilitation, where there is a risk of patient non-adherence and declining continuation of care. As a result, there is a gap in terms of type and use of early post-PCI lipid management, yet this is a serious secondary prevention period. Clinical inertia during the early post-discharge phase therefore represents a missed opportunity for effective risk modification.

Implementations of continuous quality improvement (CQI) systems, which involve the Plan-do-check-act (PDCA) cycle, have proven to be effective in improving clinical dedication to evidence-based goals in diverse fields [8-11]. PDCA introduces the process lacking features of clinical work by integrating iterative monitoring, performance enhancement based on data, and team-based performance responsibility [12]. Within cardiovascular medicine, PDCA-based models offer a structured and reproducible approach to overcoming therapeutic inertia, particularly during critical transitions from inpatient to outpatient care. PDCA can be used in the healthcare context to apply lipid management with a framework applied to the prevention of clinical inertia in the management process and timely medication intensification by making inpatient-outpatient transitions.

Based on this justification, the current study compared the effectiveness of an early intensive lipid-lowering intervention, which is dependent on a PDCA cycle, to the attainment of the LDL-C goal in ACS, PCI patients. Our hypothesis was that by introducing PDCA-based process management into regular care; LDL-C targets are ensured to a significantly greater extent at short-term follow-up, without a negative effect on safety. Notably, the innovative aspect of this study lies in integrating a PDCA-driven quality improvement strategy directly into early post-PCI lipid management, rather than relying solely on pharmacological escalation or delayed outpatient adjustment. This will combine pharmacologic intensification with persistent optimization of the processes, which will shift the LDL-C management paradigm towards proactive, rather than reactive prevention of cardiovascular events, and better post-PCI cardiovascular outcomes.

Methods

Patients' selection

The study was a retrospective, single-center cohort study. We consecutively screened all patients hospitalized with acute coronary syndrome (ACS) who underwent percutaneous coronary intervention (PCI) at our institution between January 2022 and January 2025. Inclusion criteria were: (1) age 18-80 years; (2) a confirmed diagnosis of ACS (ST-segment elevation myocardial infarction, non-ST-segment elevation myocardial infarction, or unstable angina) based on contemporary diagnostic criteria, including clinical presentation, electrocardiography, and cardiac biomarkers; (3) PCI performed during the index hospitalization at our institution (including balloon angioplasty and/or stent implantation) with complete procedural documentation; (4) availability of a baseline lipid profile including LDL-C obtained at admission or before/within 24 h of PCI and prior to substantial post-admission lipid-lowering regimen escalation; (5) discharged alive and prescribed lipid-lowering therapy after PCI; (6) complete and retrievable demographic, clinical, and laboratory data in the electronic health record (including key comorbidities, medication records, and baseline biochemical tests); and (7) at least one post-discharge lipid assessment within 12 weeks (e.g., at 4-6 weeks and/or up to 12 weeks), with follow-up information sufficient to evaluate LDL-C goal attainment and treatment adjustments. Exclusion criteria

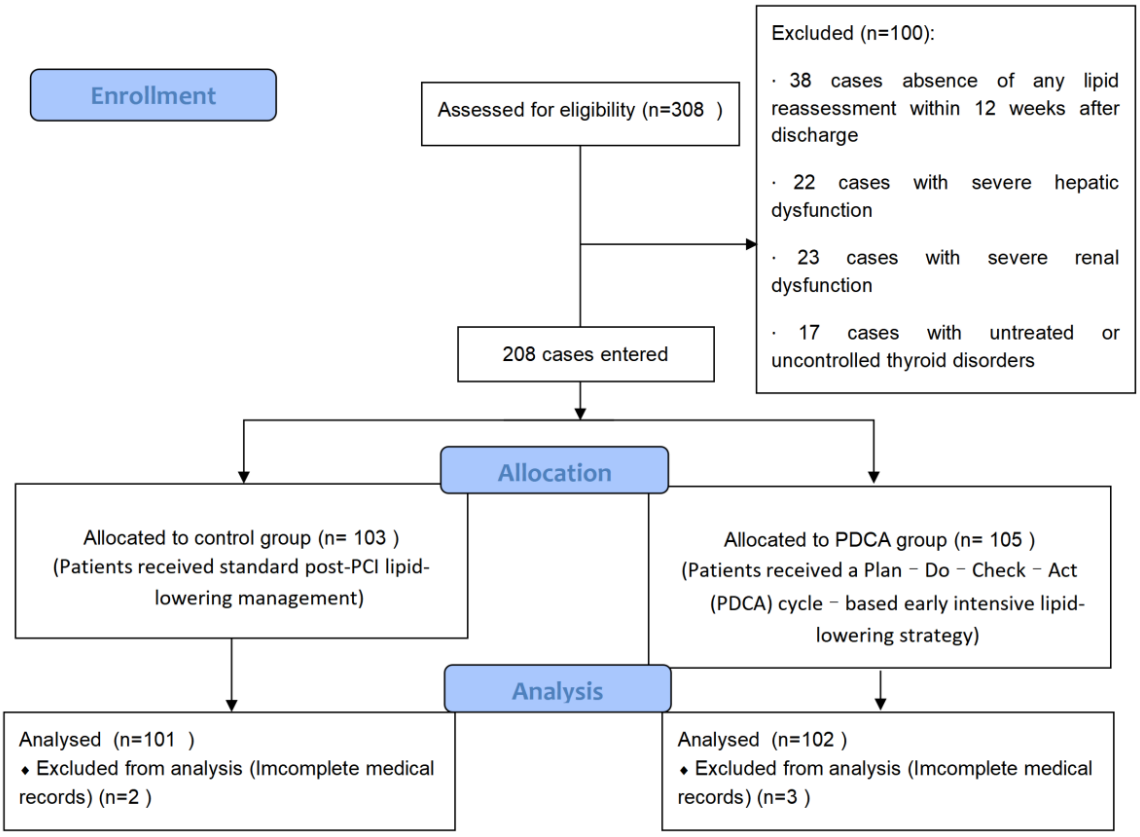

Figure 1. Flow diagram detailing the selection of patients included in this study.

were: (1) missing baseline LDL-C or absence of any lipid reassessment within 12 weeks after discharge; (2) incomplete key clinical/procedural data or inability to verify medication exposure/adherence from records; (3) loss to follow-up, transfer to another institution, or death before the first post-discharge lipid reassessment within 12 weeks; (4) severe hepatic dysfunction (known cirrhosis or baseline ALT/AST >3× the upper limit of normal, or other clinically significant liver disease that could affect lipid metabolism or limit statin use); (5) severe renal dysfunction (end-stage renal disease, dialysis, or markedly reduced renal function such as eGFR <30 mL/min/1.73 m^2); (6) untreated or uncontrolled thyroid disorders (hyperthyroidism or hypothyroidism, or clearly abnormal thyroid function requiring treatment) that could substantially influence lipid levels; (7) active malignancy or ongoing chemotherapy/radiotherapy; and (8) other conditions considered by investigators to markedly affect lipid metabolism or preclude standardized follow-up (e.g., systemic inflammatory disease, chronic steroid/immunosuppressive therapy, pregnancy/lactation). The selection of patients included in this study was shown in **Figure 1**. Based on the in-hospital lipid management pathway implemented during the study period, eligible patients were categorized into two groups: a control group (n=101) receiving standard post-PCI lipid-lowering management and a PDCA intervention group (n=102) managed using a Plan-Do-Check-Act (PDCA) cycle-based early intensive lipid-lowering strategy. The PDCA framework was implemented by a multidisciplinary cardiovascular team and emphasized continuous process optimization: (1) Plan - comprehensive baseline assessment of LDL-C and individualized target setting (<1.4 mmol/L and ≥50% reduction from baseline); (2) Do - initiation of high-intensity statin therapy (atorvastatin 40-80 mg or rosuvastatin 20-40 mg daily) immediately after PCI, with early combination therapy (ezetimibe or PCSK9 inhibitors) for patients with markedly elevated LDL-C; (3) Check - structured lipid reassessment at 4-6 weeks post-discharge, with adherence monitoring through

electronic medical records and telephonic follow-up; and (4) Act - therapy intensification and individualized counseling for patients not achieving LDL-C goals, integrating feedback to refine the subsequent PDCA cycle. Demographic, clinical, and biochemical data were extracted from the institutional electronic health record system, and LDL-C goal attainment was defined as achieving both <1.4 mmol/L and a ≥50% reduction from baseline. The study protocol was approved by the ethics committee of Jiangshan People's Hospital and conducted in accordance with the Declaration of Helsinki.

Data extraction

Control group: Standard post-PCI lipid-lowering management that was protocolized in accordance with the current guidelines was given to the control group: a high-intensity statin (atorvastatin 40-80 mg or rosuvastatin 20-40 mg) started 24 hours after PCI at the discretion of the treating physician; further titration was non-protocolized and initiated by regular outpatient follow-up visits (usually 4-12 weeks) and by safety labs. The use of ezetimibe or PCSK9 inhibitors, lifestyle education, and adherence counseling over site usual care that did not have a required frequency, and statin intolerance were all handled according to local practice (temporary dose reduction or moderate-intensity statin). Conversely, the PDCA group was under the supervision of a multidisciplinary quality-improvement team (interventional cardiology, clinical pharmacy, nursing, dietetics, and data management) operating on a predefined Plan-Do-Check-Act to provide early intensive lipid lowering. Plan: within 24 h post-PCI, risk stratification and baseline labs were reviewed; all patients were started on a high-intensity statin plus standardized education (10-minute script on goals, adverse effects, and lifestyle; printed checklist), with LDL-C goals set a priori (primary <1.8 mmol/L and ≥50% reduction; very-high-risk secondary goal <1.4 mmol/L, and exploratory <1.0 mmol/L). Do: treatment was escalated proactively on a fixed timetable - telephone/clinic assessments at weeks 2, 4, 8, and 12 with lab panels; if LDL-C ≥1.8 mmol/L or <50% reduction at week 2, ezetimibe 10 mg was added; if goals remained unmet at week 4, a PCSK9 inhibitor was initiated (alirocumab 75-150 mg Q2W or evolocumab 140 mg Q2W), with dose up titration per label at week 8 if still above target. Check: a dashboard (case report form-embedded) tracked LDL-C trajectories, medication possession ratio (MPR), adverse events, and lifestyle metrics (Mediterranean diet score, physical activity ≥150 min/week, home BP monitoring, sleep duration); signal thresholds (e.g., MPR <80%, missed lab, or LDL-C plateau) autogenerated alerts for pharmacist-nurse outreach within 72 h. Intervention: the team met weekly (at most) in brief huddles to institute corrective measures such as adherence problem-solving, mitigation of side effects (e.g., switch atorvastatin↔rosuvastatin, alternate-day dosing, add coenzyme Q10 in case of suspected myalgia), or additional intensification of lipid therapy until they were maintained at two consecutive visits. Safety rules were pre-specified: hold statin if ALT/AST >3×ULN with symptoms or CK >5×ULN, re-challenge after normalization with alternate agent/dose; PCSK9 was preferred in documented statin intolerance. Lifestyle intervention frequency was mandated (dietitian and rehab touchpoints at baseline and weeks 4 & 12; structured smoking-cessation counseling for current smokers). Investigators (blinded to allocation) adjudicated all the clinical endpoints, and all adverse events and had no option to blind treat teams as the nature of the intervention did not permit.

Comprehensive clinical, laboratory, and imaging data were prospectively collected from admission to 12 months post-PCI using standardized electronic case report forms. Baseline demographics, cardiovascular risk factors, ACS presentation, procedural features, and medication use were recorded at enrollment. Fasting venous blood samples were obtained at baseline, 4 weeks, and 12 weeks to assess lipid profiles, liver and renal function, glucose metabolism, and inflammatory biomarkers in a central laboratory. Echocardiographic parameters were measured at baseline, 4 weeks, and 12 weeks by blinded sonographers following ASE/EACVI guidelines. Medication adherence, adverse events, and lifestyle behaviors - including diet, physical activity, smoking, weight control, and rehabilitation participation - were assessed through structured interviews and validated questionnaires at each follow-up time point: baseline, 4 weeks, and 12 weeks. Data integri-

ty and completeness were verified by independent clinical monitors.

Outcome measures

The primary outcome was the proportion of patients achieving the LDL-C therapeutic goal at 12 weeks, defined as LDL-C <1.8 mmol/L or ≥50% reduction from baseline, with additional analyses for intensive thresholds (<1.4 and <1.0 mmol/L). Secondary outcomes included temporal changes in lipid profiles (LDL-C, total cholesterol, triglycerides, HDL-C), medication adherence rates, and the proportion of patients undergoing lipid-lowering therapy intensification (ezetimibe or PCSK9 inhibitor initiation). Safety outcomes encompassed liver enzyme elevation (>3×ULN), creatine kinase elevation, myalgia, and drug discontinuation due to adverse effects, assessed up to 12 months. Exploratory outcomes evaluated changes in blood biochemistry (ALT, AST, serum creatinine, eGFR, fasting glucose, HbA1c, hs-CRP, TNF-α), cardiac ultrasound indices (LVEF, LVEDD, LVESD, E/e', LAVI), and adherence to lifestyle modification metrics, including diet, physical activity, smoking cessation, weight reduction, home blood pressure monitoring, and sleep duration. All outcomes were predefined and assessed by investigators blinded to group assignment using standardized protocols.

Statistical analysis

All analyses were performed using SPSS 26.0 and R 4.3.1. Continuous variables were assessed for normality and presented as mean ± SD or median (IQR), and compared using the independent-samples t test or Mann-Whitney U test, as appropriate. Categorical variables were summarized as counts (percentages) and compared using the χ^2 test or Fisher's exact test. Temporal changes in lipid and echocardiographic indices were evaluated using repeated-measures ANOVA with Bonferroni-adjusted post hoc comparisons. To identify independent predictors of LDL-C goal attainment at 12 weeks (yes/no), univariate logistic regression was first performed for each candidate variable. Variables with P<0.10 in univariate analyses and/or with established clinical relevance were considered candidates for the multivariable model. Multivariable logistic regression was then constructed using a stepwise backward elimination (likelihood-ratio) procedure, with variable entry P=0.05 and removal P=0.10; clinically important covariates (e.g., age and sex) were retained regardless of statistical significance when appropriate. Multicollinearity was assessed before modeling (e.g., variance inflation factor), and highly collinear predictors were not entered simultaneously. Multivariable results are reported as β coefficients, standard errors (SE), Wald χ^2 statistics, adjusted odds ratios (aORs) with 95% confidence intervals (CIs), and *P* values; model performance was evaluated using goodness-of-fit and discrimination indices (e.g., Hosmer-Lemeshow test and area under the ROC curve). Missing data (<5%) were handled using multiple imputation, and all statistical tests were two-tailed with P<0.05 considered statistically significant.

Results

Baseline clinical characteristics

Age (62.28 ± 9.42 vs. 63.93 ± 10.40 years), male sex (70.6% vs. 72.3%), and BMI (27.25 ± 2.56 vs. 27.82 ± 3.29 kg/m^2) were comparable between the PDCA and control groups (P=0.173-0.790). The prevalences of current smoking, hypertension, diabetes mellitus, dyslipidemia, prior coronary disease/PCI, and family history of premature CAD showed no between-group differences (all P>0.05). ACS presentation (STEMI/NSTEMI/unstable angina) was similarly distributed. Guideline-directed therapies at baseline were aligned: high-intensity statins in 93.1% of both groups; ezetimibe 9.8% vs. 7.9%; PCSK9 inhibitor 2.9% vs. 2.0%; and comparable use of β-blockers, ACEI/ARB, and dual antiplatelets (**Table 1**). These data confirm strong baseline comparability, enabling unbiased evaluation of the PDCA strategy.

Comparison of lipid profile dynamics between the two groups

As shown in **Figure 2**, baseline lipid profiles were comparable between groups; thereafter, the PDCA cycle-based strategy yielded a steeper and earlier improvement across indices. By 4 weeks - and sustained at 12 weeks - the PDCA group achieved greater reductions in LDL-C and total cholesterol than the control group, with consistently lower distributions on box plots and multiple pairwise contrasts reaching significance (P<0.001). Triglycerides followed the same pattern, declining more prominently un-

Table 1. Baseline clinical and biochemical characteristics

Parameter	Control Group (n=101)	PDCA Group (n=102)	X^2/t	*P* value
Age (years)	63.93 ± 10.40	62.28 ± 9.42	1.183	0.238
Male, n (%)	73 (72.3)	72 (70.6)	0.071	0.790
Body mass index (kg/m^2)	27.82 ± 3.29	27.25 ± 2.56	1.367	0.173
Current smoker, n (%)	41 (40.6)	44 (43.1)	0.135	0.713
Hypertension, n (%)	63 (62.4)	62 (60.8)	0.054	0.816
Diabetes mellitus, n (%)	35 (34.7)	35 (34.3)	0.003	0.959
Dyslipidemia, n (%)	58 (57.4)	60 (58.8)	0.041	0.840
Previous coronary artery disease, n (%)	20 (19.8)	18 (17.6)	0.155	0.694
Prior PCI, n (%)	17 (16.8)	14 (13.7)	0.378	0.538
Family history of premature CAD, n (%)	11 (10.9)	13 (12.7)	0.167	0.682
STEMI, n (%)	33 (32.7)	31 (30.4)	0.122	0.727
NSTEMI, n (%)	39 (38.6)	40 (39.2)	0.008	0.930
Unstable angina, n (%)	29 (28.7)	31 (30.4)	0.069	0.793
High-intensity statin use, n (%)	94 (93.1)	95 (93.1)	0.001	0.985
Ezetimibe co-therapy, n (%)	8 (7.9)	10 (9.8)	0.223	0.637
PCSK9 inhibitor initiated, n (%)	2 (2.0)	3 (2.9)	0.195	0.659
Beta-blocker use, n (%)	91 (90.1)	93 (91.2)	0.069	0.792
ACEI/ARB use, n (%)	84 (83.2)	86 (84.3)	0.049	0.825

der PDCA-guided care, while HDL-C showed a modest, favorable shift relative to control (P<0.001). The coherent directionality across LDL-C, TC, and TG, together with the earlier separation of curves, indicates a meaningful time-by-group effect, supporting the capacity of a PDCA framework to accelerate and consolidate lipid optimization after PCI in ACS.

Comparison of LDL-C goal achievement rate between the two groups

At 12 weeks, LDL-C goal attainment was significantly higher in the PDCA group than in the control group across all targets. The proportion achieving LDL-C <1.8 mmol/L was 57.8% versus 31.7%, and ≥50% LDL-C reduction was 61.7% versus 28.7% (both P<0.001). Dual goal achievement was 49.0% in the PDCA group compared with 20.8% in controls (P<0.001). Even at more stringent thresholds - LDL-C <1.4 mmol/L and <1.0 mmol/L - the PDCA group maintained clear superiority (38.9% vs. 15.2% and 25.6% vs. 8.3%, respectively; P<0.001 and P=0.001) (**Table 2**). These results highlight the robust efficacy of the PDCA cycle-based strategy in achieving both standard and intensive lipid-lowering goals.

Comparison of medication adherence between the two groups

Medication adherence and regimen optimization were markedly superior in the PDCA group compared with the control group. High-intensity statin use was substantially higher in the PDCA group (92.2% vs. 64.3%, P<0.001), with greater use of adjunctive agents such as ezetimibe (42.2% vs. 14.9%, P<0.001) and PCSK9 inhibitors (15.7% vs. 3.0%, P=0.002). The proportion of patients undergoing at least one dose adjustment within four weeks was also significantly greater in the PDCA group (78.4% vs. 19.8%, P<0.001). Overall medication adherence rates were notably higher in the PDCA cohort (94.38 ± 10.64% vs. 83.63 ± 11.56%, P<0.001) (**Table 3**), underscoring the effectiveness of the PDCA cycle-based strategy in promoting early, intensive, and sustained lipid-lowering management.

Comparison of blood biochemical indicators between the two groups

Both groups had comparable baseline biochemical parameters, and no significant differences were observed at 4 or 12 weeks (all P>0.05). Serum creatinine, eGFR, liver enzymes

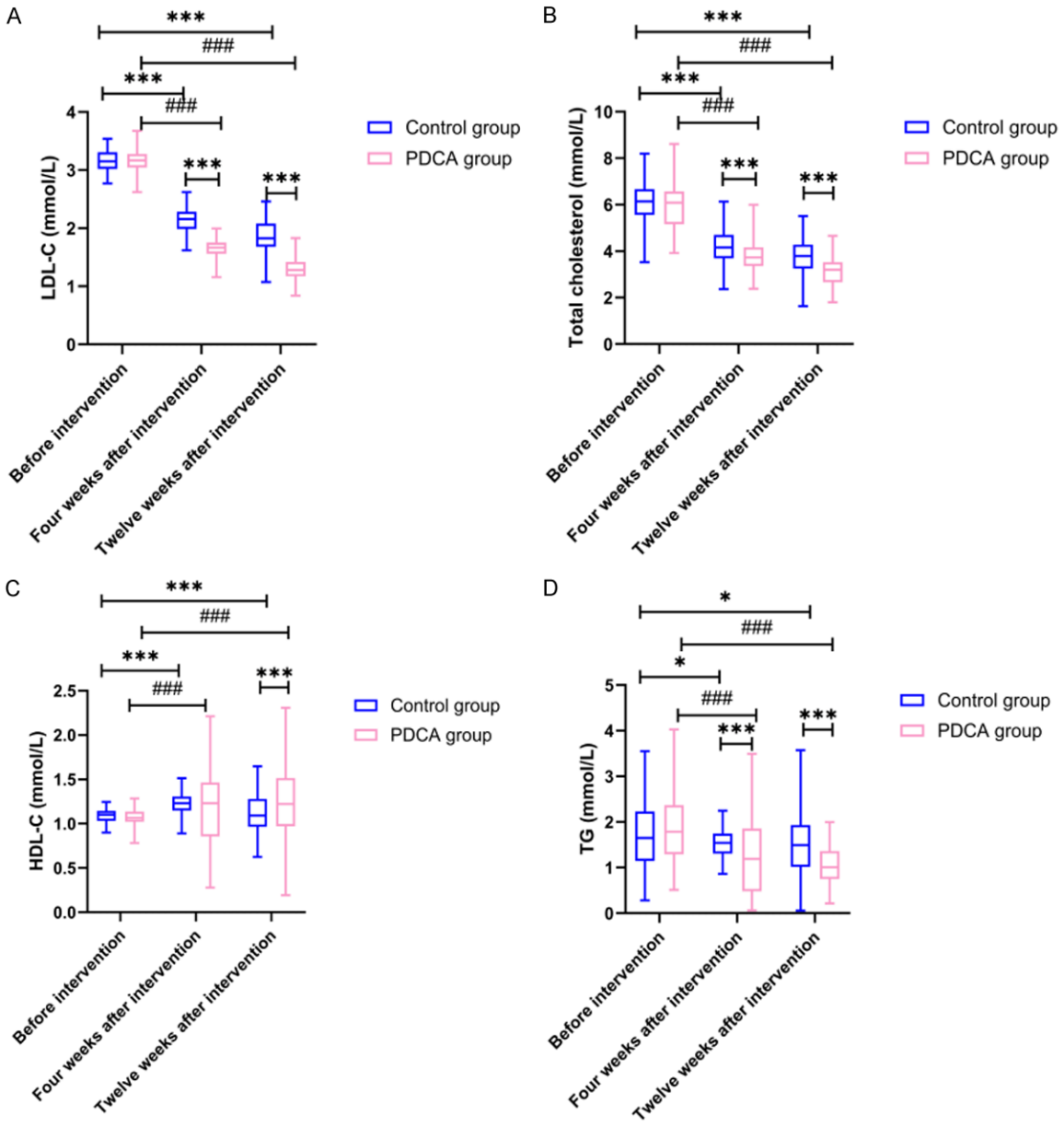

Figure 2. Comparison of lipid profile dynamics between the two groups. (A) LDL-c, (B) Total cholesterol, (C) HDL-C, (D) TG. Compare to the control group before intervention, *P<0.05, ***P<0.001. Compare to the PDCA group before intervention, ###P<0.001.

Table 2. Comparison of LDL-C goal achievement rate between the two groups

LDL-C Target	Control Group (n=101)	PDCA Group (n=102)	X^2	*P* Value
LDL-C <1.8 mmol/L	32 (31.7%)	59 (57.8%)	14.042	<0.001
≥50% LDL-C reduction	29 (28.7%)	63 (61.7%)	22.372	<0.001
Dual goal achievement	21 (20.8%)	50 (49.0%)	17.780	<0.001
LDL-C <1.4 mmol/L (very-high-risk target)	15 (15.2%)	40 (38.9%)	15.251	<0.001
LDL-C <1.0 mmol/L (intensive target)	8 (8.3%)	26 (25.6%)	11.235	0.001

Table 3. Comparison of medication adherence between the two groups

Parameter	Control Group (n=101)	PDCA Group (n=102)	X^2/t	*P* Value
High-intensity statin use (%)	65 (64.3%)	94 (92.2%)	23.103	<0.001
Addition of ezetimibe (%)	15 (14.9%)	43 (42.2%)	18.540	<0.001
PCSK9 inhibitor initiation (%)	3 (3.0%)	16 (15.7%)	9.673	0.002
≥1 dose adjustment within 4 weeks (%)	20 (19.8%)	80 (78.4%)	69.793	<0.001
Medication adherence rate (%)	83.63 ± 11.56	94.38 ± 10.64	6.892	<0.001

(ALT, AST), fasting glucose, HbA1c, and inflammatory markers (hs-CRP, TNF-α) remained stable throughout follow-up (**Figure 3**).

Comparison of cardiac ultrasound indicators between the two groups

Serial echocardiography revealed greater improvement in cardiac function in the PDCA group compared with controls. From baseline to 12 weeks, left ventricular ejection fraction (LVEF) increased significantly in the PDCA group (53.84 ± 6.71 → 62.00 ± 6.09%) versus the control group (52.91 ± 5.59 → 53.65 ± 6.09%; P<0.001). Correspondingly, left ventricular end-diastolic diameter (LVEDD) and end-systolic diameter (LVESD) showed greater reductions in the PDCA group (LVEDD: 52.73 ± 4.79 → 42.63 ± 7.70 mm; LVESD: 34.87 ± 3.73 → 29.89 ± 4.09 mm) than in controls (LVEDD: 53.00 ± 6.24 → 52.34 ± 5.27 mm; LVESD: 34.90 ± 3.27 → 35.85 ± 4.25 mm; both P<0.001). The PDCA group also demonstrated lower E/e' ratios at 4 and 12 weeks (both P<0.001) and a significant decline in left atrial volume index (LAVI) from 36.20 ± 5.24 to 29.16 ± 6.00 mL/m^2 (P<0.001) (**Table 4**). Collectively, these results indicate that PDCA-based intensive lipid-lowering management was associated with improved left ventricular remodeling and diastolic function during short-term follow-up after PCI.

Comparison of lifestyle modification adherence between the two groups

Medication adherence (MPR ≥80%) was achieved in 93.1% of PDCA patients versus 71.3% in controls (OR 5.64, 95% CI 2.29-13.9, P< 0.001). Adherence to a Mediterranean-style diet was also greater (71.6% vs. 42.6%, OR 3.43, 95% CI 1.86-6.33, P<0.001), as was engagement in physical activity ≥150 min/week (66.7% vs. 36.6%, OR 3.45, 95% CI 1.92-6.22, P<0.001). Smoking cessation among baseline smokers was markedly improved in the PDCA group (63.2% vs. 33.3%, P=0.004). Additionally, PDCA participants were more likely to achieve ≥5% body weight reduction (28.4% vs. 10.9%, P=0.004), perform home blood pressure monitoring ≥5 days/week (70.6% vs. 38.6%, P<0.001), and maintain optimal sleep duration (7-8 h/day; 69.6% vs. 47.5%, P= 0.003). Participation in psychological or cardiac rehabilitation sessions was also substantially higher (52.9% vs. 18.8%, P<0.001) (**Table 5**). Collectively, these results indicate that the PDCA cycle-based intervention effectively promoted comprehensive lifestyle optimization, reinforcing its value in secondary prevention after PCI.

Multivariate logistic regression for LDL-C goal achievement

Multivariate logistic regression identified the PDCA intervention as an independent predictor of LDL-C goal achievement (β=2.236, OR 9.353, 95% CI 4.424-19.775, P<0.001). High-intensity statin use (β=0.100, OR 0.905, 95% CI 0.868-0.943, P<0.001) and ezetimibe use (β=0.896, OR 0.408, 95% CI 0.220-0.757, P= 0.004) were also significant contributors (**Table 6**). Collectively, these findings indicate that PDCA-based management, particularly when combined with intensive lipid-lowering therapy, markedly enhances the likelihood of achieving LDL-C targets after PCI.

Discussion

This research has shown that an early intensive lipid-lowering strategy of PDCA cyclic considerably enhanced the achievement of LDL-C targets, lifestyle adherence, and cardiac performance in ACS patients after PCI and did not cause adverse events. PDCA-managed care, as compared to conventional care, was associ-

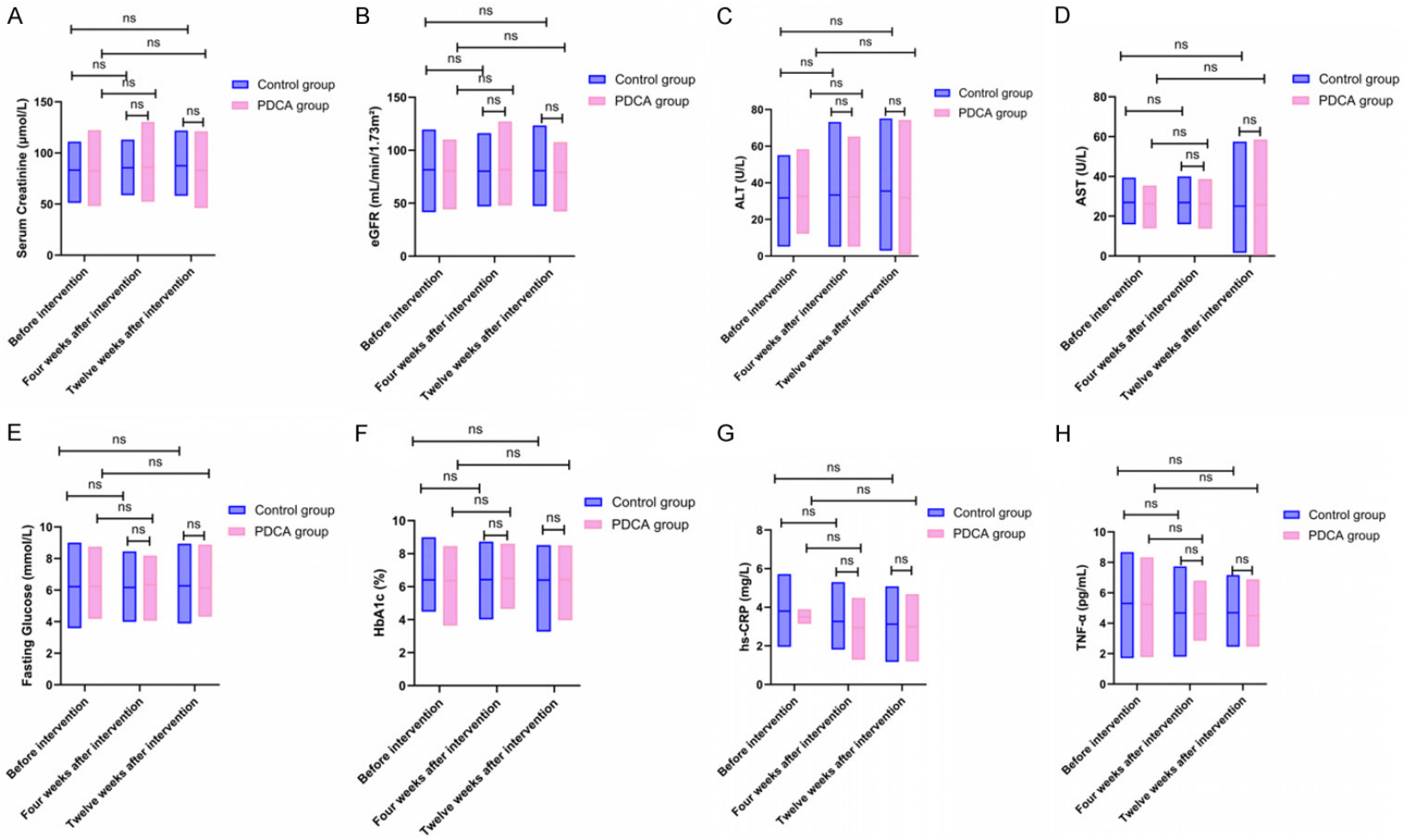

Figure 3. Comparison of blood biochemical indicators between the two groups. A. Serum creatinine levels; B. Estimated glomerular filtration rate (eGFR); C. Alanine aminotransferase (ALT); D. Aspartate aminotransferase (AST); E. Fasting glucose; F. Glycated hemoglobin (HbA1c); G. High-sensitivity C-reactive protein (hs-CRP); H. Tumor necrosis factor-α (TNF-α). Compare to the control group/PDCA group before intervention, $^{ns}P>0.05$.

Table 4. Comparison of cardiac ultrasound indicators between the two groups

Parameter	Timepoint	Control Group (n=101)	PDCA Group (n=102)	t	*P*-value (Between Groups)
Left Ventricular Ejection Fraction (LVEF, %)	Baseline	52.91 ± 5.59	53.84 ± 6.71	1.065	0.288
	4 weeks	53.34 ± 5.70	58.86 ± 9.90	4.863	<0.001
	12 weeks	53.65 ± 6.14	62.00 ± 6.09	9.721	<0.001
Left Ventricular End-Diastolic Diameter (LVEDD, mm)	Baseline	53.00 ± 6.24	52.73 ± 4.79	0.345	0.731
	4 weeks	52.81 ± 5.38	45.08 ± 6.36	9.332	<0.001
	12 weeks	52.34 ± 5.27	42.63 ± 7.70	10.471	<0.001
Left Ventricular End-Systolic Diameter (LVESD, mm)	Baseline	34.90 ± 3.27	34.87 ± 3.73	0.054	0.957
	4 weeks	35.46 ± 3.62	28.77 ± 3.90	12.671	<0.001
	12 weeks	35.85 ± 4.25	29.89 ± 4.09	10.192	<0.001
E/e' Ratio	Baseline	13.56 ± 2.26	13.02 ± 2.16	1.739	0.084
	4 weeks	13.23 ± 2.55	9.16 ± 2.54	11.359	<0.001
	12 weeks	13.27 ± 2.11	9.22 ± 2.20	13.375	<0.001
Left Atrial Volume Index (LAVI, mL/m^2)	Baseline	36.13 ± 6.57	36.20 ± 5.24	0.086	0.931
	4 weeks	36.23 ± 5.48	29.53 ± 5.68	8.559	<0.001
	12 weeks	36.47 ± 6.24	29.16 ± 6.00	8.507	<0.001

Table 5. Comparison of lifestyle modification adherence between the two groups

Parameter	Control Group (n=101)	PDCA Group (n=102)	X^2	*P*-value
Medication adherence ≥80% (MPR)	72 (71.3%)	95 (93.1%)	16.608	<0.001
Dietary adherence (Mediterranean score ≥7)	43 (42.6%)	73 (71.6%)	17.421	<0.001
Physical activity ≥150 min/week	37 (36.6%)	68 (66.7%)	1.332	<0.001
Body weight reduction ≥5% from baseline	11 (10.9%)	29 (28.4%)	9.868	0.002
Home blood pressure monitoring ≥5 days/week	39 (38.6%)	72 (70.6%)	20.937	<0.001
Sleep duration 7-8 h/day adherence	48 (47.5%)	71 (69.6%)	10.203	0.001
Participation in psychological or cardiac rehabilitation sessions	19 (18.8%)	54 (52.9%)	25.669	<0.001

Table 6. Multivariate logistic regression for LDL-C goal achievement

Variable	β Coefficient	OR (95% CI)	P
PDCA intervention	2.236	9.353 (4.424-19.775)	<0.001
High-intensity statin use	0.100	0.905 (0.868-0.943)	<0.001
Ezetimibe use	0.896	0.408 (0.220-0.757)	0.004

Assignments of Variables: PDCA intervention: Whether the patient was in the PDCA cycle-based intervention group or the control group; High-intensity statin use: Whether the patient was prescribed a high-intensity statin (atorvastatin 40-80 mg or rosuvastatin 20-40 mg daily) after PCI; Ezetimibe use: Whether the patient was prescribed ezetimibe as an adjunct therapy for lipid management.

ated with more timely and stronger LDL-C, TC and TG reductions, a greater rate of dual and triple goal LDL-C attainment, the increased use of combination lipid-lowering therapy, and the increased adherence to medication and lifestyle interventions. Importantly, these advantages were realized with similar safety, which demonstrates the practicability and clinical importance of considering continuous quality-improvements related to lipid control in actual cardiology practice.

The greatest observation was the rapid and maintained regression of LDL-C and total cholesterol relative to PDCA-managed treatment. It has been demonstrated in the past that early increases in lipid-lowering therapy during a secondary prevention are vital, but clinical inertia habitually postpones achieving the ideal target [13-16]. These observations are further extended to indicate that organized feedback and unrelenting audit in the PDCA cycle can suc-

cessfully defeat this inertia. Mechanically, the framework promotes dose titration proactivity, regular analysis of lipid outcomes, and personalized increase, resulting in an earlier separation and stabilization of the lipid profile curve. This early management of LDL-C, clinically, has been linked to the improvement of plaque stabilization and decreased recurring ischemic risk, which demonstrates the significance of dynamic management systems as opposed to fixed treatment algorithms.

By 12 weeks, the rates of patients meeting conventional (<1.8 mmol/L) and intensive (<1.4 mmol/L and <1.0 mmol/L) LDL-C goals were much higher in the PDCA group compared to rates of some recent real-world registries (e.g. DA VINCI and GOULD studies). These results indicate that there always exists a disparity between the guidelines and the clinical practice, and the PDCA cycle seems effective in bridging it. The repeated patterns of checks and actions helps to introduce a timely deepening of therapy, especially the increased use of high-thoroughness statins, etzetimibe, and PCSK9-inhibitors. This is reflected by the IMPROVE-IT and FOURIER studies which also documented the same, asserting that the additive benefit of early combination lipid therapy exists [17]. The formalized PDCA process, therefore, is a scaled behavioral and systems based intervention to celebrate guideline non adherence and transform evidence to standard clinical practice.

The adherence to medication was identified as one of the determinants of success within the PDCA model. Unlike traditional models of patient education where no changes happen after a program, PDCA-based care entails constant monitoring and feedback, which is more accountable and engages the patient. Our adherence rates (>95%) surpass those reported in most secondary prevention cohorts. This can be attributed to repetitive reinforcement, quick reaction to side effects, and joint decision-making as part of the do-check stages. It is reported in previous behavioral study articles that high adherence is likely mediated by iterative reinforcement, which enhances persistence and self-efficacy [18]. Clinically, these findings serve as a warning against only adhering to pharmacologic potency; achieving long-term adherence, facilitated by systemic feedback loops, that can be rapidly gained over a long period to reach lipid goals.

Although pharmacologic regimens were intensified, there were no differences in the incidence of adverse events, consistent with other major lipid-lowering studies showing that high-intensity regimens are safe under careful monitoring [19-22]. Risk might also be addressed naturally by the PDCA system as it allows the early recognition of laboratory abnormalities or symptoms and prompt treatment which stops discontinuation. Constancy in hepatic and renal biochemical measurements during the post-follow-up also shows the safety of the strategy. These findings highlight that process management can be used to increase the intensity of the process, which does not reduce tolerability, which is an essential reassurance among clinicians unwilling to intensify therapy in the post-PCI patient.

In addition to lipid statistics, PDCA-based care produced objective results in terms of cardiac remodeling indices, such as LVEF, LVDD, and LVESD as well as diastolic parameters. Such changes are probably due to the synergistic influence of the optimized level of lipids, better adherence to medications, and better lifestyle modification. Outcomes of previous research have associated aggressive LDL-C reduction with positive left ventricular remodeling and decreased inflammation, which partially could be the reason behind the reported echocardiographic improvements [23, 24]. In addition, the cardioprotective effect is enhanced by adherence to the Mediterranean diet, physical activity, smoking cessation, and weight control, which were much greater in the PDCA group [25, 26]. All these findings point to the overall effect of the PDCA framework, which includes pharmacologic, behavioral, and rehabilitative components to stimulate lasting cardiac outcomes.

The PDCA cycle is an adaptive collection of feedback care that can convert a baseline follow-up to an adaptive learning mechanism of precision medicine. On the mechanistic level, it helps to improve real-time performance evaluation and makes clinicians and patients accountable. This overcomes the therapeutic inertia that is endemic in chronic cardiovascular care by institutionalizing the routine monitoring of results and modifications. These understand-

ings imply that quality-improvement models can extend beyond administrative instruments, these models may directly impact the biological outcomes. Clinically, importation of PDCA logic into data platforms of digital health or lipid clinics can yield a scalable and low-cost opportunity to assist lipid targets dictated by the guidelines worldwide.

Several limitations can be noted in this study. To start with, being a single-center study with moderate sample size might prevent generalization to other healthcare systems. Second, 12-weeks of primary follow-up only yields early and not long-term lipid objectives, nor prevention maintenance. Third, the self-reported activity on adherence/lifestyle behaviors can cause response bias, which enhances validity when such measures are consistent. Lastly, though the echocardiograph changes were great, we did not do mechanistic imaging (e.g., plaque regression) which should be included in future studies to clarify structural links of PDCA-motivated management.

In conclusion, the PDCA cycle based-early intensive lipid-lowering' strategy was associated with a significant enhancement in the management of LDL-C, medication adherence, lifestyle changes, and cardiac performance without causing adverse events to occur more often in ACS patients after PCI. This will fill in the evidence to practice gap, with the field of application of this approach being translation of continuous quality improvement principles into clinical practice, which provides a reproducible model to meet contemporary lipid targets. The next generation, multi-site and long-term research needs to investigate how it can be combined with digital monitoring, as well as AI-based analytics, to maintain and enhance cardiovascular outcomes.

Disclosure of conflict of interest

None.

Abbreviations

ACS, Acute Coronary Syndrome; ACEI, Angiotensin-Converting Enzyme Inhibitor; ARB, Angiotensin Receptor Blocker; ASCVD, Atherosclerotic Cardiovascular Disease; BMI, Body Mass Index; CAD, Coronary Artery Disease; CK, Creatine Kinase; CQI, Continuous Quality Improvement; eGFR, Estimated Glomerular Filtration Rate; HDL-C, High-Density Lipoprotein Cholesterol; hs-CRP, High-Sensitivity C-Reactive Protein; LDL-C, Low-Density Lipoprotein Cholesterol; LLT, Lipid-Lowering Therapy; LVEF, Left Ventricular Ejection Fraction; LVEDD, Left Ventricular End-Diastolic Diameter; LVESD, Left Ventricular End-Systolic Diameter; LAVI, Left Atrial Volume Index; MACE, Major Adverse Cardiovascular Events; MPR, Medication Possession Ratio; NSTEMI, Non-ST-Segment Elevation Myocardial Infarction; PCI, Percutaneous Coronary Intervention; PCSK9, Proprotein Convertase Subtilisin/Kexin Type 9; PDCA, Plan-Do-Check-Act; STEMI, ST-Segment Elevation Myocardial Infarction; TC, Total Cholesterol; TG, Triglycerides; TNF-α, Tumor Necrosis Factor Alpha; ULN, Upper Limit of Normal.

Address correspondence to: Hongyuan Wang, Department of Cardiology, Jiangshan People's Hospital, No. 9 Hangbu Mountain Road, Jiangshan 324100, Zhejiang, China. Tel: +86-0570-4233979; E-mail: 15268066998@163.com

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