ORIGINAL ARTICLE

Glycemic Control and Cholesterol Control Attainment following Acute Coronary Syndrome in Patients with Type 2 Diabetes Mellitus Undergoing Percutaneous Coronary Intervention

Tanapol Juntaruk, MD[1], Peeradon Dutsadeevettakul[2], Phanthaphan Sureeyathanaphat, MD[1,3]

[1] Department of Internal Medicine, Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand; [2] Medical student, Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand; [3] Cardiology Division, Department of Internal Medicine, Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand

Background: Cardiovascular disease (CVD) is a leading cause of mortality and disability in patients with type 2 diabetes mellitus (T2DM), with elevated risks of recurrent cardiovascular events after acute coronary syndrome (ACS). Achieving optimal glycemic and lipid targets, including hemoglobin A1c (HbA1c), low-density lipoprotein cholesterol (LDL-C), and non-high-density lipoprotein cholesterol (non-HDL-C), is critical for secondary prevention in this high-risk population.

Objective: To assess HbA1c, LDL-C, and non-HDL-C target attainment rates in T2DM patients after percutaneous coronary intervention (PCI) for ACS. Additionally, to evaluate the prevalence of atherogenic dyslipidemia and explores predictive factors influencing target achievement.

Materials and Methods: A retrospective, longitudinal descriptive study was conducted on 420 patients with T2DM who experienced ACS and underwent PCI at Vajira Hospital between January 2017 and December 2021. Data on baseline characteristics, laboratory values, and medication regimens were collected. Target attainment was defined as HbA1c of less than 7%, LDL-C of less than 55 mg/dL with 50% or more reduction, and non-HDL-C of less than 85 mg/dL. Logistic regression analysis identified factors predicting target achievement.

Results: At six months, 76.0% of patients achieved HbA1c of less than 7%, while only 17.6% and 37.6% met LDL-C and non-HDL-C targets, respectively. At twelve months, HbA1c attainment remained high at 76.4%, but LDL-C and non-HDL-C targets were less frequently achieved at 21.4% and 44.0%, respectively. Atherogenic dyslipidemia prevalence decreased from 16.9% at baseline to 12.14% at twelve months. Male gender predicted HbA1c target achievement, while baseline HbA1c, sulfonylurea, and insulin use were negative predictors. Ezetimibe treatment and the civil servant health scheme positively influenced LDL-C target attainment.

Conclusion: Despite high rates of HbA1c target attainment, LDL-C, and non-HDL-C goals were achieved less frequently, highlighting gaps in lipid management in T2DM patients post-ACS. Enhanced strategies for comprehensive secondary prevention, particularly lipid control, are necessary to improve outcomes in this population.

Keywords: Type 2 diabetes mellitus; Acute coronary syndrome; HbA1c; LDL-C; Non-HDL-C; Secondary prevention; Atherogenic dyslipidemia

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Cardiovascular disease (CVD) is a major cause of mortality and disability in patients with diabetes mellitus[1]. The prevalence of CVD in diabetic patients is twice as high as that in non-diabetic individuals[2]. Particularly among patients with type 2 diabetes mellitus (T2DM), the prevalence of CVD reaches 32.2%, with most cases attributed to coronary artery disease (CAD). CVD is also responsible for up to 50.3% of deaths in individuals with T2DM[3]. Findings from the INTERHEART Study indicate that both the ApoB/ApoA1 ratio and diabetes status are significant risk factors for acute myocardial infarction (AMI)[4]. A meta-analysis further demonstrated that diabetic patients with CAD who undergo percutaneous coronary intervention (PCI) have a poorer prognosis if they presented with elevated glycosylated hemoglobin (HbA1c) levels,

Correspondence to:

Sureeyathanaphat P.

Cardiology Division, Department of Internal Medicine, Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, Bangkok 10300, Thailand.

Phone: +66-89-4314594

Email: phanthaphan@nmu.ac.th

ORCID: 0009-0008-5564-757X

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with HbA1c cut points varying across studies[5].

Previous studies have shown that coronary revascularization, including PCI, improves clinical outcomes for patients with acute coronary syndrome (ACS). However, these patients remain at higher risk of recurrent cardiovascular events compared to healthy individuals or patients with stable CAD[6]. Consequently, long-term therapy following an AMI episode, including both ST-elevation ACS (STE-ACS) and non-ST-elevation ACS (NSTE-ACS), involves a combination of medication, lifestyle modifications, and risk factor management. According to the American College of Cardiology and American Heart Association (ACC/AHA) guidelines on managing hypercholesterolemia in post-ACS patients, high-intensity statin therapy is recommended to reduce low-density lipoprotein cholesterol (LDL-C) by 50% or more[7]. Similarly, the European Society of Cardiology (ESC) guidelines suggest reducing LDL-C by 50% or more and achieving an LDL-C level below 55 mg/dL[8].

For long-term blood glucose control in diabetic patients after ACS, it is recommended to consider glucose-lowering medication if blood glucose levels exceed 180 mg/dL[9,10]. The general HbA1c target for diabetic patients is below 7%, though individualized targets may be set based on patient characteristics[10,11].

T2DM patients are at high risk of residual atherosclerotic risk due to atherogenic dyslipidemia, characterized by elevated triglycerides, high small LDL-C, and low high-density lipoprotein cholesterol (HDL-C). Thus, in addition to managing LDL-C and HbA1c levels, guidelines on hypercholesterolemia management recommend a non-HDL-C target of less than 85 mg/dL in patients with atherosclerotic cardiovascular disease (ASCVD)[7].

Despite the importance of secondary prevention following AMI and the need for controlling LDL and HbA1c levels in high-risk diabetic patients, studies indicate that most patients fail to meet LDL-C targets[12,13]. Consistent with previous studies, only 30.1% to 40.3% of Thai patients with STE-ACS achieved their LDL-C goals[14,15].

Studies on diabetic patients who underwent PCI found that only 49.9% to 64.3% reached HbA1c targets below 7%[16,17]. In Thailand, research focusing on T2DM patients using HbA1c measurements showed that only 26.3% to 35.6% met the target HbA1c of less than 7%[18-20]. Additionally, another study found that only 34.4% of post-ACS patients achieved their non-HDL-C target, and those with non-HDL-C above 130 mg/dL had a higher incidence of major adverse cardiovascular events (MACE) compared to patients with non-HDL-C below 100 mg/dL[21].

At Vajira Hospital, a large number of diabetic patients experience ACS and receive PCI each year. During follow-up visits after hospital discharge, physicians often focus on reducing LDL-C to target levels but may not emphasize blood glucose and non-LDL cholesterol control. Furthermore, this patient population has limited research on blood glucose control. Therefore, the present study aimed to evaluate the success rate of HbA1c, LDL-C, and non-HDL-C control and to investigate the prevalence of atherogenic dyslipidemia in T2DM patients post-PCI for ACS at the Faculty of Medicine, Vajira Hospital, Navamindradhiraj University.

Objective

The primary objectives of the present study were to investigate the attainment rates of HbA1c, LDL-C, and non-HDL-C levels meeting target goals in patients with type 2 diabetes who have experienced ACS and were treated with coronary angiography and PCI at the Faculty of Medicine Vajira Hospital, Navamindradhiraj University. Additionally, the present study aimed to examine the prevalence of atherogenic dyslipidemia in these patients who received the same treatment.

The secondary objective was to identify factors predicting the ability to achieve target goals for these patients' HbA1c, LDL-C, and non-HDL-C levels.

Materials and Methods

Study design

The present study was a retrospective longitudinal descriptive study. The study population consisted of all patients with T2DM and AMI who underwent coronary angiography and PCI at the Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, between January 1, 2017, and December 31, 2021.

The inclusion criteria for this study were that participants had to be 18 years or older. They were required to have a diagnosis of AMI, including STE-ACS and NSTE-ACS, and to have been treated with PCI. Only data from the first MI occurrence was included for cases where PCI was performed multiple times. Additionally, participants were required to have a prior diagnosis of T2DM or to receive a T2DM diagnosis during the index admission for PCI. Participants were required to have undergone

treatment with LDL-lowering drugs and blood glucose control medication or diet control for at least 12 months following the index admission. Laboratory results, including HbA1c and LDL-C levels, had to be available within one month of the index admission. Additionally, complete laboratory data, including HbA1c, total cholesterol, LDL-C, HDL-C, and triglyceride levels must be available 6- and 12-months post-index admission.

The exclusion criteria for the present study included a follow-up period of less than 12 months after index admission and severe comorbidities or a life expectancy of less than one year, such as advanced cancer or terminal-stage diseases. Notably, there were no discontinuation criteria for the present study.

Sample size calculation

To determine the required sample size, the population proportion (p) was based on findings from the previous study in Vietnam[13]. The present study indicated that approximately 20% of high-cardiovascular-risk diabetes patients achieved an LDL-C level below 1.8 mmol/L or 70 mg/dL. Hence, the proportion p is 0.20.

The allowable margin of error (d) was set at 0.04. Substituting these values into the formula:

$N = 1.96^2 \times [0.20 \times (1–0.20)] / (0.04)^2 = 384$

Thus, a sample size of 384 participants was required for the present study.

Data collection

The authors examined the electronic medical records for baseline characteristics and laboratory values, including age, gender, body mass index (BMI), comorbidities, duration of diabetes, HbA1c level, total cholesterol (TC), LDL-C, HDL-C, and triglycerides levels, type of ACS, medications received, patient's health coverage scheme, and primary follow-up clinic for diabetes. The authors also collected the HbA1c level, total cholesterol, LDL-C, HDL-C, and triglycerides levels at six months and twelve months after the index event from the electronic medical records to evaluate the primary outcomes. Diagnosis and type of ACS were based on the diagnosis given in the electronic medical records. Patients were considered achievers if their HbA1c was less than 7% at the follow-up period. The LDL-C goal was achieved if there was a reduction in LDL-C of more than 50% and LDL-C level of less than 55 mg/dL at follow-up. The non-HDL-C could be calculated as TC – HDL-C. Patients met the non-HDL cholesterol goal if their non-HDL cholesterol level was less than 85 mg/dL at the follow-up period. Lastly, atherogenic dyslipidemia is defined as having a triglyceride level of more than 150 mg/dL and HDL-C of less than 40 mg/dL. For missing data, only participants with complete data for all variables of interest were included in the study.

Statistical analysis

Quantitative data such as age, BMI, duration of diabetes, HbA1c level, total cholesterol, LDL-C, HDL-C, and triglycerides levels. For continuous quantitative variables, statistical analysis was performed using the student's t-test, with statistical significance defined as a p-value of less than 0.05. Continuous variables with a normal distribution were presented as mean and standard deviation, while continuous variables with a skewed distribution are presented as median and interquartile range.

Qualitative data included gender, comorbidities, type of ACS, medications received, and follow-up clinic. These data were presented using frequency and percentage distributions.

Univariate and multivariate logistic regression analyses were employed to determine factors predicting unsuccessful control of HbA1c, LDL-C, and non-HDL-C levels. All data analyses were conducted using the IBM SPSS Statistics for MacOS, version 29.0 (IBM Corp., Armonk, NY, USA).

Ethical considerations

The present study received approval from the Human Research Ethics Committee at Navamindradhiraj University (COA 130/66E).

Results

Patient baseline characteristics

Between January 2017 and December 2021, 420 adult patients with T2DM patients and AMI underwent coronary angiography and PCI were included in the present study. The baseline characteristics are shown in Table 1. The average age of the patients was 66.79±12.76 years and 39.8% were female. The mean BMI of the patients was 24.47±4.58 kg/m^2. The incidence of comorbidities was as follows: hypertension at 100%, history of cerebral infarction at 5.7%, peripheral artery disease at 0.5%, and chronic kidney disease at 38.6%. The mean duration of diabetes at the time of the study was 4.61±6.07 years. One hundred seven patients (25.5%) had STE-ACS, while 313 patients (74.5%) had NSTE-ACS. Three hundred thirty-six patients (80.0%) received

Table 1. Baseline characteristics and laboratory values of patients in the study

Characteristics	All patient (n=420)
Female sex; n (%)	167 (39.8)
Age (years); mean±SD	66.79±12.76
BMI (kg/m^2); mean±SD	24.47±4.58
Primary clinic for diabetes; n (%)	
Cardiology	336 (80.0)
Endocrine	7 (1.7)
General medicine	13 (3.1)
Combined	63 (15.0)
Other	1 (0.2)
Health coverage; n (%)	
Universal coverage	253 (60.2)
Social security	39 (9.3)
Civil servant benefit	93(22.1)
Self-funded	35 (8.3)
Smoking status; n (%)	
Current smoker	65 (15.5)
Ex-smoker	148 (35.2)
Non-smoker	207 (49.3)
Comorbidity; n (%)	
Hypertension	420 (100)
History of cerebral infarction	24 (5.7)
Peripheral artery disease	2 (0.5)
Chronic kidney disease	162 (38.6)
Diabetes duration to index time (years); mean±SD	4.61±6.07
Type of ACS; n (%)	
STE-ACS	107 (25.5)
NSTE-ACS	313 (74.5)
Baseline of Laboratory result at index time; mean±SD	
HbA1c (%)	6.88±1.90
Total cholesterol (mg/dL)	184.50±55.32
LDL-C (mg/dL)	119.66±49.74
HDL-C (mg/dL)	46.95±13.20
Triglyceride (mg/dL)	143.28±82.11
Non-HDL-C (mg/dL)	137.12±55.21
eGFR (mL/minute)	64.36±30.91
CKD stage; n (%)	
G1-G2	256 (61.0)
G3	86 (20.5)
G4	31 (7.4)
G5	47 (11.2)
Diabetes treatment at index time; n (%)	
Sulfonylurea	69 (16.4)
TZD	19 (4.5)
SGLT2I	38 (9.0)
Insulin	67 (16.0)
Biguanide	110 (26.2)
DPP4i	25 (6.0)
GLP1a	5 (1.2)
Dyslipidemia treatment at index time; n (%)	
Statin	415 (98.8)
Statin intensity	
• Low	5 (1.2)
• Moderate	1 (0.2)
• High	414 (98.6)
Ezetimibe	70 (16.7)
Fenofibrate	15 (3.6)
PCSK9i	0 (0.0)

SD=standard deviation; BMI=body mass index; ACS=acute coronary syndrome; STE-ACS=ST-elevation ACS; NSTE-ACS=non-ST-elevation ACS; HbA1c=hemoglobin A1c; LDL-C=low-density lipoprotein cholesterol; HDL-C=high-density lipoprotein cholesterol; eGFR=estimated glomerular filtration rate; CKD=chronic kidney disease; TZD=thiazolidinedione; SGLT2I=sodium-glucose cotransporter-2 inhibitor; DPP4i=dipeptidyl peptidase-4 inhibitor; GLP1a=glucagon-like peptide-1 agonist; PCSK9i=proprotein convertase subtilisin kexin type 9 inhibitor

diabetes care at cardiology clinics, followed by 63 (15.0%) at the combined clinics, 13 (3.1%) at the general medicine clinics, and seven (1.7%) at the endocrine clinics. One patient (0.2%) was treated elsewhere. Two hundred fifty-three patients (60.2%) had universal health coverage, 93 (22.1%) had civil servant benefit schemes, 39 (9.3%) had social security schemes, and 35 (8.3%) were self-funded. Regarding smoking status, 65 patients (15.5%) were current smokers, 148 (35.2%) were ex-smokers, and 207 (49.3%) were non-smokers.

For diabetes treatment, 110 patients (26.2%) received biguanides, 69 (16.4%) sulfonylureas, 67 (16.0%) insulin, 38 (9.0%) sodium-glucose cotransporter-2 (SGLT2) inhibitors, 25 (6.0%) dipeptidyl peptidase-4 (DPP4) inhibitors, 19 (4.5%) thiazolidinediones (TZDs), and five (1.2%) glucagon-like peptide-1 (GLP-1) receptor agonists. For dyslipidemia management, 415 patients (98.8%) were prescribed statins, high-intensity in 414 patients (98.6%) with five (1.2%) receiving low-intensity, and one (0.2%) receiving moderate-intensity statins. Additionally, 70 patients (16.7%) were treated with ezetimibe, and 15 (3.6%) with fenofibrate, while no patients received proprotein convertase subtilisin kexin type 9 (PCSK9 inhibitors).

At baseline, the mean HbA1c was 6.88±1.90%, total cholesterol 184.50±55.32 mg/dL, LDL-C 119.66±49.74 mg/dL, HDL-C 46.95±13.20 mg/dL, triglycerides 143.28±82.11 mg/dL, and non-HDL-C

Table 2. HbA1c and LDL achievement rates at 6 and 12 months

	At 6 months; n (%)		At 12 months; n (%)	
	Achieved	Non-achieved	Achieved	Non-achieved
HbA1c	319 (76.0)	101 (24.0)	321 (76.4)	99 (23.6)
LDL	74 (17.6)	346 (82.4)	90 (21.4)	330 (78.6)
Non-HDL	158 (37.6)	262 (62.4)	185 (44.0)	235 (56.0)

HbA1c=hemoglobin A1c; LDL=low-density lipoprotein; HDL=high-density lipoprotein

137.12±55.21 mg/dL. The mean estimated glomerular filtration rate (eGFR) was 64.36±30.91 mL/minute. Chronic kidney disease (CKD) stages were distributed as 256 patients (61.0%) in G1-G2, 86 (20.5%) in G3, 31 (7.4%) in G4, and 47 (11.2%) in G5.

HbA1c, LDL-C, and non-HDL-C attainment

At six months post-ACS, 319 patients (76.0%) achieved HbA1c of less than 7%, while 101 (24.0%) did not. Three hundred twenty-one patients (76.4%) reached this target by twelve months, whereas 99 (23.6%) did not. For LDL-C, at six months 346 patients (82.4%) failed to achieve the goal of less than 55 mg/dL of LDL, and only 74 (17.6%) reached the target. By twelve months, 90 patients (21.4%) met the LDL-C goal, while 330 (78.6%) did not. Regarding non-HDL-C, 158 patients (37.6%) achieved the target by six months, while 262 (62.4%) did not. One hundred eighty-five patients (44.0%) reached the goal by twelve months, whereas 235 (56.0%) did not. The HbA1c, LDL-C, and non-HDL-C attainment rates are shown in Table 2.

Prevalence of atherogenic dyslipidemia

The prevalence of atherogenic dyslipidemia in the entire population was 16.9%, 12.62%, and 12.14% at baseline, six months, and twelve months, respectively.

Predictive factors for the ability to achieve target goals for HbA1c, LDL-C, and non-HDL-C levels

The results of the univariate and multivariate logistic regression analyses were summarized in Table 3-5. Male gender (adjusted odds ratio [AOR] 3.12, 95% CI 1.63 to 5.95, p≤0.001) was associated with the ability to reach the HbA1c target. Whereas the higher baseline of HbA1c (AOR 0.60, 95% CI 0.45 to 0.79, p≤0.001), treatment of sulfonylurea at index time (AOR 0.29, 95% CI 0.13 to 0.62, p=0.002), and treatment with insulin at index time (AOR 0.11, 95% CI 0.05 to 0.24, p≤0.001) decreased the probability of achieving the HbA1c goal.

For the ability to reach target goals for LDL-C, the civil servant benefit scheme (AOR 2.11, 95% CI 1.12 to 3.99, p=0.022), and ezetimibe treatment at index time (AOR 3.02, 95% CI 1.70 to 5.38, p≤0.001) were predictive factors to reach the target goal. In contrast, the increase in BMI (AOR 0.92, 95% CI 0.86 to 0.98, p=0.007) was associated with the likelihood of failure to reach the goal. Lastly, the only predictive factor associated with the ability to reach the target non-HDL-C was the civil servant benefit scheme (AOR 3.26, 95% CI 1.82 to 5.85, p≤0.001).

Discussion

Importantly, the present study is one of the first in Thailand to examine diabetic control by achievement of the HbA1c of less than 7% in patients with T2DM who have experienced ACS and were treated with coronary angiography and PCI, as they are at very high risk for recurrent cardiovascular events. The authors are also the first to assess lipid management in ACS patients in Thailand, using intensive LDL-C control defined by the reduction of more than 50% and to less than 55 mg/dL and non-HDL-C targets to less than 85 mg/dL, according to the 2019 ESC guidelines. The authors also study the prevalence of atherogenic dyslipidemia, which is one of the major risk factors for CVD in people with T2DM and people with abdominal obesity and insulin resistance or impaired glucose tolerance.

HbA1c is a vital marker of long-term glycemic control and is closely linked to the risk of both microvascular and macrovascular complications in diabetic patients undergoing PCI. Notably, the present study revealed that 76.0% and 76.4% of patients achieved an HbA1c level of less than 7% at six and twelve months, respectively. This represents a higher rate of HbA1c attainment compared to other studies involving populations with similar baseline HbA1c levels[17,22]. Several factors may have contributed to these favorable outcomes. Enhanced patient education and counseling on glycemic management, coupled with close follow-up and robust medication titration protocols, played a pivotal role. The availability of newer antidiabetic therapies with proven efficacy in achieving glycemic targets may also have positively influenced the results. Furthermore, the low baseline HbA1c levels in this patient population may have facilitated higher attainment rates, as patients with lower initial HbA1c often require fewer intensive interventions to achieve the target. In addition, the male gender has a strong association with the ability to reach the HbA1c target. This could be explained by various hypotheses such as differences in glucose

Table 3. Univariate and multivariate analyses to identify potential predictive factors for the inability to achieve target goals for HbA1c levels

Variable associated with achieving HbA1c	6-month				12-month			
	Univariate		Multivariate		Univariate		Multivariate	
	OR (95% CI)	p-value	AOR (95% CI)	p-value	OR (95% CI)	p-value	AOR (95% CI)	p-value
Male	1.88 (1.20 to 2.96)	0.006	2.48 (1.29 to 4.77)	0.006	2.46 (1.56 to 3.90)	<0.001	3.12 (1.63 to 5.95)	<0.001
Age (years)	1.03 (1.00 to 1.04)	0.006	1.00 (0.97 to 1.04)	0.856	1.02 (1.00 to 1.04)	0.38		
BMI (kg/m^2)	0.92 (0.88 to 0.97)	<0.001	0.98 (0.91 to 1.04)	0.447	0.92 (0.88 to 0.96)	<0.001	0.97 (0.90 to 1.04)	0.361
Cardio-clinic as primary clinic for diabetes	1.56 (0.92 to 2.65)	0.099			2.15 (1.28 to 3.61)	0.004	1.44 (0.69 to 3.00)	0.332
Health coverage								
Universal coverage	1.64 (0.77 to 3.49)	0.198			1.61 (0.74 to 3.49)	0.226		
Social security	2.39 (0.81 to 6.99)	0.113			1.78 (0.62 to 5.10)	0.286		
Civil servant benefit	1.79 (0.76 to 4.19)	0.180			1.32 (0.56 to 3.09)	0.525		
Self-funded	1 (Ref.)				1 (Ref.)			
Smoking status								
Current smoker	1.34 (0.68 to 2.66)	0.399			1.31 (0.66 to 2.59)	0.443		
Ex-smoker	1.04 (0.64 to 1.70)	0.864			1.06 (0.64 to 1.73)	0.830		
Non-smoker	1 (Ref.)				1 (Ref.)			
Comorbidity								
Hypertension	-	-			-	-	-	-
History of cerebral infarction	0.40 (0.17 to 0.94)	0.036			0.60 (0.25 to 1.44)	0.250		
Peripheral artery disease	-	-			-	-	-	-
Chronic kidney disease	1.36 (0.84 to 2.19)	0.211			0.96 (0.60 to 1.52)	0.848		
Diabetes duration to index time (years)	0.94 (0.91 to 0.98)	<0.001	0.97 (0.92 to 1.01)	0.157	0.92 (0.89 to 0.95)	<0.001	0.96 (0.91 to 1.01)	0.098
STE-ACS	0.86 (0.52 to 1.42)	0.552			1.26 (0.74 to 2.15)	0.396		
Baseline of laboratory result at index time								
HbA1c (%)	0.39 (0.32 to 0.48)	<0.001	0.58 (0.42 to 0.78)	<0.001	0.49 (0.41 to 0.57)	<0.001	0.60 (0.45 to 0.79)	<0.001
HbA1c <7%	18.52 (10.69 to 32.08)	<0.001	1.87 (0.69 to 5.06)	0.215	10.35 (6.20 to 17.27)	<0.001	0.74 (0.26 to 2.14)	0.579
Total cholesterol (mg/dL)					1.00 (0.99 to 1.01)	0.444		
LDL-C (mg/dL)	1.01 (0.99 to 1.02)	0.085			1.00 (0.99 to 1.01)	0.194		
HDL-C (mg/dL)	1.00 (0.98 to 1.03)	0.831			1.02 (0.99 to 1.03)	0.087		
Triglyceride (mg/dL)	1.00 (0.99 to 1.00)	0.928			0.99 (0.99 to 1.00)	<0.001	1.00 (0.99 to 1.01)	0.920
Non-HDL-C (mg/dL)	1.00 (0.99 to 1.00)	0.320			1.00 (0.99 to 1.01)	0.788		
eGFR (mL/minute)	1.00 (0.99 to 1.01)	0.910			0.99 (0.99 to 1.01)	0.701		
CKD stage								
G1-G2	1.06 (0.53 to 2.12)	0.867			1.28 (0.63 to 2.58)	0.495		
G3	1.49 (0.65 to 3.40)	0.646			1.26 (0.56 to 2.84)	0.574		
G4	1.32 (0.44 to 3.99)	0.618			1.31 (0.46 to 3.77)	0.616		
G5	1 (Ref.)				1 (Ref.)			
Diabetes treatment at index time								
Sulfonylurea	0.30 (0.15 to 0.59)	<0.001	0.65 (0.31 to 1.35)	0.248	0.16 (0.08 to 0.34)	<0.001	0.29 (0.13 to 0.62)	0.002
TZD	2.43 (0.74 to 8.00)	0.143			1.51 (0.54 to 4.26)	0.434		
SGLT2I	0.47 (0.19 to 1.15)	0.100			0.59 (0.24 to 1.48)	0.264		
Insulin	0.08 (0.04 to 0.16)	<0.001	0.37 (0.17 to 0.80)	0.012	0.04 (0.02 to 0.09)	<0.001	0.11 (0.05 to 0.24)	<0.001
Biguanide	0.31 (0.17 to 0.56)	<0.001	0.56 (0.29 to 1.07)	0.081	0.26 (0.14 to 0.50)	<0.001	0.55 (0.28 to 1.07)	0.078
DPP4i	1.23 (0.43 to 3.49)	0.701			0.39 (0.13 to 1.24)	0.112		
GLP1a	0.23 (0.02 to 2.42)	0.223			1.05 (0.22 to 5.01)	0.948		
Dyslipidemia treatment at index time								
Statin	-	-	-	-	-	-	-	-
Ezetimibe	0.75 (0.42 to 1.34)	0.335			0.84 (0.51 to 1.39)	0.504		
Fenofibrate	0.88 (0.27 to 2.81)	0.822			0.33 (0.12 to 0.88)	0.026		
PCSK9i	-	-	-	-	-	-	-	-

OR=odds ratio; AOR=adjusted odds ratio; CI=confidence interval; BMI=body mass index; STE-ACS=ST-elevation acute coronary syndrome; HbA1c=hemoglobin A1c; LDL-C=low-density lipoprotein cholesterol; HDL-C=high-density lipoprotein cholesterol; eGFR=estimated glomerular filtration rate; CKD=chronic kidney disease; TZD=thiazolidinedione; SGLT2I=sodium-glucose cotransporter-2 inhibitor; DPP4i=dipeptidyl peptidase-4 inhibitor; GLP1a=glucagon-like peptide-1 agonist; PCSK9i=proprotein convertase subtilisin kexin type 9 inhibitor

Table 4. Univariate and multivariate analyses to identify potential predictive factors for the inability to achieve target goals for LDL-C levels

Variable associated with achieving LDL	6-month				12-month			
	Univariate		Multivariate		Univariate		Multivariate	
	OR (95% CI)	p-value	AOR (95% CI)	p-value	OR (95% CI)	p-value	AOR (95% CI)	p-value
Male	0.79 (0.47 to 1.30)	0.350			0.70 (0.44 to 1.12)	0.132		
Age (years)	1.03 (1.01 to 1.05)	0.016			1.02 (1.01 to 1.04)	0.013		
BMI (kg/m^2)	0.87 (0.82 to 0.93)	<0.001	0.92 (0.85 to 0.99)	0.026	0.90 (0.85 to 0.95)	<0.001	0.92 (0.86 to 0.98)	0.007
Cardio-clinic as primary clinic for dyslipidemia	1.99 (0.95 to 4.20)	0.068			0.78 (0.44 to 1.36)	0.373		
Health coverage								
Universal coverage	1 (Ref.)		1 (Ref.)		1 (Ref.)		1 (Ref.)	
Social security	2.07 (0.87 to 4.95)	0.101	3.07 (1.12 to 8.43)	0.029	1.70 (0.75 to 3.86)	0.206	1.70 (0.69 to 4.19)	0.248
Civil servant benefit	4.63 (2.60 to 8.24)	<0.001	3.35 (1.56 to 7.16)	0.002	3.74 (2.18 to 6.41)	<0.001	2.11 (1.12 to 3.99)	0.022
Self-funded	1.04 (0.34 to 3.16)	0.949	1.21 (0.36 to 4.10)	0.755	1.17 (0.46 to 3.01)	0.744	0.96 (0.34 to 2.72)	0.935
Smoking status								
Current smoker	1 (Ref)				1 (Ref)			
Ex-smoker	1.17 (0.53 to 2.60)	0.696			0.78 (0.38 to 1.58)	0.487		
Non-smoker	1.24 (0.58 to 2.65)	0.584			0.98 (0.51 to 1.90)	0.950		
Comorbidity								
Hypertension	-	-			-	-		
History of cerebral infarction	0.63 (0.18 to 2.19)	0.470			1.54 (0.61 to 3.84)	0.359		
Peripheral artery disease	-	-			-	-		
Chronic kidney disease	1.90 (1.15 to 3.16)	0.013			1.53 (0.95 to 2.45)	0.078		
Diabetes duration to index time (years)	1.02 (0.98 to 1.06)	0.428						
STE-ACS	0.85 (0.49 to 1.53)	0.586						
Baseline of laboratory result at index time								
HbA1c (%)	0.87 (0.74 to 1.02)	0.091			0.98 (0.86 to 1.11)	0.735		
HbA1c <7%	1.52 (0.83 to 2.76)	0.174			1.10 (0.65 to 1.85)	0.734		
Total cholesterol (mg/dL)	0.98 (0.97 to 0.99)	<0.001	0.99 (0.97 to 1.01)	0.320	0.99 (0.98 to 0.99)	<0.001	0.98 (0.96 to 1.00)	0.118
LDL-C (mg/dL)	0.98 (0.97 to 0.98)	<0.001	0.99 (0.98 to 1.00)	0.173	0.99 (0.98 to 0.99)	<0.001	0.99 (0.99 to 1.00)	0.214
HDL-C (mg/dL)	1.01 (0.99 to 1.03)	0.412			1.00 (0.98 to 1.02)	0.741		
Triglyceride (mg/dL)	1.00 (0.99 to 1.00)	0.038			1.00 (0.99 to 1.00)	0.901		
Non-HDL-C (mg/dL)	0.98 (0.97 to 0.99)	<0.001	1.00 (0.97 to 1.02)	0.852	0.99 (0.98 to 0.99)	<0.001	1.01 (0.99 to 1.03)	0.298
eGFR (mL/minute))	0.98 (0.98 to 0.99)	<0.001	0.99 (0.96 to 1.01)	0.348	0.99 (0.98 to 1.00)	0.016		
CKD stage								
G1-G2	1 (Ref.)		1 (Ref.)		1 (Ref.)		1 (Ref.)	
G3	1.49 (0.78 to 2.87)	0.229	0.97 (0.30 to 3.21)	0.965	1.13 (0.61 to 2.09)	0.71	1.045 (0.53 to 2.08)	0.900
G4	5.38 (2.43 to 11.90)	<0.001	2.02 (0.31 to 12.96)	0.459	2.17 (0.96 to 4.93)	0.063	1.61 (0.65 to 4.03)	0.307
G5	1.77 (0.80 to 3.87)	0.157	0.40 (0.05 to 3.38)	0.402	2.59 (1.32 to 5.08)	0.006	1.84 (0.86 to 3.90)	0.115
Diabetes treatment at index time								
Sulfonylurea	0.79 (0.34 to 1.82)	0.579			0.44 (0.20 to 0.94)	0.043		
TZD	0.89 (0.22 to 3.59)	0.870			1.27 (0.44 to 3.67)	0.655		
SGLT2I	2.35 (1.05 to 5.29)	0.038			1.91 (0.97 to 3.76)	0.061		
Insulin	1.17 (0.56 to 2.43)	0.680			1.50 (0.79 to 2.84)	0.218		
Biguanide	0.60 (0.30 to 1.20)	0.148			0.85 (0.45 to 1.58)	0.847		
DPP4i	3.82 (1.51 to 9.70)	0.005	1.58 (0.56 to 4.42)	0.386	1.95 (0.77 to 4.92)	0.159		
GLP1a	0.82 (0.08 to 8.67)	0.871			0.80 (0.15 to 4.39)	0.797		
Dyslipidemia treatment at index time								
Statin	-	-			-	-		
Ezetimibe	2.40 (1.33 to 4.32)	0.004	1.22 (0.56 to 2.65)	0.621	3.65 (2.24 to 5.95)	<0.001	3.02 (1.70 to 5.38)	<0.001
Fenofibrate	1.14 (0.31 to 4.21)	0.845			1.11 (0.34 to 3.66)	0.86		
PCSK9i	-	-			-	-		

OR=odds ratio; AOR=adjusted odds ratio; CI=confidence interval; BMI=body mass index; STE-ACS=ST-elevation acute coronary syndrome; HbA1c=hemoglobin A1c; LDL-C=low-density lipoprotein cholesterol; HDL-C=high-density lipoprotein cholesterol; eGFR=estimated glomerular filtration rate; CKD=chronic kidney disease; TZD=thiazolidinedione; SGLT2I=sodium-glucose cotransporter-2 inhibitor; DPP4i=dipeptidyl peptidase-4 inhibitor; GLP1a=glucagon-like peptide-1 agonist; PCSK9i=proprotein convertase subtilisin kexin type 9 inhibitor

Table 5. Univariate and multivariate analyses to identify potential predictive factors for the inability to achieve target goals for non-HDL-C levels

Variable associated with achieving non-HDL	6-month				12-month			
	Univariate		Multivariate		Univariate		Multivariate	
	OR (95% CI)	p-value	AOR (95% CI)	p-value	OR (95% CI)	p-value	AOR (95% CI)	p-value
Male	0.95 (0.64 to 1.42)	0.809			0.87 (0.59 to 1.29)	0.490		
Age (years)	1.02 (1.01 to 1.04)	0.005	1.01 (0.99 to 1.02)	0.639	1.02 (1.00 to 1.03)	0.031		
BMI (kg/m^2)	0.96 (0.92 to 1.01)	0.078			0.94 (0.90 to 0.98)	0.005	0.97 (0.92 to 1.02)	0.192
Cardio-clinic as primary clinic for dyslipidemia	1.44 (0.87 to 2.41)	0.160			1.20 (0.74 to 1.95)	0.461		
Health coverage								
Universal coverage	1 (Ref.)		1 (Ref.)		1 (Ref.)		1 (Ref.)	
Social security	1.00 (0.43 to 1.93)	0.816	1.03 (0.47 to 2.27)	0.933	1.00 (0.44 to 1.85)	0.785	0.95 (0.45 to 2.01)	0.898
Civil servant benefit	3.69 (2.25 to 6.06)	<0.001	2.48 (1.35 to 4.53)	0.003	4.20 (2.52 to 7.02)	<0.001	3.26 (1.82 to 5.85)	<0.001
Self-funded	1.55 (0.75 to 3.21)	0.236	1.57 (0.70 to 3.48)	0.272	1.71 (0.84 to 3.48)	0.139	1.70 (0.80 to 3.59)	0.167
Smoking Status								
Current smoker	1 (Ref.)				1.11 (0.64 to 1.95)	0.705		
Ex-smoker	2.04 (1.07 to 3.87)	0.030			1.02 (0.67 to 1.56)	0.934		
Non-smoker	1.74 (0.94 to 3.24)	0.079			1 (Ref.)			
Comorbidity								
Hypertension								
History of cerebral infarction	0.67 (0.27 to 1.65)	0.383			0.62 (0.26 to 1.48)	0.281		
Peripheral artery disease								
Chronic kidney disease	0.96 (0.64 to 1.45)	0.856			0.99 (0.67 to 1.47)	0.956		
Diabetes duration to index time (years)	1.01 (0.98 to 1.04)	0.642			1.00 (0.97 to 1.03)	0.931		
STE-ACS	1.04 (0.66 to 1.64)	0.863			1.05 (0.67 to 1.63)	0.845		
Baseline of laboratory result at index time								
HbA1c (%)	0.93 (0.84 to 1.04)	0.206			0.97 (0.86 to 1.08)	0.562		
HbA1c <7%	1.25 (0.80 to 1.95)	0.326			1.21 (0.79 to 1.86)	0.385		
Total cholesterol (mg/dL)	0.99 (0.98 to 0.99)	<0.001	0.99 (0.98 to 1.01)	0.291	0.99 (0.98 to 0.99)	<0.001	0.99 (0.98 to 1.01)	0.334
LDL-C (mg/dL)	0.99 (0.98 to 0.99)	<0.001	1.00 (0.99 to 1.01)	0.542	0.99 (0.98 to 0.99)	<0.001	1.01 (0.99 to 1.01)	0.240
HDL-C (mg/dL)	1.01 (0.99 to 1.02)	0.358			1.01 (0.99 to 1.02)	0.240		
Triglyceride (mg/dL)	0.99 (0.99 to 1.00)	<0.001	1.00 (0.98 to 1.01)	0.213	0.99 (0.99 to 1.00)	<0.001	0.99 (0.99 to 1.00)	0.284
Non-HDL-C (mg/dL)	0.99 (0.98 to 0.99)	<0.001	1.00 (0.98 to 1.01)	0.560	0.99 (0.98 to 0.99)	<0.001	0.99 (0.98 to 1.01)	0.499
eGFR (mL/minute)	1.00 (0.99 to 1.01)	0.624			1.00 (0.99 to 1.01)	0.631		
CKD stage								
G1-G2	1.18 (0.62 to 2.27)	0.616			1.08 (0.58 to 2.03)	0.802		
G3	1.04 (0.49 to 2.20)	0.922			1.00 (0.45 to 1.91)	0.835		
G4	1.82 (0.72 to 4.59)	0.207			1.44 (0.58 to 3.58)	0.433		
G5	1 (Ref.)				1 (Ref.)			
Diabetes treatment at index time								
Sulfonylurea	0.58 (0.30 to 1.11)	0.099			0.52 (0.29 to 0.93)	0.030		
TZD	0.29 (0.08 to 1.14)	0.076			0.99 (0.41 to 2.39)	0.985		
SGLT2I	3.09 (1.44 to 6.62)	0.004	1.90 (0.83 to 4.34)	0.129	1.46 (0.79 to 2.71)	0.229		
Insulin	0.79 (0.43 to 1.46)	0.452			1.04 (0.60 to 1.81)	0.898		
Biguanide	1.12 (0.66 to 1.91)	0.670			1.16 (0.70 to 1.93)	0.569		
DPP4i	4.12 (1.51 to 11.22)	0.006	1.39 (0.50 to 3.84)	0.532	2.56 (1.03 to 6.40)	0.044		
GLP1a	0.79 (0.10 to 6.14)	0.823			1.44 (0.35 to 5.94)	0.615		
Dyslipidemia treatment at index time								
Statin								
Ezetimibe	3.11 (1.82 to 5.30)	<0.001	1.72 (0.88 to 3.36)	0.111	2.28 (1.47 to 3.53)	<0.001	1.58 (0.95 to 2.65)	0.079
Fenofibrate	0.22 (0.05 to 1.02)	0.053			0.50 (0.17 to 1.46)	0.204		
PCSK9i								

OR=odds ratio; AOR=adjusted odds ratio; CI=confidence interval; BMI=body mass index; STE-ACS=ST-elevation acute coronary syndrome; HbA1c=hemoglobin A1c; LDL-C=low-density lipoprotein cholesterol; HDL-C=high-density lipoprotein cholesterol; eGFR=estimated glomerular filtration rate; CKD=chronic kidney disease; TZD=thiazolidinedione; SGLT2I=sodium-glucose cotransporter-2 inhibitor; DPP4i=dipeptidyl peptidase-4 inhibitor; GLP1a=glucagon-like peptide-1 agonist; PCSK9i=proprotein convertase subtilisin kexin type 9 inhibitor

homeostasis, treatment response, and psychological factors[23,24].

ACS patients are considered at high risk for ASCVD, and it is essential to maximize LDL-C reduction in the treatment of those patients. In the present study, only 17.6% and 21.4% of patients achieved the target of more than 50% reduction and LDL-C of less than 55 mg/dL at six and twelve months, respectively, despite most patients receiving high-intensity statin therapy at baseline. These findings are consistent with a study by Jain et al., which observed that among 575 patients with ACS, only 20.87% achieved the intensive LDL-C target of less than 55 mg/dL[25]. Similarly, another retrospective study of ACS patients reported that only 34.6% met the intensive LDL-C target recommended by the 2019 ESC guidelines after three months[26]. In an international study conducted by Buddhari et al., which used a less stringent LDL-C goal of less than 70 mg/dL, only 15.4% of ACS patients treated with lipid-lowering therapy achieved the target[27]. The low LDL-C attainment rate highlights the gap between guideline recommendations and real-world lipid control outcomes, even with the availability of newer lipid-lowering medications in recent years. Several factors may explain the low LDL-C attainment observed in this study. First, some patients may have experienced statin-related side effects, leading to drug discontinuation during treatment. However, this study did not assess the statin persistence rate. Second, only 16.7% of patients received ezetimibe therapy, which is not affordable for most patients unless the civil servant health scheme covers them or can pay out-of-pocket. The authors also found that the civil servant benefit scheme and ezetimibe treatment at index time are the strongly associated factors in reaching the LDL-C target, which was expected for the above-mentioned reasons. Notably, none of the patients were prescribed PCSK9 inhibitors during the time of the study. In addition, physician inertia may have contributed to the failure of many patients to reach the LDL-C target. Thus, the present study results could contribute to plans to encourage more aggressive LDL-C treatment in ACS patients as they are at very high risk for cardiovascular events. Lastly, the authors found that an increase in BMI is associated with the likelihood of failing to reach the LDL-C goal. This could be due to the direct relationship between increasing BMI and raised LDL-C levels in obese patients.

It should be noted that the definition of LDL-C "achievement" in this study was stringent, requiring both a 50% or less of reduction and an LDL-C level of less than 55 mg/dL. While this aligns with current guideline recommendations for very high-risk patients, it may underestimate the proportion of patients who experienced clinically meaningful LDL-C improvements that did not fully meet this strict target. Therefore, some patients with substantial LDL-C reductions may still have derived cardiovascular benefit, even if they were not classified as having "achieved" the target in the study analysis.

Non-HDL cholesterol is a simple, fasting-independent marker that provides a more accurate assessment of atherogenic risk than individual lipoproteins, particularly in individuals with metabolic disorders. In the present study on ACS patients, the authors observed that 37.6% and 44% of participants achieved the non-HDL-C target at six and twelve months, respectively, exceeding the proportion of those who met the LDL-C target. Notably, a higher percentage of patients reached the non-HDL-C target after twelve months of treatment compared to six months. The non-HDL-C attainment rate in the present study is comparable to but higher than, the findings of Al-Sabti et al., which reported only a 27.4% attainment rate in patients with diabetes mellitus and established ASCVD[28]. In addition, the present study result found that the civil servant benefit scheme is the only strong predictive factor to reach the non-HDL target. This could relate to the affordability of various lipid-lowering therapies by these patients.

The prevalence of atherogenic dyslipidemia in the study population was 16.90% at baseline, 12.62% at six months, and 12.14% at twelve months. Comparison with other studies was challenging, as no previous study has specifically examined the prevalence of atherogenic dyslipidemia in ACS patients. However, a cross-sectional observational study by Halcox et al. reported a prevalence of 9.9% in patients with at least one cardiovascular risk factor but without a history of CVD[29]. This suggests that patients with established ASCVD, including ACS patients, have a residual cardiovascular risk characterized by high triglycerides and low HDL-C.

Limitation

The present study has limitations. First, being retrospective in nature, it is subject to confounding factors that should be considered when interpreting the outcomes. Second, the external validity of the results may be limited by the specific population and single-center setting, and since participants

were selected based on available medical records, the findings may not be generalizable to broader populations or those with different demographic or clinical characteristics. Third, the authors did not assess medication changes or patient adherence during the follow-up period, which could have influenced HbA1c, lipid levels, and the achievement of treatment targets. Fourth, inclusion of only patients with complete laboratory data and at least 12 months of follow-up may introduce selection bias and omit individuals with early complications or mortality. Fifth, data collection relied entirely on electronic medical records, which may contain inconsistencies or documentation errors. Sixth, adjustment for potential confounders such as lifestyle factors and socioeconomic status was not possible, and the exclusion of patients with severe comorbidities may further limit generalizability. Despite these limitations, the study underscores the importance of monitoring HbA1c and lipid levels in type 2 diabetes patients after ACS, both at the time of the index event and throughout the follow-up period, highlighting the need for effective management of both diabetes and dyslipidemia. Further studies examining the impact of medication adherence, treatment strategies, and the role of newer therapies in improving lipid control are needed to reduce residual cardiovascular risk.

Conclusion

Most patients achieved the target HbA1c of less than 7%. Despite being at very high risk for cardiovascular events, many patients demonstrated low attainment rates for LDL-C targets. However, a higher proportion of patients were able to reach the target non-HDL-C. These findings highlight the need for more aggressive lipid management, particularly in high-risk ACS patients with diabetes.

What is already known about this topic?

T2DM patients have high recurrent cardiovascular risk after ACS, and while glycemic targets are often achieved, lipid targets, especially LDL-C, remain poorly met in real-world practice. Data from Southeast Asian post-PCI populations are still limited.

What does this study add?

In a Thai post-ACS PCI cohort, HbA1c targets were commonly achieved, but LDL-C and non-HDL-C goals remained low at 6 to 12 months. The study identifies predictors of goal attainment and highlights ongoing gaps in lipid management, emphasizing the need for more intensive lipid-lowering strategies.

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Authors' contribution

TJ drafted and submitted the research proposal, collected and analyzed the data, interpreted the findings, and prepared the manuscript. PD collected and analyzed the data, interpreted the findings, and prepared the manuscript. PS drafted and submitted the research proposal, interpreted the findings, prepared the manuscript, and approved the final version of the manuscript.

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Conflicts of interest

The authors have no conflict of interest.

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