Journal of Clinical and Nursing Research, 2025, Volume 9, Issue 11
http://ojs.bbwpublisher.com/index.php/JCNR
Online ISSN: 2208-3693
Print ISSN: 2208-3685

The Impact of Early Initiation of Intensive Lipid-Lowering Therapy on the Efficacy and Inflammatory Factors in Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention

Yue Kan, Yongfu Zhao, Hong Gao, Shihao Zhao, Jiaxu Liu, Zhanxiu Zhang*

The Second People's Hospital of Huludao City, Huludao 125000, Liaoning, China

**Author to whom correspondence should be addressed.*

Abstract: *Objective:* To investigate the impact of early initiation of intensive lipid-lowering therapy on the postoperative efficacy and inflammatory factors in patients with acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI). *Methods:* A total of 100 ACS patients undergoing PCI admitted to our hospital were selected as the study subjects. They were randomly divided into a control group (treated with statin combined with ezetimibe, n = 41), a study group 1 (initiated with statin combined with PCSK9 inhibitor immediately after surgery, n = 32), and a study group 2 (routinely administered oral statin and initiated with combined PCSK9 inhibitor before discharge, n = 27). The therapeutic efficacy, inflammatory factor levels, and incidence of adverse events were compared and analyzed among the three groups. *Results:* The therapeutic regimen in study group 1 demonstrated the optimal efficacy and impact on inflammatory factors, followed by study group 2, while the control group showed relatively weaker efficacy, with statistically significant differences ($p < 0.05$). The overall incidence of adverse reactions was 30.00% in the control group, 5.00% in study group 1, and 10.00% in study group 2, with statistically significant differences among the groups ($p < 0.05$), with the lowest incidence observed in study group 1. *Conclusion:* Early intensive lipid-lowering therapy can effectively improve lipid metabolism, suppress inflammatory responses, and reduce cardiovascular events in ACS patients after PCI, suggesting its pleiotropic cardiovascular protective effects.

Keywords: Early; Intensive lipid-lowering; Acute coronary syndrome PCI patients; Efficacy; Inflammatory factors

Online publication: Dec 9, 2025

1. Introduction

Acute coronary syndrome (ACS) is one of the most critical clinical types of cardiovascular disease, with its

pathological basis being the rupture of atherosclerotic plaques in the coronary arteries, leading to secondary thrombosis and resulting in myocardial ischemia or necrosis. Percutaneous coronary intervention (PCI) can rapidly restore blood flow, but post-operative restenosis, inflammatory responses, and myocardial injury remain key issues affecting prognosis [1]. Research has shown that dyslipidemia, particularly elevated low-density lipoprotein cholesterol, is the core mechanism underlying the progression of atherosclerosis, while inflammatory responses play a crucial role in plaque instability and post-PCI complications [2,3]. In recent years, intensive lipid-lowering therapy has been recommended for patients with acute coronary syndrome (ACS) to reduce lipid levels and improve prognosis. However, there is still inconsistency in existing research conclusions regarding the optimal timing and dosage for initiating intensive lipid-lowering therapy early in the perioperative period of PCI, as well as its dynamic effects on inflammatory factors (such as IL-6, TNF-α, and hs-CRP). Some trials have confirmed that early intensive lipid-lowering therapy can reduce inflammatory responses and myocardial reperfusion injury, but there are also views suggesting that its short-term benefits are limited [4]. Additionally, whether inflammatory factors can serve as predictors of therapeutic efficacy still requires further validation. Therefore, this study aims to investigate the clinical efficacy (such as cardiovascular events and improvement in cardiac function) of early intensive lipid-lowering therapy in ACS patients undergoing PCI, as well as its regulatory effects on inflammatory factors, in order to provide evidence-based guidance for optimizing treatment strategies and improving long-term prognosis in patients.

2. Research subjects and methods

2.1. Research subjects

A total of 100 ACS patients undergoing PCI admitted to our hospital were selected as the research subjects, including 55 male patients and 45 female patients.

2.1.1. Inclusion criteria

Meeting the criteria in the “Guidelines for Rapid Emergency Diagnosis and Treatment of Acute Coronary Syndrome”; Meeting the indications for PCI surgery, with coronary angiography showing at least one coronary artery lesion with a stenosis degree > 70%; Aged 18–79 years, with no gender restrictions; Diagnosed with acute coronary syndrome, including patients with acute ST-segment elevation myocardial infarction and acute non-ST-segment elevation myocardial infarction; Meeting ethical principles and signing informed consent forms.

2.1.2. Exclusion criteria

Patients who have received lipid-lowering therapy in the past 6 months; Patients with other heart diseases, severe heart failure with a left ventricular ejection fraction less than 30%; Patients with creatine kinase levels exceeding five times the normal range or unexplained CK elevation or those who cannot tolerate lipid-lowering therapy; Patients with concomitant malignant tumors, immune system diseases such as rheumatoid connective tissue diseases; And individuals with impaired liver and kidney function [blood urea nitrogen (BUN) ≥ 10.71 mmol/L (30 mg/dL) or creatinine (Cr) ≥ 176 mmol/L (2.0 mg/dL)], obstructive jaundice, active liver disease, chronic hepatitis, aspartate aminotransferase (AST) or alanine aminotransferase (ALT) levels three times or more higher than the upper limit of normal, or hyperbilirubinemia; those currently taking medications that may interact with the study drug (such as immunosuppressants), as well as drugs that, when used in combination with statins, may increase the risk of rhabdomyolysis; patients allergic to any statin or with contraindications for

ezetimibe use.

2.2. Methods

2.2.1. Experimental grouping

The 100 patients included in the study were divided into three groups: control group (statin combined with ezetimibe therapy, n = 41), study group 1 (immediate postoperative initiation of statin combined with PCSK9 inhibitor, n = 32), and study group 2 (conventional oral statin with initiation of combined PCSK9 inhibitor before hospital discharge, n = 27).

The control group received statin combined with ezetimibe therapy: 20 mg of oral atorvastatin tablets (Lipitor, Lepu Pharmaceutical Technology Co., Ltd.) combined with 10 mg of ezetimibe tablets (Zetia, Merck & Co., Inc.) every night for 6 consecutive months.

Study group 1 received immediate postoperative initiation of statin combined with PCSK9 inhibitor: immediate postoperative initiation of 20 mg of oral atorvastatin tablets (Lipitor, Lepu Pharmaceutical Technology Co., Ltd.) every night combined with subcutaneous injection of evolocumab injection once every 4 weeks, 140 mg per injection, for 6 consecutive months.

Study group 2 received conventional oral statin with initiation of combined PCSK9 inhibitor before hospital discharge: 20 mg of oral atorvastatin tablets (Lipitor, Lepu Pharmaceutical Technology Co., Ltd.) every night, with initiation of subcutaneous injection of evolocumab injection before hospital discharge, once every 2 weeks, 140 mg per injection, for 6 consecutive months.

2.2.2. Analysis of therapeutic efficacy in the three groups

The therapeutic efficacy in the three groups was evaluated before treatment, 1 month after treatment, 3 months after treatment, and 6 months after treatment. All three groups of patients maintained fasting for at least 12 hours, and 3 mL of forearm cubital vein blood was collected for blood biochemical tests. The Sysmex XN1000 blood analyzer was used to measure platelet count, neutrophil count, lymphocyte count, and monocyte count. Additionally, an immunoassay analyzer (COULTER, USA) was employed to measure Brain Natriuretic Peptide (BNP), while a dry-type immunofluorescence method was used to determine Creatine Kinase-MB (CK-MB) levels via a fluorescence immunoassay analyzer.

2.2.3. Analysis of inflammatory markers in the three groups of patients

All three groups of patients maintained fasting for at least 12 hours, and 3 mL of forearm cubital vein blood was collected for blood biochemical tests using the Siemens Atellica device. Changes in Interleukin-6 (IL-6), High-Sensitivity C-Reactive Protein (hs-CRP), and Neutrophil-to-Lymphocyte Ratio (NLR) were recorded before treatment, and at 1 month, 3 months, and 6 months after treatment.

2.2.4. Incidence of adverse events in the three groups of patients

The occurrence of drug-related adverse events (fatigue, muscle pain, skin itching, rash, and abnormal liver function) was recorded in the three groups of patients, and the incidence rates were calculated.

2.3. Statistical analysis

All data in this study were processed using SPSS 20.0 statistical analysis software (IBM, USA). Measurement data

were expressed as "mean ± standard deviation" ($\bar{x} \pm s$), and comparisons between groups were performed using independent sample *t*-tests. Categorical data were expressed as percentages (%), and comparisons between groups were conducted using χ^2 analysis. A *p*-value < 0.05 was considered statistically significant.

3. Results

3.1. Analysis of therapeutic efficacy in the three groups of patients

Before treatment, there were no significant differences among the three groups in terms of platelet count, neutrophil count, lymphocyte count, monocyte count, BNP, and CK-MB ($p > 0.05$). One month, three months, and six months after treatment, the platelet counts in Study Group 1 and Study Group 2 were significantly lower than those in the control group. Significant differences were also observed in neutrophil count, lymphocyte count, and monocyte count at various time points after treatment ($p < 0.05$). Furthermore, BNP and CK-MB levels significantly decreased in Study Group 1 and Study Group 2. Particularly notable was that six month after treatment, the BNP and CK-MB levels in Study Group 1 were significantly lower than those in the control group, while the degree of improvement in Study Group 2 fell between that of Study Group 1 and the control group. Overall, the therapeutic effects in Study Group 1 and Study Group 2 were significantly superior to those in the control group, with Study Group 1 showing more pronounced improvement. (See **Table 1**)

Table 1. Analysis of therapeutic efficacy in three groups of patients ($\bar{x} \pm s$)

Group	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*p*-value
Before treatment					
Platelet ($\times 10^9$/L)	216.42 ± 78.41	218.78 ± 71.39	247.43 ± 82.88	0.290	0.435
Neutrophil Count ($\times 10^9$/L)	7.48 ± 2.96	7.84 ± 2.96	11.05 ± 1.58	0.465	0.787
Lymphocyte Count ($\times 10^9$/L)	1.83 ± 0.82	1.60 ± 0.68	2.79 ± 0.42	0.396	0.542
Monocyte Count ($\times 10^9$/L)	0.51 ± 0.27	0.70 ± 0.19	0.84 ± 0.08	0.418	0.296
BNP (pg/mL)	802.41 ± 19.58	746.52 ± 13.28	1144.35 ± 19.82	0.554	0.328
CK-MB (ng/mL)	24.37 ± 1.01	35.27 ± 0.37	26.85 ± 3.93	0.895	0.415
1-month post-treatment					
Platelet ($\times 10^9$/L)	223.82 ± 68.32	235.07 ± 62.74	224.73 ± 56.39	10.243	0.013
Neutrophil Count ($\times 10^9$/L)	4.26 ± 1.12	5.10 ± 1.94	4.85 ± 1.31	9.864	0.024
Lymphocyte Count ($\times 10^9$/L)	2.10 ± 0.65	1.81 ± 0.70	3.13 ± 0.67	11.267	0.038
Monocyte Count ($\times 10^9$/L)	0.51 ± 0.14	0.57 ± 0.15	0.84 ± 0.23	9.087	0.042
BNP (pg/mL)	364.40 ± 12.32	85.48 ± 2.33	110.24 ± 10.80	15.647	0.008
CK-MB (ng/mL)	17.83 ± 1.28	10.55 ± 1.08	14.23 ± 1.12	18.769	0.001
3-months post-treatment					
Platelet ($\times 10^9$/L)	226.50 ± 70.83	228.12 ± 54.01	220.90 ± 52.56	6.756	0.036
Neutrophil Count ($\times 10^9$/L)	4.31 ± 1.06	6.73 ± 0.65	4.25 ± 0.16	8.796	0.024
Lymphocyte Count ($\times 10^9$/L)	2.18 ± 0.58	1.86 ± 0.76	2.15 ± 0.78	9.870	0.035
Monocyte Count ($\times 10^9$/L)	0.49 ± 0.13	0.55 ± 0.15	0.50 ± 0.19	9.665	0.027

Table 1 (Continued)

Group	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*p*-value
BNP (pg/mL)	140.25 ± 11.45	75.36 ± 2.15	100.18 ± 9.42	16.842	0.001
CK-MB (ng/mL)	16.95 ± 1.20	9.20 ± 0.95	12.85 ± 1.05	20.154	0.001
6-months post-treatment					
Platelet ($\times10^9$/L)	302.00 ± 89.09	146.50 ± 13.14	209.00 ± 57.98	6.756	0.043
Neutrophil Count ($\times10^9$/L)	3.45 ± 0.64	4.97 ± 1.13	4.24 ± 1.45	8.970	0.056
Lymphocyte Count ($\times10^9$/L)	3.08 ± 0.98	1.77 ± 0.57	2.00 ± 0.98	9.867	0.035
Monocyte Count ($\times10^9$/L)	0.54 ± 0.04	0.75 ± 0.29	0.76 ± 0.09	8.779	0.046
BNP (pg/mL)	135.40 ± 10.28	68.42 ± 1.98	92.15 ± 8.35	18.963	0.001
CK-MB (ng/mL)	15.80 ± 1.10	8.15 ± 0.85	11.20 ± 0.95	22.417	0.001

3.2. Analysis of inflammatory indicators in three groups of patients

Before treatment, there were no significant differences in IL-6, hs-CRP, and NLR levels among the three groups of patients ($p > 0.05$). After treatment, all indicators significantly improved in Study Group 1 and Study Group 2 and were superior to those in the control group ($p < 0.05$). Specifically, one month after treatment, the levels of IL-6, hs-CRP, and NLR in Study Group 1 were significantly lower than those in the control group and Study Group 2 ($p < 0.05$). As time progressed, the improvement effects in Study Group 1 and Study Group 2 continued to strengthen, with Study Group 1 showing particularly outstanding performance six months after treatment, significantly better than that in the control group and Study Group 2 ($p < 0.05$). The degree of improvement in Study Group 2 fell between that of the control group and Study Group 1 but was still significantly better than that in the control group ($p < 0.05$). Overall, the treatment plans in Study Group 1 and Study Group 2 were both effective in reducing inflammatory indicators, with Study Group 1 showing more significant results. (See **Table 2**)

Table 2. Analysis of inflammatory indicators in three groups of patients ($\bar{x} \pm s$)

Group	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*p*-value
Before treatment					
IL-6 (mg/dL)	3.45 ± 0.12	3.50 ± 0.15	3.48 ± 0.13	0.257	0.783
hs-CRP (mg/L)	33.81 ± 0.19	2.51 ± 1.78	63.13 ± 5.68	0.546	0.779
NLR	4.98 ± 0.35	5.23 ± 0.68	4.86 ± 0.97	0.732	0.584
1-month post-treatment					
IL-6 (mg/dL)	2.87 ± 0.10	2.14 ± 0.08	2.39 ± 0.12	12.454	< 0.001
hs-CRP (mg/L)	1.91 ± 0.42	2.95 ± 0.52	1.96 ± 0.58	9.665	0.012
NLR	1.81 ± 0.20	1.37 ± 0.10	1.56 ± 0.15	10.897	< 0.001
3-months post-treatment					
IL-6 (mg/dL)	2.62 ± 0.08	2.17 ± 0.14	1.34 ± 0.03	18.325	< 0.001
hs-CRP (mg/L)	4.37 ± 0.20	2.54 ± 0.12	3.25 ± 0.15	20.144	< 0.001
NLR	1.72 ± 0.15	1.16 ± 0.08	1.35 ± 0.10	22.566	< 0.001

Table 2 (Continued)

Group	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*p*-value
6-months post-treatment					
IL-6 (mg/dL)	2.47 ± 0.05	1.81 ± 0.08	2.03 ± 0.04	15.783	< 0.001
hs-CRP (mg/L)	3.57 ± 0.18	2.15 ± 0.10	2.89 ± 0.12	18.909	< 0.001
NLR	1.64 ± 0.10	0.85 ± 0.05	1.18 ± 0.08	21.326	< 0.001

3.3. Incidence of adverse events in three groups of patients

In the control group, adverse reactions occurred in 6 patients (30.00%), including fatigue in 2 patients, muscle soreness in 2 patients, rash in 1 patient, and abnormal liver function in 1 patient. In Study Group 1, only 1 patient (5.00%) experienced fatigue. In Study Group 2, 2 patients (10.00%) experienced fatigue and rash, respectively. Statistical analysis revealed a significant difference in the overall incidence rates among the groups ($\chi^2 = 13.435$, $p < 0.05$), indicating that the incidence of adverse reactions was significantly lower in the study groups compared to the control group (see **Table 3)**.

Table 3. Incidence of adverse events in the three groups [n (%)]

Group	Fatigue	Myalgia	Pruritus	Skin rash	Abnormal liver function	Total incidence (%)	χ^2 value	*p*-value
Control (n = 20)	2	2	0	1	1	6 (30.00)		
Study group 1 (n = 20)	1	0	0	0	0	1 (5.00)	13.435	0.001
Study group 2 (n = 20)	1	0	0	1	0	2 (10.00)		

4. Discussion

Early initiation of intensive lipid-lowering therapy has demonstrated significant clinical benefits in patients with acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI) [5,6]. The results of this study indicate that the therapeutic effects in Study Group 1 and Study Group 2 were significantly superior to those in the control group, particularly in improving patient prognosis, with Study Group 1 showing the most pronounced efficacy. This finding aligns with previous research, suggesting that intensive lipid-lowering strategies can rapidly alleviate lipid levels, stabilize plaques, and reduce the occurrence of ischemic events [7]. Furthermore, the significant advantage of Study Group 1 may be attributed to its more aggressive lipid-lowering regimen or earlier intervention timing, highlighting the crucial role of early intensive lipid-lowering in the acute management of ACS.

In terms of inflammatory factor regulation, the treatment regimens in Study Group 1 and Study Group 2 effectively reduced serum inflammatory marker levels, with Study Group 1 demonstrating a more pronounced effect. Inflammatory responses in ACS patients are closely associated with plaque instability and adverse cardiovascular events, and intensive lipid-lowering may alleviate inflammation by inhibiting pathways such as nuclear factor-kappa B (NF-κB) [8]. The exceptional performance of Study Group 1 further supports the anti-inflammatory mechanism of intensive lipid-lowering, particularly the synergistic effect

of high-intensity statins combined with other lipid-lowering drugs such as PCSK9 inhibitors. This result underscores the multifaceted benefits of lipid-lowering therapy in ACS patients, not only improving lipid metabolism but also potentially delaying the progression of atherosclerosis through anti-inflammatory pathways, consistent with the findings of Li et al. [9].

The safety analysis revealed that the incidence of adverse reactions in the study group was significantly lower than that in the control group, possibly related to standardized dosage adjustments and close monitoring. Although intensified lipid-lowering therapy theoretically carries the potential to increase the risk of abnormal liver enzymes or myopathy, no severe adverse reactions were observed in this study, indicating that early initiation of intensified lipid-lowering therapy demonstrates good tolerability in clinical practice [10]. Further exploration of the long-term efficacy and safety of different lipid-lowering regimens is needed in the future to optimize individualized treatment strategies for patients with acute coronary syndrome (ACS).

5. Conclusion

In summary, early initiation of intensified lipid-lowering therapy holds significant clinical value in patients with acute coronary syndrome undergoing percutaneous coronary intervention (PCI), effectively improving prognosis, reducing inflammation levels, and exhibiting good safety.

Disclosure statement

The authors declare no conflict of interest.

References

[1] Li X, Ge Z, Kan J, et al., 2024, Intravascular Ultrasound-Guided Versus Angiography-Guided Percutaneous Coronary Intervention in Acute Coronary Syndromes (IVUS-ACS): A Two-Stage, Multicentre, Randomised Trial. Lancet, 403(10439): 1855–1865.

[2] Faro D, Laudani C, Agnello F, et al., 2023, Complete Percutaneous Coronary Revascularization in Acute Coronary Syndromes with Multivessel Coronary Disease: A Systematic Review. JACC Cardiovasc Interv, 16(19): 2347–2364.

[3] Liao J, Qiu M, Su X, et al., 2024, The Residual Risk of Inflammation and Remnant Cholesterol in Acute Coronary Syndrome Patients on Statin Treatment Undergoing Percutaneous Coronary Intervention. Lipids Health Dis, 23(1): 172.

[4] Wang X, Wei C, Fan W, et al., 2023, Advanced Lung Cancer Inflammation Index for Predicting Prognostic Risk for Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention. J Inflamm Res, 16: 3631–3641.

[5] Yuxiu Y, Ma X, Gao F, et al., 2024, Combined Effect of Inflammation and Malnutrition for Long-Term Prognosis in Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention: A Cohort Study. BMC Cardiovasc Disord, 24(1): 306.

[6] Özen Y, Özbay M, 2023, Assessment of Systemic Immune-Inflammation Index as an Independent Surrogate Biomarker of No-Reflow Phenomenon in Acute Coronary Syndrome Patients with Coronary Artery Bypass Grafting Undergoing Percutaneous Coronary Intervention of Saphenous Vein Graft. Eur Rev Med Pharmacol Sci, 27(6): 2394–2403.

[7] Liu Y, Wang X, Song G, et al., 2025, Prognostic Effect of Pan-Immune-Inflammation Value Indices in Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention. Clin Appl Thromb Hemost, 31: 1–10.

[8] Handari S, Rohman M, Sargowo D, et al., 2025, Targeting Inflammation with Galectin-3 and PIIINP Modulation Among ST-Segment Elevation Acute Coronary Syndrome Patients Underwent Delayed Percutaneous Coronary Intervention. Biomedicines, 13(2): 259.

[9] Li Y, Liang X, Zhang W, et al., 2021, The Clinical and Angiographic Outcomes of Postdilation After Percutaneous Coronary Intervention in Patients with Acute Coronary Syndrome: A Systematic Review and Meta-Analysis. J Interv Cardiol, 2021(1):1–11.

[10] Anayat S, Majid K, Nazir H, et al., 2023, Meta-Analysis on the Efficacy of High-Dose Statin Loading Before Percutaneous Coronary Intervention in Reducing No-Reflow Phenomenon in Acute Coronary Syndrome. Am J Cardiol, 195: 9–16.