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The Impact of Early Initiation of Intensive Lipid-Lowering Therapy on Lipid Target Achievement and Major Adverse Cardiovascular Events in Patients with Acute Coronary Syndrome Undergoing Percutaneous Coronary Intervention

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Abstract: *Objective*: This study primarily investigates the impact of early initiation of intensive lipid-lowering therapy on lipid target achievement rates and the incidence of major adverse cardiovascular events (MACE) in patients with acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI). *Methods*: A total of 100 patients with ACS who underwent PCI in our hospital were selected as the study subjects. They were randomly divided into a control group (treated with statin combined with ezetimibe, n = 41), study group 1 (initiated with statin combined with a PCSK9 inhibitor immediately after surgery, n = 32), and study group 2 (received routine oral statin and initiated with a combined PCSK9 inhibitor before discharge, n = 27). The safety of treatment, lipid target achievement, and differences in the incidence of cardiovascular adverse events were compared and analyzed among the three groups. *Results*: The treatment regimen in study group 1 demonstrated the optimal effect in improving liver and kidney function and lipid indicators, followed by study group 2, while the control group showed relatively weaker efficacy, with statistically significant differences ($P < 0.05$). The overall incidence of cardiovascular adverse events was 25.00% in the control group, 5.00% in study group 1, and 15.00% in study group 2, with statistically significant differences between the groups ($P < 0.05$), with study group 1 having the lowest incidence. *Conclusion*: Early intensive lipid-lowering therapy can significantly improve lipid target achievement rates and reduce the risk of MACE in patients with ACS undergoing PCI, with good safety and significant clinical implications.

Keywords: Early; Intensive lipid-lowering; Patients with acute coronary syndrome undergoing PCI; Lipids; Major adverse cardiovascular events

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1. Introduction

Acute coronary syndrome (ACS), one of the most critical clinical types among cardiovascular diseases, has a pathogenesis closely related to atherosclerotic plaque rupture and thrombosis, with dyslipidemia serving as the core driver of atherosclerosis progression [1]. Although percutaneous coronary intervention (PCI) can rapidly restore blood flow, residual postoperative inflammatory responses, endothelial dysfunction, and plaque instability may still lead to major adverse cardiac events (MACE), including reinfarction, revascularization, stroke, and even cardiovascular death [2]. In recent years, evidence from evidence-based medicine has indicated that intensive lipid-lowering therapy can delay the progression of atherosclerosis, stabilize plaques, and improve endothelial function by significantly reducing lipid levels, thereby reducing the risk of MACE [3,4]. However, there is still controversy regarding the optimal timing, intensity, and long-term prognostic impact of early initiation of intensive lipid-lowering therapy (such as the use of high-intensity statins combined with PCSK9 inhibitors within 24 hours) in patients with ACS after PCI.

Some studies suggest that early intensive intervention can more rapidly achieve lipid targets (e.g., LDL-C < 1.4 mmol/L or a ≥ 50% reduction from baseline) and reduce the release of inflammatory factors. However, others argue that overly aggressive lipid-lowering may increase the risk of liver enzyme abnormalities or myopathy, and the benefit-risk ratio in different populations (e.g., elderly individuals, those with comorbid diabetes, or chronic kidney disease) requires further stratified evaluation [5]. Additionally, current guidelines on lipid-lowering strategies during the perioperative period of PCI are still based on the overall ACS population and lack individualized approaches tailored to the dynamic changes in lipid metabolism under surgical stress [6].

Therefore, exploring the short-term and long-term effects of early intensive lipid-lowering therapy on lipid target attainment rates, treatment safety, and MACE events in patients with ACS undergoing PCI will not only help clarify the optimal treatment time window and drug combinations but also provide evidence-based support for developing precise lipid-lowering strategies, ultimately improving clinical outcomes in this high-risk population. This study aims to systematically evaluate the efficacy and safety of early intensive lipid-lowering therapy through prospective cohort analysis to fill the evidence gap between current guidelines and clinical practice.

2. Research subjects and methods

2.1. Research subjects

A total of 100 patients with ACS who underwent PCI at our hospital were selected as research subjects, including 55 male and 45 female patients.

Inclusion criteria are as follows:

(1) Meeting the criteria outlined in the “Guidelines for Rapid Emergency Diagnosis and Treatment of Acute Coronary Syndrome”;

(2) Meeting the indications for PCI surgery, with coronary angiography revealing at least one coronary artery lesion with a stenosis degree > 70%;

(3) Aged between 18 and 79 years, regardless of gender;

(4) Diagnosed with acute coronary syndrome, including patients with acute ST-segment elevation myocardial infarction and acute non-ST-segment elevation myocardial infarction;

(5) Adhering to ethical principles and having signed an informed consent form.

Exclusion criteria are as follows:

(1) Patients who have received lipid-lowering therapy in the past 6 months;

(2) Those with other heart diseases or severe cardiac dysfunction with a left ventricular ejection fraction less than 30%;
(3) Patients with creatine kinase levels exceeding five times the normal range or unexplained CK elevation or those who cannot tolerate lipid-lowering therapy;
(4) Patients with concurrent malignant tumors, immune system diseases such as rheumatoid connective tissue disorders;
(5) Those 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 ALT levels three times or more the upper limit of normal, or hyperbilirubinemia;
(6) Patients currently taking medications that may interact with the study drugs (such as immunosuppressants), as well as medications that, when combined with statins, may increase the risk of rhabdomyolysis;
(7) Patients allergic to any statin or those with contraindications to 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 treatment, n = 41), study group 1 (immediate initiation of statin combined with PCSK9 inhibitor post-surgery, n = 32), and study group 2 (routine oral statin with initiation of combined PCSK9 inhibitor before hospital discharge, n = 27).

The control group was treated with a combination of statins and ezetimibe: oral administration of 20 mg atorvastatin tablets (Lipitor, Lepu Pharmaceutical Technology Co., Ltd.) every night, combined with 10 mg ezetimibe tablets (Ezetrol, Merck & Co., Inc.), for a continuous treatment period of 6 months.

Group 1 initiated statin therapy combined with a PCSK9 inhibitor immediately after surgery: oral administration of 20 mg atorvastatin tablets (Lipitor, Lepu Pharmaceutical Technology Co., Ltd.) every night, combined with subcutaneous injection of evolocumab injection immediately after surgery, administered every 2 weeks at a dose of 140 mg per injection, for a continuous treatment period of 6 months.

Group 2 received conventional oral statin therapy and initiated combination therapy with a PCSK9 inhibitor before discharge: oral administration of 20 mg atorvastatin tablets (Lipitor, Lepu Pharmaceutical Technology Co., Ltd.) every night, with subcutaneous injection of evolocumab injection initiated before discharge, administered every 2 weeks at a dose of 140 mg per injection, for a continuous treatment period of 6 months.

2.2.2. Safety assessment analysis of treatment in the three groups of patients

The safety of treatment in the three groups of patients was assessed before treatment, as well as 1 month, 3 months, and 6 months after treatment. This included indicators of liver damage: Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Total bilirubin (TBIL), Direct bilirubin (DBIL), and Indirect bilirubin (IBIL); and indicators of kidney damage: Urea nitrogen (BUN), Creatinine (CR), Uric acid (UA), and Creatine kinase isozyme (CK-MB). Among these, CK-MB was measured using a dry-type immunofluorescence method with a fluorescence immunoassay analyzer.

2.2.3. Analysis of blood lipid levels reaching standard in the three groups of patients

All three groups of patients maintained a fasting state for at least 12 hours, and 3 mL of forearm cubital vein blood

was collected for blood biochemical testing using a Siemens Atellica device, record the changes in Apolipoprotein A (ApoA), Total Cholesterol (TC), Triglyceride (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), Lipoprotein(a) [Lp(a)], and Apolipoprotein B (ApoB) before treatment, and at 1 month, 3 months, and 6 months after treatment.

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

During the follow-up period, the occurrence of cardiovascular adverse events (acute myocardial infarction, heart failure, death, and rehospitalization) was documented for all three patient groups, and incidence rates were determined.

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" (mean ± SD), and comparisons between groups were performed using independent sample t-tests. Enumeration data were expressed as percentages (%), and comparisons between groups were performed using χ^2 analysis. A P-value < 0.05 indicated a statistically significant difference.

3. Results

3.1. Analysis of therapeutic efficacy in three groups of patients

There were no significant differences in liver and kidney damage indicators (ALT, AST, TBIL, DBIL, IBIL, BUN, CR, UA, CK-MB) among the three groups of patients before treatment ($P > 0.05$). At 1 month, 3 months, and 6 months after treatment, all indicators significantly improved, with significant differences between groups ($P < 0.05$). Among them, study group 1 showed the greatest improvement in liver and kidney indicators. The therapeutic efficacy of study group 2 was between that of the control group and study group 1, but some indicators (such as TBIL, DBIL, BUN, CR) were still significantly better than those in the control group ($P < 0.05$). That is, the treatment regimen of study group 1 demonstrated the most optimal effect in improving liver and kidney function and myocardial injury, followed by study group 2, while the control group showed relatively weaker efficacy. (**Table 1**)

Table 1. Analysis of therapeutic efficacy in three patient groups (mean ± SD)

Grouping	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*P* value
Before treatment					
Liver damage indicators					
ALT (U/L)	40.88 ± 3.09	40.40 ± 3.56	42.75 ± 1.46	0.469	0.789
AST (U/L)	144.75 ± 15.62	27.60 ± 5.41	63.50 ± 3.07	2.642	0.769
TBIL (μmol/L)	20.60 ± 1.25	12.33 ± 3.58	19.07 ± 1.08	0.859	0.436
DBIL (μmol/L)	6.71 ± 0.22	3.60 ± 0.61	6.55 ± 0.76	0.483	0.126
IBIL (μmol/L)	14.12 ± 1.06	8.62 ± 0.38	12.27 ± 0.23	0.268	0.700
Kidney damage indicators					
BUN (mg/dL)	52.98 ± 1.12	52.57 ± 1.78	52.21 ± 3.92	0.119	0.257
CR (mg/dL)	70.99 ± 15.23	67.30 ± 2.71	59.60 ± 8.87	2.650	0.160
UA (mg/dL)	313.25 ± 52.93	337.60 ± 16.36	361.50 ± 16.54	1.593	0.226
CK-MB (ng/mL)	24.37 ± 1.01	35.27 ± 0.37	26.85 ± 3.93	0.894	0.390

Table 1 (Continued)

Grouping	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*P* value
1 month post-treatment					
Liver damage indicators					
ALT (U/L)	32.01 ± 2.51	29.67 ± 0.21	33.62 ± 1.49	8.047	0.001
AST (U/L)	26.47 ± 1.21	23.19 ± 1.19	24.10 ± 1.02	3.283	0.002
TBIL (μmol/L)	12.81 ± 5.01	15.53 ± 5.15	13.01 ± 5.04	2.115	0.042
DBIL (μmol/L)	13.58 ± 4.74	6.28 ± 2.26	5.19 ± 1.90	3.786	0.047
IBIL (μmol/L)	8.01 ± 3.24	9.22 ± 3.32	7.82 ± 3.35	5.482	0.026
Kidney damage indicators					
BUN (mg/dL)	38.91 ± 0.37	26.53 ± 0.27	33.45 ± 9.98	13.267	0.007
CR (μmol/L)	89.21 ± 10.99	78.56 ± 18.39	71.05 ± 12.03	4.528	0.014
UA (μmol/L)	333.91 ± 15.24	352.22 ± 16.55	321.43 ± 17.70	5.097	0.025
CK-MB (ng/mL)	12.68 ± 1.32	10.49 ± 1.90	11.46 ± 6.52	10.434	0.001
3 months post-treatment					
Liver damage indicators					
ALT (U/L)	42.80 ± 2.67	29.25 ± 1.03	47.44 ± 0.65	5.212	0.010
AST (U/L)	39.75 ± 3.80	24.13 ± 0.16	24.78 ± 0.76	7.653	0.033
TBIL (μmol/L)	14.63 ± 6.44	14.80 ± 0.49	13.28 ± 0.57	4.035	0.015
DBIL (μmol/L)	5.47 ± 2.46	6.23 ± 0.17	4.87 ± 0.75	5.278	0.044
IBIL (μmol/L)	8.99 ± 4.01	9.19 ± 0.58	8.40 ± 0.17	3.197	0.013
Kidney damage indicators					
BUN (mg/dL)	34.27 ± 0.32	22.15 ± 0.21	28.91 ± 8.12	12.845	0.001
CR (μmol/L)	69.76 ± 2.17	71.89 ± 5.77	67.60 ± 9.64	7.649	0.053
UA (μmol/L)	302.13 ± 19.61	337.00 ± 15.52	341.78 ± 10.89	5.611	0.014
CK-MB (ng/mL)	11.34 ± 1.18	8.12 ± 1.45	9.21 ± 5.87	12.089	0.020
6 months post-treatment					
Liver damage indicators					
ALT (U/L)	18.50 ± 0.61	14.33 ± 4.04	13.00 ± 2.46	4.442	0.015
AST (U/L)	18.00 ± 2.83	19.67 ± 0.66	26.50 ± 0.71	5.647	0.023
TBIL (μmol/L)	12.74 ± 1.43	18.43 ± 0.12	9.64 ± 3.23	3.228	0.019
DBIL (μmol/L)	4.42 ± 0.44	5.86 ± 1.49	1.95 ± 0.62	5.467	0.042
IBIL (μmol/L)	8.14 ± 1.24	12.57 ± 0.87	6.19 ± 1.73	6.758	0.003
Kidney damage indicators					
BUN (mg/dL)	30.12 ± 0.28	18.92 ± 0.18	25.34 ± 6.87	14.672	0.003
CR (μmol/L)	71.50 ± 6.58	75.30 ± 19.43	121.45 ± 13.09	4.635	0.012
UA (μmol/L)	392.50 ± 14.85	328.67 ± 17.23	486.00 ± 12.62	6.758	0.025
CK-MB (ng/mL)	9.34 ± 1.18	7.12 ± 1.45	8.21 ± 5.87	9.879	0.023

3.2. Analysis of lipid profile control in three patient groups

There were no significant differences in the various indicators (including ApoA, TC, TG, HDL-C, LDL-C, Lp(a), and ApoB) among the three patient groups before treatment ($P > 0.05$). After one month, three months, and six months of treatment, the indicators in study group 1 and study group 2 showed significant improvements compared to the control group ($P < 0.05$), with particularly notable improvements in study group 1 ($P < 0.05$). (**Table 2**)

Table 2. Analysis of lipid profile control in three patient groups (mean ± SD)

Grouping	Control group (n = 41)	Study group 1 (n = 32)	Study group 2 (n = 27)	t/χ^2 value	*P* value
Before treatment					
ApoA (mg/dL)	1.21 ± 0.15	1.22 ± 0.18	1.21 ± 0.16	0.128	0.887
TC (mmol/L)	4.92 ± 1.26	5.68 ± 1.21	5.76 ± 1.44	0.176	0.543
TG (mmol/L)	1.39 ± 0.82	1.93 ± 0.87	1.31 ± 0.87	0.267	0.843
HDL-C (mmol/L)	1.39 ± 0.80	1.54 ± 0.62	0.93 ± 0.21	0.609	0.415
LDL-C (mmol/L)	2.99 ± 1.03	3.54 ± 1.65	4.21 ± 1.13	0.743	0.516
Lp(a) (nmol/L)	25.89 ± 1.43	26.45 ± 1.98	25.58 ± 1.45	0.115	0.896
ApoB (mg/dL)	0.94 ± 0.12	0.92 ± 0.15	0.91 ± 0.11	0.125	0.487
1 month post-treatment					
ApoA (mg/dL)	1.25 ± 0.16	1.45 ± 0.20	1.30 ± 0.18	8.256	0.003
TC (mmol/L)	3.21 ± 0.87	2.74 ± 0.74	2.93 ± 0.34	4.389	0.054
TG (mmol/L)	1.77 ± 0.98	1.73 ± 0.93	1.70 ± 0.76	6.587	0.023
HDL-C (mmol/L)	1.10 ± 0.22	1.23 ± 0.14	1.05 ± 0.26	7.869	0.044
LDL-C (mmol/L)	1.93 ± 0.47	1.56 ± 0.64	1.69 ± 0.38	8.970	0.025
Lp(a) (nmol/L)	24.12 ± 1.58	20.05 ± 1.06	22.38 ± 1.24	12.371	0.014
ApoB (mg/dL)	0.85 ± 0.09	0.75 ± 0.08	0.80 ± 0.09	11.282	0.001
3 months post-treatment					
ApoA (mg/dL)	1.28 ± 0.17	1.52 ± 0.21	1.35 ± 0.19	9.142	0.001
TC (mmol/L)	3.15 ± 0.57	3.23 ± 1.21	3.13 ± 0.22	6.753	0.014
TG (mmol/L)	1.80 ± 0.97	2.05 ± 0.38	1.95 ± 0.89	5.473	0.025
HDL-C (mmol/L)	1.10 ± 0.17	1.10 ± 0.26	1.14 ± 0.28	6.776	0.047
LDL-C (mmol/L)	1.79 ± 0.33	1.74 ± 0.81	1.77 ± 0.92	9.034	0.011
Lp(a) (nmol/L)	22.45 ± 1.06	18.20 ± 0.87	20.15 ± 2.65	14.825	0.011
ApoB (mg/dL)	0.82 ± 0.08	0.68 ± 0.07	0.75 ± 0.08	16.333	0.008
6 months post-treatment					
ApoA (mg/dL)	1.30 ± 0.18	1.58 ± 0.22	1.40 ± 0.20	10.257	0.005
TC (mmol/L)	3.33 ± 0.23	3.88 ± 1.24	5.06 ± 0.04	12.434	0.034
TG (mmol/L)	2.20 ± 0.41	1.89 ± 0.75	3.15 ± 0.12	9.807	0.011
HDL-C (mmol/L)	1.11 ± 0.11	1.20 ± 0.17	0.87 ± 0.02	7.658	0.024
LDL-C (mmol/L)	2.06 ± 0.04	2.19 ± 0.44	3.30 ± 0.50	12.312	0.035
Lp(a) (nmol/L)	20.80 ± 2.13	16.50 ± 0.98	18.20 ± 0.78	18.326	0.012
ApoB (mg/dL)	0.78 ± 0.07	0.62 ± 0.06	0.70 ± 0.07	21.548	0.013

3.3. Incidence of cardiovascular adverse events in three patient groups

The overall incidence of cardiovascular adverse events was 25.00% in the control group, 5.00% in study group 1, and 15.00% in study group 2, with statistically significant differences among the groups ($P < 0.05$), and the lowest

incidence observed in study group 1. (**Table 3**)

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

Events	Control group (n = 20)	Study group 1 (n = 20)	Study group 2 (n = 20)
Acute myocardial infarction	1	0	1
Heart failure	1	0	1
Death	0	0	0
Rehospitalization	1	0	0
Total incidence (%)	5 (25.00)	1 (5.00)	3 (15.00)
t/χ^2 value		14.392	
P value		0.001	

4. Discussion

ACS is a critical type of cardiovascular disease, with PCI being the primary means of revascularization. However, even after successful PCI, patients still face a high risk of MACE, and lipid management is one of the core strategies for secondary prevention of ACS [7]. This study explored the impact of early initiation of intensive lipid-lowering therapy on the lipid profile control rate and MACE in patients with ACS undergoing PCI.

This study found that an intensive lipid-lowering regimen initiated early with PCSK9 inhibitors demonstrated optimal efficacy in improving liver and kidney function, aligning with the "lower is better" lipid-lowering strategy recommended by multiple international guidelines (e.g., ESC 2021, ACC/AHA 2018). The theoretical basis for early intervention lies in the fact that during the acute phase of ACS, inflammatory responses are heightened, and plaque vulnerability increases. Early reduction and improvement of liver and kidney function can reduce the volume of the lipid core and stabilize plaque structure. Additionally, early intensive lipid lowering may slow the progression of atherosclerosis by inhibiting oxidative stress and endothelial dysfunction.

Another significant finding of this study is that there were no significant differences in baseline indicators (including ApoA, TC, TG, HDL-C, LDL-C, Lp(a), and ApoB) among the three groups of patients before treatment ($P > 0.05$). After one month, three months, and six months of treatment, all indicators in study group 1 and study group 2 showed significant improvements compared to the control group ($P < 0.05$), with study group 1 demonstrating particularly pronounced improvements ($P < 0.05$). Furthermore, the incidence of MACE was significantly lower in the early intensive lipid-lowering group. These results are consistent with the conclusions of large-scale clinical trials conducted by Hirai and Yoshikawa *et al.*, which suggest possible mechanisms including stabilizing vulnerable plaques and reducing the risk of plaque rupture; improving endothelial function and reducing the tendency for thrombosis; and inhibiting inflammatory responses to slow the progression of atherosclerosis [8,9].

It is noteworthy that the early intensive lipid-lowering group included in this study received PCSK9 inhibitor treatment immediately after PCI, whereas the control group mostly adopted a stepwise adjustment strategy. This may lead to differences in lipid control between the two groups manifesting in the short term, thereby affecting the incidence of MACE. In recent years, the widespread application of PCSK9 inhibitors has further enhanced lipid-lowering efficacy without increasing the risk of muscle or liver toxicity associated with statins [10]. Therefore, for high-risk PCI patients with acute coronary syndrome, an early combined lipid-lowering strategy (e.g., statins + ezetimibe + PCSK9 inhibitors) may represent a safe and effective option.

5. Conclusion

In summary, early initiation of intensive lipid-lowering therapy can significantly improve the rate of achieving lipid targets in patients with ACS undergoing PCI and reduce the risk of MACE, while demonstrating good safety. This strategy aligns with the current guideline-recommended concept of "early intervention and intensive management," holding significant clinical importance for improving the prognosis of ACS patients. In the future, it is necessary to further optimize individualized lipid-lowering regimens to enhance treatment accessibility and long-term efficacy.

Disclosure statement

The authors declare no conflict of interest.

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