Factors associated with unattained LDL-cholesterol goals in Chinese patients with acute coronary syndrome one year after percutaneous coronary intervention

Wenduo Zhang, MD, Fusui Ji, MB, Xue Yu, MS, Xinyue Wang, MS

Abstract
Reducing low-density lipoprotein cholesterol (LDL-C) to target ≤1.81 mmol/L is a common therapeutic goal after acute coronary syndrome (ACS). This study aimed to examine the factors associated with reaching or not this LDL-C target after 1 year of optimal statin therapy postpercutaneous coronary intervention (PCI). This was a retrospective study of 633 consecutive prospectively enrolled patients with ACS treated between January 2011 and December 2012 at the Beijing Hospital (China). All patients were treated with PCI and statins for 1 year. A multivariate analysis was carried out to identify the factors associated with reaching the LDL-C target of ≤1.81 mmol/L. The rate of unreached LDL-C goal after 1 year was 48%. Compared with those who achieved their LDL-C goal, patients not achieving their LDL-C goal showed a higher proportion of females (37.9% vs 28.7%, \( P < 0.001 \)), higher LDL-C levels at admission (2.82 ± 0.75 vs 2.08 ± 0.70 mmol/L, \( P < 0.001 \)); lower proportion of patients with a history of PCI (17.6% vs 24.8%, \( P = 0.03 \)), and younger age (66.7 ± 10.6 vs 68.9 ± 10.1 years, \( P = 0.009 \)). A multivariate analysis showed that lower LDL-C levels on admission were predictive of LDL-C goal achievement (odds ratio [OR] = 4.81; 95% confidence interval [CI]: 3.46–6.70; \( P < 0.001 \)), together with older age (OR: 0.98; 95% CI: 0.96–0.997; \( P = 0.026 \)), and male gender (OR: 0.64; 95% CI: 0.42–0.98; \( P = 0.040 \)).

Abbreviations: ACS = acute coronary syndrome, CHD = coronary heart disease, LDL-C = low-density lipoprotein cholesterol, PCI = percutaneous coronary intervention, TG = triglycerides.

Keywords: acute coronary syndrome, coronary artery disease, low-density lipoprotein cholesterol, percutaneous coronary intervention, statin, treatment target

1. Introduction
Acute coronary syndrome (ACS) refers to a wide spectrum of diseases caused by acute myocardial ischemia and/or necrosis secondary to reduced coronary blood flow caused by unstable angina, non-ST-elevation myocardial infarction, or ST-elevation myocardial infarction.\(^1\) There are about 230 million patients with cardiovascular diseases in China alone.\(^1\) Age, male gender, dyslipidemia, obesity, tobacco exposure, diabetes, hypertension, and a previous history of cardiovascular diseases are major risk factors for ACS.\(^1,2\) Elevated low-density lipoprotein cholesterol (LDL-C) is an independent risk factor for coronary heart disease (CHD).\(^3,4\) Epidemiological studies and clinical trials have shown that plasma cholesterol levels are linearly correlated with the prognosis of CHD.\(^5,6\)

Lipid-lowering therapy is used to reduce the risk of recurrent cardiovascular events among patients with ACS. Nevertheless, since the primary aim of LDL-C-lowering therapy is the prevention of a novel coronary event, risk stratification is needed to be determined before treatment.\(^7\) The latest guidelines and studies suggest that LDL-C target for patients with ACS should be ≤1.81 mmol/L after percutaneous coronary intervention (PCI).\(^8-10\) Supporting this target, previous studies suggested that cardiovascular events could be reduced by 20% to 50% when LDL-C levels are decreased to ≤1.81 mmol/L.\(^11,12\) Statins are presently the main drugs used to decrease LDL-C levels; their efficacy in reducing secondary coronary events is well-established.\(^1,3,14\)

PCI is presently the gold standard treatment for ACS, especially for patients with significant left main coronary artery disease, patients with 3-vessel disease, or patients with suboptimal revascularization and ongoing symptoms despite maximal nonsurgical therapy.\(^11,12\) These patients are considered at high recurrence risk and their LDL-C levels should be ≤1.81 mmol/L.\(^12\) The DYSSIS-China study showed that the global proportion of patients reaching this LDL-C target was only 61.5%, and even reaching proportions as low as 39.7% and 54.8% in very high-risk and high-risk patients, respectively.\(^15\) A previous study from Hong Kong showed a low rate of reaching LDL-C goals at
2. Methods

2.1. Study design

This was a retrospective study of consecutive, prospectively enrolled patients with ACS treated between January 1st, 2011 and December 31st, 2012 at the Department of Cardiology of Beijing Hospital (China). Four cardiologists participated in the original study. The study was approved by the ethics committee of the Beijing Hospital. All patients provided a written informed consent.

2.2. Patients

The inclusion criteria were: available coronary angiography showing stenosis ≥75% (or ≥50% for the left main coronary artery) in at least 1 main coronary artery (left main coronary artery, left anterior descending artery, circumflex branch of left coronary artery, or right coronary artery); underwent PCI; and had been treated with optimal statin regimen for 1 year after PCI.

The exclusion criteria were: allergy or intolerance to statins; stroke or history of visceral bleeding disorder within 6 months; severe liver disease or coagulation abnormalities; history of valvular heart disease, cardiomyopathy, myocarditis, congenital heart disease, peripheral vascular disease, or infective endocarditis; chronic kidney disease stage >III; cancer patients with a life expectancy <1 year; chronic heart failure; or any serious disease that could affect the short-term prognosis.

After screening the 1000 eligible subjects of the original prospective study, 300 were excluded (210 patients with ACS symptoms but angiography findings did not meet the stenosis criteria, 30 patients underwent coronary artery bypass graft, 10 patients refused PCI, and 50 patients declined participation) and 700 patients met the criteria and were included in the present retrospective study. All these subjects had received secondary prevention for CAD and were followed up for 1 year after PCI. Sixty-seven (9.6%) patients were lost to follow-up and 1-year follow-up data were available for 633 patients (90.4%).

2.3. Data collection

Data were prospectively recorded and included gender, age, and risk factors (diabetes, obesity, hypertension, and family history) at baseline. Serum levels of total cholesterol, LDL-C, high-density lipoprotein cholesterol, and triglycerides (TG) were collected at admission and after 1 year of treatment. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, or taking antihypertensive medication.[17] Diabetes was defined as fasting plasma glucose levels ≥7.0 mmol/L, postprandial glucose levels ≥11.1 mmol/L, and/or glycated hemoglobin ≥6.5%, or taking antidiabetes medication.[18] Obese was defined as a body mass index ≥30.0 kg/m².[19] Smoking was defined as using tobacco at admission.

2.4. Treatments

PCI was performed in a standard manner. Patients received aspirin (100–300 mg/day) and clopidogrel (300–600 mg) at least 2 hours before PCI. Glycoprotein IIb/IIIa inhibitors were used at the surgeon’s discretion. Angiographic success was defined as a final stenosis <20%.

An aspirin dose of 300 mg/day was prescribed for 1 to 3 months, followed by 100 mg/day lifelong. Clopidogrel (75 mg/day) was given for at least 1 year after PCI.

Patients received statin treatment according to the clinical opinion of their treating physician and according to current guidelines.[18,3,10] Treatment included atorvastatin 20 mg/day, rosuvastatin 10 mg/day, pravastatin 40 mg/day, fluvastatin 80 mg/day, and simvastatin 20 mg/day. Compliance was self-reported.

2.5. LDL-C goal

The LDL-C goal of ≤1.81 mmol/L was selected according to the 2007 Chinese adult dyslipidemia Prevention Guidelines.[20] LDL-C levels were determined using the Friedewald equation.[21] Patients were grouped according to whether they had reached the <1.81 mmol/L goal by 1 year after PCI or not.

2.6. Statistical analysis

Results were presented as mean ± standard deviation or median with interquartile range, as appropriate. Non-normally distributed data (according to the Kolmogorov–Smirnov test) were log-transformed to normalize their distribution. One-way ANOVA with the LSD post hoc test was used for data analysis. Categorical data were presented as proportions and analyzed using the Chi-square test. Variables that were significantly associated with the outcome in univariate analyses were entered in a multiple logistic regression model to examine the association of the baseline parameters with reaching the LDL-C target. Statistical analyses were performed with SPSS 17.0 (IBM, Armonk, NY). Two-sided P-values < 0.05 were considered statistically significant.

3. Results

3.1. Characteristics of the patients

Table 1 presents the characteristics of the patients. Mean age was 67.7 years, and 43.5% of the patients were female. Among the 633 patients, only 48% reached the LDL-C goal after 1 year of post-PCI statin treatment. Compared with those who reached the LDL-C goal, patients who did not reach the goal displayed a higher proportion of females (37.9% vs 29.7%, P = 0.02) and these patients were younger (66.7 ± 10.6 vs 68.9 ± 10.1 years, P = 0.009). There were no significant differences in body mass index, hypertension, smoking, hyperlipidemia, diabetes, and stroke between the 2 groups. There was a higher proportion of patients with a history PCI among the patients reaching the goal (24.8% vs 17.6%, P = 0.03). The baseline values of TG, total cholesterol, and LDL-C were higher among the patients who did not reach the goal compared with those who did (all P < 0.001).

Table 2 shows that there were no differences among patients undergoing different statin treatments (all P > 0.05), except that rosuvastatin led to a higher reduction of LDL-C levels than the other statins (P < 0.05). There were no differences in the proportion of patients reaching the LDL-C goal among the different treatments (P > 0.05).
3.2. Predictors of reaching the LDL-C target

The multivariate analysis showed that lower LDL-C levels on admission were predictive of reaching the LDL-C goal (odds ratio [OR] = 4.81; 95% confidence interval [CI]: 3.46–6.70; *P* < 0.001), together with older age (OR: 0.98; 95% CI: 0.96–0.99; *P* = 0.026), and male gender (OR: 0.64; 95% CI: 0.42–0.98; *P* = 0.040) (Table 3).

4. Discussion

Reducing LDL-C to ≤1.81 mmol/L is a common therapeutic goal after ACS,[16–3,10] but there is a lack of real-world data about the proportion of patients with ACS reaching this LDL-C goal after 1 year of post-PCI statin therapy. Therefore, the aim of the present study was to examine the rate of patients reaching the LDL-C target of ≤1.81 mmol/L of post-PCI statin treatment and the factors associated with goal fulfillment. Results showed that lower LDL-C levels at admission were predictive of LDL-C goal achievement, together with older age and male gender.

The present study showed that after 1 year of post-PCI conventional statin therapy in high-risk patients, the proportion of patients reaching the LDL-C goal was 48%. This rate was higher than that of the DYSIS-China study (38%)[15] and a study from Hong Kong,[16] but these previous studies used a primary

### Table 1

Baseline characteristics of the patients according to whether or not they reached the LDL-C target of ≤1.81 mmol/L by one year after PCI.

| Characteristic                  | Unattained goal (n = 330, 52%) | Attained goal (n = 303, 48%) | *P*
|--------------------------------|--------------------------------|-------------------------------|-----
| Age, year                      | 66.7 ± 10.6                    | 68.9 ± 10.1                   | 0.009
| Gender (male, %)               | 205 (62.1%)                    | 216 (71.3%)                   | 0.018
| Body mass index, kg/m²         | 25.6 ± 3.2                     | 25.6 ± 4.0                    | 0.600
| Hypertension                   | 233 (70.6%)                    | 219 (72.3%)                   | 0.726
| Diabetes                       | 108 (32.7%)                    | 110 (36.3%)                   | 0.403
| Stroke                         | 4 (1.2%)                       | 2 (0.6%)                      | 0.398
| Smoking                        | 137 (41.5%)                    | 122 (40.3%)                   | 0.455
| Prior PCI                       | 58 (17.6%)                     | 75 (24.8%)                    | 0.032
| Hyperlipidemia                 | 182 (55.2%)                    | 173 (57.1%)                   | 0.480
| TC, mmol/L                     | 2.82 ± 0.75                    | 2.08 ± 0.70                   | 0.001
| HDL-C, mmol/L                  | 1.03 ± 0.28                    | 1.00 ± 0.27                   | 0.117
| TC, mmol/L                     | 2.83 ± 0.75                    | 2.82 ± 0.91                   | 0.001
| HDL-C, mmol/L                  | 1.82 ± 1.02                    | 1.45 ± 0.73                   | 0.001
| Coronary lesion                | LMCA                           | 32 (9.7%)                     | 0.554
| Triple                         | 213 (64.6%)                    | 192 (63.4%)                   | 0.361
| Affected vessels               | Single                         | 36 (10.9%)                    | 0.091
| Double                         | 81 (24.6%)                     | 79 (26.1%)                    | 0.420
| Triple                         | 192 (61.9%)                    | 101 (72.1%)                   | 0.881
| Reached LDL-C goal             | Unattained goal (n = 330, 52%) | Attained goal (n = 303, 48%) | 0.091

**HDL-C** = high-density lipoprotein cholesterol, **LDL-C** = low-density lipoprotein cholesterol, **LMCA** = left main coronary artery, **PCI** = percutaneous coronary intervention, **TC** = total cholesterol, **TG** = triglycerides.

### Table 2

Characteristics of the patients according to the statin therapy.

| Statin          | N   | Age, year | Male/female | BMI, kg/m² | Hypertension, % | Diabetes, % | Stroke, % | Smoking, % | Prior PCI, % | Hyperlipidemia, % | LDL-C, mmol/L | HDL-C, mmol/L | TC, mmol/L | TG, mmol/L | LDL-C reduction | Affected vessels | Reached LDL-C goal |
|-----------------|-----|-----------|-------------|------------|----------------|-------------|-----------|-------------|--------------|------------------|--------------|--------------|------------|-----------|----------------|----------------|------------------|
| Atorvastatin    | 310 | 68.6 ± 10.1 | 2.01     | 25.4 ± 3.7 | 220 (71.0%) | 111 (35.8%) | 4 (0.3%) | 130 (41.9%) | 63 (20.3%) | 169 (65.4%) | 2.46 ± 0.91 | 1.01 ± 0.26 | 4.29 ± 1.16 | 1.56 ± 0.87 | 6.6 ± 1.64 | 37 (11.9%) |
| Rosuvastatin    | 140 | 66.3 ± 10.1 | 2.01     | 25.5 ± 3.8 | 105 (75.2%) | 52 (37.1%)  | 0 (0.0%) | 60 (42.9%)  | 31 (22.1%) | 84 (60.0%)  | 2.47 ± 0.69 | 4.31 ± 0.95 | 1.00 ± 0.31 | 1.73 ± 1.00 | 14.0 ± 2.90 | 9 (6.4%)  |
| Pravastatin     | 53  | 67.5 ± 10.9 | 1.91     | 25.3 ± 3.4 | 29 (54.7%)  | 14 (24.5%)  | 0 (0.0%) | 21 (39.6%)  | 11 (20.8%) | 25 (47.2%)  | 2.56 ± 0.76 | 1.09 ± 0.35 | 4.31 ± 1.12 | 1.58 ± 0.82 | 6.0 ± 4.30 | 11 (20.8%) |
| Fluvastatin     | 19  | 67.9 ± 11.2 | 1.41     | 26.7 ± 3.1 | 17 (22.2%)  | 8 (42.1%)   | 0 (0.0%) | 10 (52.6%)  | 4 (21.1%)  | 11 (57.8%)  | 2.79 ± 0.51 | 0.96 ± 0.19 | 4.62 ± 0.72 | 1.38 ± 1.37 | 5.3 ± 7.28 | 0 (0.0%)  |
| Simvastatin     | 111 | 67.2 ± 11.1 | 1.81     | 25.2 ± 3.2 | 70 (71.4%)  | 37 (33.3%)  | 2 (1.8%) | 50 (45.0%)  | 25 (22.5%) | 66 (59.5%)  | 2.39 ± 0.72 | 4.18 ± 1.05 | 1.00 ± 0.23 | 1.65 ± 0.82 | 2.6 ± 2.6% | 10 (9.0%) |

**BMI** = body mass index, **C** = low-density lipoprotein cholesterol, **HDL-C** = high-density lipoprotein cholesterol, **LMCA** = left main coronary artery, **PCI** = percutaneous coronary intervention, **TC** = total cholesterol, **TG** = triglycerides.
A goal of 2.6 mmol/L instead of 1.81 mmol/L, but the Hong Kong study suggested that further LDL-C reduction below 2.6 mmol/L did not result in any additional benefit. Indeed, based on the survival curves in relation to LDL-C levels, no additional benefit was observed below 2.4 mmol/L.

There are a number of possible reasons for discrepancies among studies. First, with the implementation of recent guidelines, physicians have an improved awareness of the LDL-C goals for very high-risk patients. Second, differences in follow-up time could play a role. Third, the proportion of high-risk patients in the DYSIS-China study was lower than in the present study. Fourth, the distributions of statin treatments were different among studies. Finally, the differences among studies could be due to study population and target LDL-C levels. Indeed, this could be due to ethnic differences between the Chinese and Caucasian populations, leading to the inapplicability of the thresholds used in Western populations. Additional studies are still necessary to examine the exact outcomes of LDL-C reduction in these patients.

Different statins have different LDL-C reduction potency. The magnitude of LDL-C reduction observed in the present study is comparable to that reported by a meta-analysis. The present study showed that rosvastatin had the best LDL-C-lowering effect compared with the other statins. Nevertheless, the rates of achieving the LDL-C goal were similar among the different statin treatments. A possible explanation is that patients with a greater number of factors of poor prognosis could be more aggressively treated with more potent statins, but the present study was not designed to assess this factor and additional studies are necessary.

The present study showed that there was a higher proportion of females among the patients not reaching the LDL-C goals, which is supported by a previous study. Therefore, it could be hypothesized that female patients with high-risk CAD should receive more attention during postoperative management. Female gender is generally considered a protective factor against CHD, but this protective effect disappears after menopause. Since the mean age was around 67 to 69 years in the present study, it could be supposed that most of these women were menopausal, but this specific factor was not examined in the present study.

The rates of reaching the LDL-C goal was higher in patients with a PCI history, which could be related to these patients being more aggressively treated, as previously suggested. Indeed, previous studies showed that target achievement was associated with compliance and that compliance was associated with a higher number of follow-up visits. TG levels in patients who did not reach the LDL-C goal were higher than those in patients who reached the goal (P < 0.001). European guidelines recommend that TG-targeting treatment should be started when TG levels are >1.7 mmol/L in high-risk CAD patients. Diabetes mellitus is often combined with hypertriglyceridemia and some studies showed that the rates of achieving the LDL-C goal was lower in ACS patients with diabetes mellitus combined with hypertriglyceridemia, but the present study and a recent study did not reach this conclusion. Additional studies are necessary to clarify these points.

The present study is not without limitations. First, it was from a single hospital and only involved patients who were managed by cardiologists. Therefore, these findings may not reflect the trends in achieving lipid goals in other Asian countries or the practices of primary care physicians. Second, because of the retrospective nature of the study, some data were not available. Menopause has a strong influence on the CAD risk in women, but it was not collected. The exact dose of statin was not collected either, which constitutes a major limitation. Third, only patients who had complete lipid profile data were included. Patients without recent lipid profiles could be more likely to be undertreated, and the present study may have underestimated the actual number of patients who are achieving their lipid goals in clinical practice. Fourth, patients could not be stratified according to the type of ACS they experienced before PCI. Finally, the patients were followed up for 1 year only, preventing the observation of the long-term effects of statin therapy after PCI, as well as the prognosis of the patients. The results of the present study need to be confirmed by multicenter studies and larger sample size.

### 5. Conclusions

In conclusion, among patients treated with statin after PCI for ACS, 48% did not reach the LDL-C target of <1.81 mmol/L after 1 year of optimal post-PCI statin treatment. The multivariate analysis suggested that higher LDL-C levels at admission, younger age, and female gender were independently associated with not reaching the LDL-C target after 1 year of optimal statin therapy after PCI. Additional multicenter studies are necessary to confirm and refine these results, but young or female patients should be given a closer follow-up after PCI and more aggressive lipid-lowering could be necessary, including diet and a combination of drugs.

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