Cholesterol Goal Attainment in Patients with Coronary Heart Disease and Elevated Coronary Risk: Results of the Hong Kong Hospital Audit Study

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ABSTRACT

Objective: We sought to determine 1) long-term lipid-lowering treatment patterns; 2) cholesterol goal attainment rates and possible determinants of goal achievement; and 3) effects of cholesterol goal attainment on coronary events in hospitalized Hong Kong patients.

Methods: In this retrospective cohort analysis, records of two public Hong Kong hospitals were reviewed for 196 adults (69% with coronary heart disease (CHD) or CHD-risk equivalent) who received at least one lipid-lowering therapy during hospitalization. Low-density lipoprotein cholesterol (LDL-C) targets were <2.6 mmol/l (<100 mg/dL) for patients with CHD or CHD risk equivalents and <3.37 mmol/l (<130 mg/dL) for those without.

Results: Most participants were initiated on regimens of low to midequipotency doses and never had their regimens adjusted to higher potency. Approximately 44% of patients not at LDL-C at baseline failed to achieve goal during a median follow-up of 1.9 years. Patients with higher coronary risk and/or LDL-C levels at baseline were less likely than their lower-risk counterparts to achieve goal; for each 1-mmol/l (38.7-mg/dL) increase in LDL-C at baseline, the likelihood of attaining goal declined by 64%. Patients achieving cholesterol goal had significantly longer cardiovascular event-free times.

Conclusions: A total of 44% of Hong Kong patients not at LDL-C goals at baseline did not achieve them over 1.9 years. More effective and well-tolerated therapies, including adjunctive regimens (e.g., ezetimibe-statin, niacin-statin), may be necessary to enhance LDL-C goal achievement and increase event-free time.

Keywords: coronary disease, hydroxymethylglutaryl CoA reductase inhibitors, hypercholesterolemia, prevention and control.

Introduction

Cardiovascular disease, including coronary heart disease (CHD) and stroke, is a leading cause of morbidity and mortality. Each year, these conditions account for 17 million deaths worldwide [1]. In Hong Kong, since the 1960s, CHD has been second only to cancer as the most common cause of death [2], and it is the most common organ-specific cause of death [3]. Although the mortality rate associated with CHD has markedly declined in the United States and parts of Western Europe during the past 40 years, the rate has been increasing in Hong Kong [3].

Coronary heart disease is a major cause of both mortality and morbidity in Hong Kong. According to 2003 estimates, each year, heart disease accounted for 14.6% of all deaths, and diseases of the circulatory system accounted for 8.5% of all hospitalizations (~103,000 of 1.2 million) [4]. In addition, CHD is a major source of health-care expenditures. In 2000 to 2001, the total cost of managing acute myocardial infarction (MI) alone was $45 million (HKD 349 million), or 0.5% of total health-care expenditures [5]. Nevertheless, absolute cardiovascular risk is a continuous rather than a categorical variable, and even patients without prior CHD may be at increased risk because of major cardiovascular risk factors (e.g., smoking, hypertension).

Chinese populations have traditionally been perceived to have lower cholesterol levels than Caucasians [6–8]. Nevertheless, rapid economic growth and urbanization in recent decades have been associated with a high prevalence of CHD and cholesterol levels in Hong Kong. As of 2003/2004, life expectancy at birth was higher in Hong Kong than in the United States for both males (78.5 years) and females.
the burden of chronic diseases (including CHD) is known to increase with rising life expectancy [4]. A recent long-term study showed that 54.2% of 17,716 participants had a total cholesterol level >5.2 mmol/l (>200 mg/dL), and 44.3% (including 41.5% of men) had 10-year absolute coronary risk ≥20% [9].

Landmark randomized controlled trials have demonstrated that intensive lipid-lowering therapy significantly reduces cardiovascular events and progression of coronary atherosclerosis compared with more moderate regimens [10–12]. These and other findings led a US consensus panel to urge consideration of new, lower targets for low-density lipoprotein cholesterol (LDL-C) among certain high-risk patients [13]. Nevertheless, recent studies have shown that patients are not being treated even to older, less aggressive cholesterol goals and that lipid-lowering treatments are dominated by statin monotherapy prescribed at low- to moderate-equidose potency [14–19].

The objectives of the Hong Kong Hospital Audit Study were to determine 1) long-term lipid-lowering treatment patterns; 2) cholesterol goal attainment rates and possible determinants of goal achievement; and 3) effects of cholesterol goal attainment on coronary events in hospitalized Hong Kong patients.

Methods

The Hong Kong Hospital Audit Study was a 2-year multicenter, retrospective cohort analysis involving medical records of two public Hong Kong hospitals. Records of patients who had been admitted to the emergency department secondary to acute MI were identified by computer database system at the two centers.

Patients

Study participants were adults (>18 years of age) who had been hospitalized between January 2001 and December 2002 and had admission LDL-C > 2.59 mmol/l (>100 mg/dL). Participants must not have used lipid-lowering medication for at least 6 months prior to admission (baseline period; Fig. 1). Lipid-lowering drug therapy was initiated during the hospital stay, and eligible patients remained on lipid-lowering therapy for at least 3 months after discharge.

Data Collection

This study was conducted at two of the largest acute-care regional hospitals in Hong Kong. One is located in a recently developed satellite town and the other is in a relatively old part of the city. Data collected are hence reasonably representative of the general population of Hong Kong. Anonymized case notes of patients with acute MI who were admitted during the specified period were identified using the computer system of the two hospitals. Patients with complete records for at least 2 years after the index date were recruited in the study.

Data were collected at baseline, or within 6 months before the index prescription date (Fig. 1). The index prescription date was defined as the date of the first lipid-lowering medication prescription. Initial lipid-lowering therapies were categorized by 1) equidose potency according to Maron’s formulation [19]; and 2) each patient’s baseline LDL-C category: category 1 < 3.2 mmol/l (<125 mg/dL), category 2 = 3.2 mmol/l to 4.5 mmol/l (125–175 mg/dL), and category 3 > 4.5 mmol/l (>175 mg/dL). Baseline data were collected in part to confirm that the patient had not received lipid-lowering therapy. Data collected during the study period (median = 1.9 years) included LDL-C goal attainment (see below), changes in equipotency doses, and coronary events. All recruited patients were followed up by cardiologists at the two centers, such that patients’ medical records included both inpatient and outpatient data.

Cholesterol Goal Attainment

For patients with CHD or CHD-risk equivalents, the LDL-C goal was <2.6 mmol/l (<100 mg/dL) [20]. Patients with CHD had MI, angina pectoris, or a history of coronary revascularization (percutaneous coronary intervention, coronary artery bypass grafting), while patients with CHD risk equivalents had diabetes mellitus, stroke, and/or peripheral vascular disease. For high-risk non-CHD patients, the LDL-C goal was <3.37 mmol/l (<130 mg/dL) [20].

Statistical Methods

Patients were risk-stratified based on baseline data as CHD/CHD equivalent or high-risk non-CHD. Goal attainment was evaluated at 3-month intervals until the final follow-up laboratory results were obtained. Nevertheless, not all patients had a cholesterol goal measurement at each 3-month point because this was a clinic-based study and not performed according to a protocol with scheduled assessments.

For patients with missing data, the most recent laboratory data were carried forward as the endpoint values. Factors associated with LDL-C goal attainment

Figure 1 Study design.
were evaluated using logistic regression analysis. The logistic model included age, sex, baseline smoking, baseline drinking, baseline hypertension, baseline diabetes mellitus, baseline CHD, baseline LDL-C, baseline lipid-lowering equipotency [19], and changes in equipotency potency during study follow-up. Odds ratios (ORs) and 95% confidence intervals (CIs) were computed.

Kaplan–Meier estimates were used to evaluate the impact of LDL-C goal attainment prior to event on event-free time (time to initial or subsequent event posthospitalization). Significance was at two-tailed \( \alpha = 0.05 \). SAS version 8.02 (SAS Institute, Cary, NC) was used for statistical analyses.

Results

Patients

A total of 315 patient records were identified randomly from the two hospitals. Three of these were excluded because of inconsistent data. Twenty-one others were omitted because of patient death within 30 days of admission. Of the remaining 291 patients, 196 received at least one lipid-lowering therapy during their hospital stay and comprise the study population.

Baseline characteristics for patients with or without CHD are summarized in Table 1. Approximately 69% of patients had CHD or a CHD-risk equivalent, and 46% had hypertension. The mean LDL-C was 3.92 mmol/l (152 mg/dL), and the mean total cholesterol was 5.47 mmol/l (212 mg/dL).

Lipid-Lowering Therapy

Initial lipid-lowering therapy by baseline LDL-C is presented in Figure 2 and Table 2. Overall, most regimens were initiated at low to midequipotency doses (potency ≤ 3), with initial potency of 0 to 3 in 152 (78%) of patients. Frequencies of very high equipotency dose regimens (potency 5) increased across rising LDL-C categories. Changes in lipid-lowering therapy are shown in Table 2. Changes in equipotency potency were infrequent. Approximately 60% of the initial regimens remained unchanged during the study period. Downtitration was relatively common in patients receiving high or very high equipotency dose treatments, occurring in

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**Table 1** Baseline characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>CHD/CHD-equivalent group (N = 135)</th>
<th>Non-CHD high risk group (N = 61)</th>
<th>All patients (N = 196)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD) age, year</td>
<td>63.70 (11.26)</td>
<td>61.15 (13.69)</td>
<td>62.90 (12.09)</td>
</tr>
<tr>
<td>No. (%) male</td>
<td>97 (71.85)</td>
<td>47 (77.05)</td>
<td>144 (73.5)</td>
</tr>
<tr>
<td>Mean (SD) TC, mmol/L</td>
<td>5.38 (1.23)</td>
<td>5.64 (1.21)</td>
<td>5.47 (1.22)</td>
</tr>
<tr>
<td>Mean (SD) LDL-C, mmol/L</td>
<td>3.41 (1.0)</td>
<td>3.71 (1.06)</td>
<td>3.92 (1.09)</td>
</tr>
<tr>
<td>Mean (SD) triglycerides, mmol/L</td>
<td>1.15 (0.32)</td>
<td>1.19 (0.35)</td>
<td>1.16 (0.33)</td>
</tr>
<tr>
<td>Mean (SD) HDL-C, mmol/L</td>
<td>1.89 (0.03)</td>
<td>1.66 (0.77)</td>
<td>1.81 (0.96)</td>
</tr>
<tr>
<td>No. (%) with CHD or CHD risk equivalent</td>
<td>135 (100)</td>
<td>NA</td>
<td>135 (68.9)</td>
</tr>
<tr>
<td>No. (%) with diabetes mellitus</td>
<td>60 (44.44)</td>
<td>NA</td>
<td>60 (30.6)</td>
</tr>
<tr>
<td>No. (%) with hypertension</td>
<td>66 (49.25)</td>
<td>25 (40.98)</td>
<td>91 (46.4)</td>
</tr>
<tr>
<td>No. (%) current smokers</td>
<td>76 (56.30)</td>
<td>39 (63.93)</td>
<td>115 (58.7)</td>
</tr>
<tr>
<td>No. (%) consuming alcohol*</td>
<td>16 (11.85)</td>
<td>8 (13.11)</td>
<td>24 (12.2)</td>
</tr>
</tbody>
</table>

*Patients reporting being “social” or “heavy” drinkers (number of units per week not quantified). CHD, coronary heart disease; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol.

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![Figure 2](image-url) Initial lipid-lowering therapy by baseline low-density lipoprotein cholesterol (LDL-C) category. Category 1: LDL-C < 3.2 mmol/l (<125 mg/dL), category 2: LDL-C 3.2–4.5 mmol/l (125–176 mg/dL), category 3: LDL-C > 4.5 mmol/l (>176 mg/dL). Equidose potencies by Maron’s formulation [19].
approximately 24% of equipotency-dose 4 and 40% of equipotency-dose 5 regimens.

**Cholesterol Goal Attainment**

As shown in Figure 3, 55.8% of patients not at goal at baseline (60.1% of all patients) achieved LDL-C goals after 1.9 years of follow-up (or at the most recent laboratory measurement carried forward in patients without endpoint data). Patients in the highest LDL-C category (>4.5 mmol/l [>175 mg/dL]) were least likely to achieve their LDL-C goals (Fig. 3A). LDL-C goal attainment tended to be less frequent with increasing lipid-lowering regimen equipotency potency (Fig. 3B) and was lowest among patients who received very high equipotency dose regimens (≤8%). Nevertheless, high equipotency doses were more commonly used in patients with high baseline LDL-C (Fig. 3A).

Based on logistic regression analysis, CHD/CHD equivalent patients were at lower odds of goal attainment compared with non-CHD patients (Table 3). The OR of goal attainment was 19% lower for CHD/CHD-equivalent patients compared with individuals without CHD or CHD risk equivalents (95% CI = 0.080–0.459). The only other factor impacting goal attainment apart from baseline CHD risk (Table 3) was baseline LDL-C concentration. Increases in baseline LDL-C levels significantly reduced the odds of goal attainment. For each increase of 1 mmol/l in baseline LDL-C, there was a 64% lower odds of attaining LDL-C goal (95% CI = 0.449–0.904).

Patients who attained LDL-C goal had significantly longer event-free time (time before experiencing a subsequent event or recurrent event posthospitalization and hence this can be stated as time to subsequent

### Table 2 Changes in equipotency potencies of lipid-lowering therapies

<table>
<thead>
<tr>
<th>Initial potency</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (n = 2)</td>
<td>50.0</td>
<td>0</td>
<td>50.0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1 (n = 25)</td>
<td>0</td>
<td>60.0</td>
<td>24.0</td>
<td>12.0</td>
<td>4.0</td>
<td>0</td>
</tr>
<tr>
<td>2 (n = 64)</td>
<td>0</td>
<td>8.2</td>
<td>62.3</td>
<td>18.0</td>
<td>8.2</td>
<td>3.3</td>
</tr>
<tr>
<td>3 (n = 33)</td>
<td>0</td>
<td>6.1</td>
<td>9.1</td>
<td>9.1</td>
<td>69.7</td>
<td>6.1</td>
</tr>
<tr>
<td>4 (n = 10)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>30.0</td>
<td>10.0</td>
<td>60.0</td>
</tr>
</tbody>
</table>

*Equidose potency according to Maron’s formulation [19].
†N-values in first column total 195 instead of 196 because there was 1 patient with missing information regarding dose.

![Figure 3](link) Low-density lipoprotein cholesterol (LDL-C) goal attainment by LDL-C category (A) and baseline equipotency dose (B). For definitions of LDL-C categories, see Figure 2 legend. Statin equipotency potency according to Maron’s formulation [19].
event) compared with patients who did not attain goal. The Kaplan-Meier hazard plot shown in Figure 4 demonstrates a significant increase in cardiovascular event-free time (or time to event) for patients achieving LDL-C goals. The time until the first 25% of events was 69 days for those achieving goal compared with 27 days for those not achieving goal ($P < 0.05$). The median event-free time in patients achieving LDL-C goal (396 days) was significantly higher compared with patients not achieving goal (61 days).

**Discussion**

The Hong Kong Hospital Audit Study demonstrated that 44% of hospitalized patients not at LDL-C goal at baseline failed to achieve their cholesterol goals by a median follow-up of 1.9 years. Goal achievement was significantly less frequent among patients with CHD/CHD risk equivalents (OR = 0.210) and/or elevated LDL-C levels. For each 1-mmol/l (38.7-mg/dL) increase in baseline LDL-C, the likelihood of achieving goal declined by 64%. Patients who achieved LDL-C goals had significantly reduced risks of coronary events and significantly longer cardiovascular event-free times than those who did not. Goal achievement may be particularly difficult in patients with CHD and CHD-risk equivalents in part because of both their frequently elevated baseline LDL-C levels and their more stringent LDL-C goals compared with patients having risk factors only.

Lipid-lowering treatment was dominated by low to medium equidose-potency regimens, most of which (>60%) were never adjusted to higher doses. Goal achievement did not tend to increase with increasing baseline lipid-lowering regimen potency; for instance, <8% of patients receiving very high equipotency dose regimens achieved LDL-C goal. The low goal attainment among patients receiving very high equipotency dose treatments is probably confounded by increased coronary risk and/or LDL-C levels in these patients, with the high equidose potency being a probable surrogate for advanced risk (and/or very elevated LDL-C). Neither baseline lipid-lowering treatment equidose potency nor changes in potency during the study were associated with any significant reduction in coronary events. In addition, 24% to 40% of high equipotency regimens needed to be adjusted downward.

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**Table 3** Results of logistic regression analysis: odds ratios (ORs) and 95% confidence intervals (95% CIs) for cholesterol goal attainment according to factors in the model

<table>
<thead>
<tr>
<th>OR (point estimate)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age*</td>
<td>1.010</td>
</tr>
<tr>
<td>Females†</td>
<td>0.555</td>
</tr>
<tr>
<td>Smoker‡</td>
<td>0.610</td>
</tr>
<tr>
<td>Consumer of alcohol§</td>
<td>1.840</td>
</tr>
<tr>
<td>Hypertension¶</td>
<td>1.311</td>
</tr>
<tr>
<td>CHD/CHD equivalent**</td>
<td>0.192</td>
</tr>
<tr>
<td>Baseline LDL-C††</td>
<td>0.637</td>
</tr>
<tr>
<td>Initial Statin Potency = 2‡‡</td>
<td>3.348</td>
</tr>
<tr>
<td>Initial Statin Potency = 3‡‡</td>
<td>1.938</td>
</tr>
<tr>
<td>Initial Statin Potency = 4‡‡</td>
<td>3.656</td>
</tr>
<tr>
<td>Initial Statin Potency = 5‡</td>
<td>3.667</td>
</tr>
<tr>
<td>Potency Change (vs. no change)</td>
<td>1.153</td>
</tr>
</tbody>
</table>

*aFor each increasing year; †vs. males; ‡vs. nonsmokers; §vs. nonconsumer of alcohol; ¶vs. normotensive; ‡‡vs. without CHD or CHD risk equivalents; ††for each increase of 1 mmol/l (38 mg/dL); ‡‡vs. potency 1 (low potency). CHD, coronary heart disease; LDL-C, low-density lipoprotein cholesterol.

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**Follow-up CVD Events By Goal Attainment**

<table>
<thead>
<tr>
<th>Attained goal before event</th>
<th>Time to First 25% Events</th>
<th>Number of patients that had events</th>
<th>Censored observation</th>
<th>% censored</th>
</tr>
</thead>
<tbody>
<tr>
<td>No (n= 155)</td>
<td>27</td>
<td>91</td>
<td>64</td>
<td>41.29</td>
</tr>
<tr>
<td>Yes (n= 40)</td>
<td>69</td>
<td>13</td>
<td>27</td>
<td>67.50</td>
</tr>
</tbody>
</table>

**Results: Kaplan Meier Curves for Event Free Time**

*Figure 4 Kaplan–Meier hazard plots for cardiovascular event-free time in patients who achieved or did not achieve low-density lipoprotein cholesterol goals. CVD, cardiovascular disease.*
Suboptimal clinical management of dyslipidemia, including the use of predominantly low to midequipotency dose regimens, infrequent dose adjustment, and use of combination regimens, suboptimal goal achievement, and particularly low goal attainment with increasing baseline coronary risk and/or LDL-C levels observed in the Hong Kong Hospital Audit Study extend findings of previous studies [14,15,17,18,21–26]. In addition, prior work demonstrated significant reductions in the risk of cardiovascular events in patients achieving cholesterol goals [27–29]. For instance, the Return on Expenditure Achieved for Lipid Therapy (REALITY-Sweden) study showed that patients achieving cholesterol goals were 24% less likely to experience a cardiovascular event compared with their counterparts who did not attain goal [29]. Uptitration of statin monotherapy is of limited incremental value in lowering cholesterol [30,31] and may be associated with increasing frequencies of adverse effects. Though intriguing, our findings of suboptimal cholesterol goal attainment in patients receiving high-dose lipid-lowering monotherapy, which may be largely secondary to high baseline LDL-C levels and/or absolute cardiovascular risk in these patients, need to be evaluated further in larger, well-controlled studies involving a more heterogeneous patient population followed up for a longer interval.

To achieve increasingly aggressive consensus cholesterol targets [13,32], new clinical strategies are warranted. One proven approach to enhance goal achievement is dual cholesterol inhibition [33–37]. Although 3-hydroxy-3-methylglutaryl coenzyme A reductase (HMG-CoA) reductase inhibitors (statins) effectively inhibit the rate-limiting enzyme in hepatic cholesterol biosynthesis, dietary and biliary absorption of cholesterol accounts for an equivalent portion of circulating cholesterol [38]. Via interaction with the NPC1L1 protein, ezetimibe inhibits absorption of cholesterol from the gastrointestinal tract [39].

Relevant studies have shown significant increases in LDL-C-lowering capacity [40,41] and cholesterol goal achievement with ezetimibe statins compared with statin monotherapy (with or without titration) in patients not at goal on statin monotherapy. Gagné et al. observed that ezetimibe-statins were associated with a goal attainment rate of 71.5% compared with only 18.9% for ongoing statin treatment plus placebo ($P < 0.001$) [33]. In the Ezetimibe add-on to statin for effectiveness trial, 71.0% of patients receiving ezetimibe-statins met their LDL-C goal, whereas only 20.6% of those on placebo-statin obtained goal ($P < 0.001$) [35]. In the Vytorin Versus Atorvastatin study, 85.4% of patients with CHD or CHD risk equivalents achieved the LDL-C goal of <2.6 mmol/l (<100 mg/dL) using ezetimibe-simvastatin compared with 70.0% of those using atorvastatin alone ($P < 0.001$) [36].

Potential approaches to enhance goal achievement also include other adjunctive regimens, such as niacin-statin [42] and ezetimibe bile acid resins (sequestrants) [43]. Behavioral techniques also have been used. In a previous Hong Kong study, 80.8% of patients who received lipid-modifying therapies (for primary prevention) with individualized counseling by pharmacists achieved their consensus cholesterol goals compared with 58.3% of those in the noncounseled control group [44].

Limitations of the present study include its retrospective design and relatively small patient population, which leaves open the possibility of type II error. Further, the small patient population was evaluated in an acute-care setting and included patients at high coronary risk (mostly hospitalized patients with CHD). Therefore, the findings are not necessarily generalizable to the Hong Kong ambulatory-care population at large but rather toward those in similar hospitals. By studying an in-hospital population, we cannot exclude certain biases. Standing medical orders for, and scheduled dosing of, lipid-modifying therapies might have helped to promote cholesterol goal achievement through enhanced medication compliance. This might have led to our overestimating percent goal attainment in the overall (ambulatory-care) population.

The duration of follow-up was also relatively short at approximately 2 years. LDL-C goal achievement is difficult to maintain over the long term [27]. The goal achievement rate of approximately 56% at approximately 2 years in the Hong Kong Hospital Audit Study may thus overestimate longer-term goal attainment rates. The limited baseline period (6 months) may have led to an underestimation of patients with CHD/CHD risk equivalents. This in turn might also have resulted in the 56% goal attainment being an overestimation given that patients at higher risk (e.g., CHD patients) were less likely to achieve goal. Finally, the present study did not assess the effects on goal attainment and cardiovascular risk reduction of different levels of compliance, which is known to be suboptimal in patients receiving lipid-lowering therapies [45,46].

**Conclusions**

In the Hong Kong Hospital Audit Study, 44% of hospitalized patients not at cholesterol goals at baseline did not achieve their consensus LDL-C targets. Most participants were initiated on regimens of low to mid-equipotency doses and never had their regimens adjusted to higher potency. Patients with higher baseline coronary risk and/or LDL-C levels were less likely than their lower-risk counterparts to achieve goals. For every 1 mmol/l (38.7 mg/dL) increase in baseline LDL-C, the likelihood of attaining goal declined by 64%. Patients achieving goals had significantly longer
times to cardiovascular events. More effective and well-tolerated therapies, including adjunctive regimens (e.g., ezetimibe-statins, niacin-statins), may be necessary to enhance LDL-C goal achievement and delay coronary events in Hong Kong patients.

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