Roles of High-Density Lipoprotein Cholesterol in Patients With Acute Myocardial Infarction

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Abstract: Many observational studies showed high-density lipoprotein cholesterol (HDL-C) is a strong inverse predictor of cardiovascular (CV) outcome. However, recent large clinical trials evaluating therapies to raise HDL-C level in those already on statin therapy have been discouraging. This complexity is not well-known.

A total of 28,357 acute myocardial infarction (AMI) patients were enrolled in the Korea Acute Myocardial Infarction Registry (KAMIR), which was a prospective, multicenter, nationwide, web-based database of AMI in Korea. From this registry, we evaluated 3574 patients with AMI who have follow-up HDL-C level to investigate its association with clinical outcomes. The primary endpoint was the relationship between follow-up change in HDL-C and a 12-month composite of major adverse cardiac events (MACEs).

Patients with initial HDL-C > 40 mg/dL showed significantly lower rates of 12-month MACEs, especially cardiac deaths (P < 0.001). When patients were stratified into 4 groups according to the change of HDL-C, patients with decreasing HDL-C showed significantly higher rates of 12-month MACEs as comparable with patients with increasing HDL-C. A multivariate analysis indicated that HDL-C level was a significant predictor of CV events (hazard ratio, 1.38; 95% confidence interval, 1.12–1.71) after correcting for confounding variables.

The follow-up change in HDL-C level was significantly related with CV outcomes in patients with AMI.

(Observational Study)

INTRODUCTION

Lowering low-density lipoprotein cholesterol (LDL-C) level with statins has been shown to reduce the cardiovascular (CV) events.1 However, significant residual CV risk remains even after achieving optimal LDL-C levels with statin therapy.2,3 These ongoing events have driven researchers to identify factors that contribute to the CV risk that persists in patients taking a statin. Over the last few years, epidemiological studies have provided evidence that low concentrations of high-density lipoprotein cholesterol (HDL-C) are associated with an increased risk of coronary artery disease (CAD) and CV events.4–6 Indeed, a meta-analysis of 4 large prospective studies concluded that every 1-mg/dL decrease in HDL-C is associated with a 2% to 3% increase in CV events.7 However, clinical trials on agents that increase HDL-C levels have failed to improve clinical outcomes despite substantial increases in HDL-C levels.2,7,8 Further studies are needed to re-evaluate the role of HDL-C in patients with acute myocardial infarction (AMI). Additionally, very few studies have evaluated the clinical impacts of follow-up changes of HDL-C in patients taking a statin. The purposes of this study were to re-investigate the relationship between HDL-C level and CV events in patients with AMI and to analyze the impacts of increasing or decreasing HDL-C level during statin therapy on CV events.

METHODS

Study Population

The Korea Acute Myocardial Infarction Registry (KAMIR) database is a Korean prospective, open, observational, multicenter, on-line registry to investigate risk factors for mortality in patients with AMI and to establish universal management for preventing AMI with support from the Korean Circulation Society. It began in November 2005, and 53 centers that were capable of performing a large number of primary percutaneous coronary interventions (PCIs) participated. The study protocol was approved by the institutional review board at each participating center.

As shown in Figure 1, a total of 28,357 patients with AMI were identified from the KAMIR. Of these patients, 3365...
patients with insufficient initial lipid profile data and 6740 who had not been followed up clinically for 12 months were excluded and the remaining 18252 patients were analyzed for the effects of initial HDL-C level (HDL-C >40 mg/dL or HDL-C <40 mg/dL) on CV events (phase 1 analysis). Among them, 3574 patients had sufficient 12-month lipid profile and clinical outcome data. Using these data, we analyzed the effects of a follow-up change in HDL-C level after statin therapy on CV events (phase 2 analysis). The patients were classified into 4 groups according to the follow-up change in HDL-C level (group 1: initial HDL-C ≥40 mg/dL and follow-up HDL-C ≥40 mg/dL, n = 1446; group 2: initial HDL-C ≥40 mg/dL and follow-up HDL-C <40 mg/dL, n = 592; group 3: initial HDL-C <40 mg/dL and follow-up HDL-C ≥40 mg/dL, n = 503; and group 4: initial HDL-C <40 mg/dL and follow-up HDL-C <40 mg/dL, n = 1033).

Study Endpoints
All definitions of the clinical outcomes in this study were based on the recommendations of the Academic Research Consortium. The primary endpoint was the relationship between follow-up change in HDL-C and a 12-month composite of major adverse cardiac events (MACEs), which was composed of all-cause mortality, recurrent MI, and repeat revascularization. The secondary endpoints were individual components of the primary endpoint, cardiac death, and coronary artery bypass grafting from any cause.

Statistical Analysis
Statistical analyses were carried out using the SPSS Statistics for Windows ver. 17.0 software (SPSS Inc, Chicago, IL). A 2-sided \( P < 0.05 \) was considered significant. Continuous variables are presented as means ± standard deviations and were evaluated for normality of the distribution and compared using Student \( t \) test or analysis of variance accordingly. The continuous parameters with a skewed distribution were logarithmically transformed. Categorical variables are presented as frequencies and percentages and were compared using the \( \chi^2 \) or Fisher exact test, when appropriate. The Kaplan–Meier method and log-rank test were used to assess 12-month event-free survival for the MACEs. Multivariate Cox proportional hazards regression analyses were performed to determine independent variables associated with 12-month MACEs. All variables (age, sex, body mass index [BMI], Killip class, hypertension, DM, dyslipidemia, smoking, previous CAD, lipid profile, high-sensitivity C-reaction protein [hsCRP], and NT-proBNP) were entered en bloc, and the results are expressed with a hazard ratio (HR) and 95% confidence interval (CI).

RESULTS

Baseline Characteristics
The baseline characteristics, CV risk factors, in-hospital medications, initial laboratory findings, and angiographic findings according to initial HDL-C level are summarized in Table 1. Patients with a low initial HDL-C level (<40 mg/dL) were younger, more male, had a higher BMI, a poorer Killip class, less ST-segment elevation MI, and higher rates of hypertension, diabetes mellitus (DM), and smoking than those in the higher HDL-C group. This group also showed higher levels of creatinine, hsCRP, and NT-proBNP, but lower maximal levels of creatine kinase-MB and troponin I. Patients with a low initial HDL-C level showed more 3-vessel disease and stent implantation on angiography than those with a higher initial HDL-C level.
| TABLE 1. Baseline Patient Characteristics     | Phase 1 Analysis (n = 18,252) | Phase 2 Analysis (n = 3,574) | P       |
|---------------------------------------------|------------------------------|-----------------------------|---------|
|                                            | Group 1 (n = 14,068) | Group 2 (n = 3,592) | Group 3 (n = 592) | Group 4 (n = 103) |
| Age, y                                      | 67 ± 12.9                      | 62.8 ± 13.1                    | <0.001  | 63.1 ± 12.7                      | 61.8 ± 12.1                    | <0.001  | 61.7 ± 12.9                      |
| Sex, male (%)                               | 10,161 (70%)                   | 2,219 (65%)                    | <0.001  | 2,219 (65%)                      | 459 (77%)                      | <0.001  | 231 (38%)                       |
| Body mass index, kg/m²                      | 23.5 ± 5.7                     | 23.0 ± 5.8                     | <0.001  | 23.0 ± 5.8                       | 17.0 ± 2.8                     | <0.001  | 12.8 ± 2.7                       |
| Smoking                                     | 1,482 (20%)                    | 315 (6%)                       | <0.001  | 315 (6%)                         | 52 (10%)                       | <0.001  | 27 (14%)                        |
| Cardiovascular risk factors                 | 513 (4%)                      | 118 (3%)                       | <0.001  | 118 (3%)                         | 23 (5%)                        | <0.001  | 17 (8%)                         |
| Diabetes mellitus                           | 272 (7%)                      | 64 (2%)                        | <0.001  | 64 (2%)                          | 12 (2%)                        | <0.001  | 9 (1%)                          |
| Hypertension                                | 5,003 (12%)                    | 1,058 (26%)                    | <0.001  | 1,058 (26%)                      | 213 (36%)                      | <0.001  | 19 (3%)                         |
| Dyslipidemia                                | 759 (10%)                     | 114 (3%)                       | <0.001  | 114 (3%)                         | 20 (4%)                        | <0.001  | 11 (1%)                         |
| Total cholesterol, mg/dL                   | 206 ± 43.2                     | 168 ± 42.2                     | <0.001  | 168 ± 42.2                       | 21 ± 31.1                      | <0.001  | 20 ± 30.1                       |
| Triglyceride, mg/dL                         | 190.3 ± 43.2                   | 172.2 ± 42.2                   | <0.001  | 172.2 ± 42.2                     | 174.5 ± 43.9                   | <0.001  | 171 ± 43.9                      |
| HDL-C, mg/dL                                | 43.9 ± 12.9                    | 37.6 ± 13.6                    | <0.001  | 37.6 ± 13.6                      | 35.9 ± 13.7                    | <0.001  | 33.1 ± 12.8                     |
| Apolipoprotein B, mg/dL                     | 121 ± 32.9                     | 114 ± 31.9                     | <0.001  | 114 ± 31.9                       | 107 ± 28.6                     | <0.001  | 101 ± 27.6                      |
| Creatinine, mg/dL                           | 0.8 ± 0.5                      | 0.7 ± 0.4                      | <0.001  | 0.7 ± 0.4                        | 0.6 ± 0.4                      | <0.001  | 0.6 ± 0.4                        |
| Glucose, mg/dL                              | 106.3 ± 18.9                   | 102.3 ± 17.9                   | <0.001  | 102.3 ± 17.9                     | 97.1 ± 16.9                    | <0.001  | 97.1 ± 16.9                     |
| Hemoglobin, g/dL                            | 13.8 ± 1.3                     | 13.7 ± 1.3                     | <0.001  | 13.7 ± 1.3                       | 13.5 ± 1.3                     | <0.001  | 13.5 ± 1.3                      |
| LVEF                                         | 54.6 ± 11.2                    | 53.4 ± 10.9                    | <0.001  | 53.4 ± 10.9                      | 53.0 ± 10.6                    | <0.001  | 52.8 ± 10.6                     |
| Initial laboratory findings                 |                              |                              |         |                              |                              |         |                              |
| Maximal CK, IU/L                            | 572.0 ± 92.1                   | 523.0 ± 86.1                   | <0.001  | 523.0 ± 86.1                    | 516.1 ± 83.9                   | <0.001  | 514.7 ± 83.4                    |
| Maximal CK-MB, ng/mL                        | 43.3 ± 9.2                     | 36.2 ± 8.6                     | <0.001  | 36.2 ± 8.6                       | 35.3 ± 7.7                     | <0.001  | 35.0 ± 7.6                      |
| hsCRP, mg/dL                                | 6.8 ± 2.1                      | 6.5 ± 1.9                      | <0.001  | 6.5 ± 1.9                        | 6.3 ± 1.7                      | <0.001  | 6.1 ± 1.6                       |
| NT-proBNP, pg/mL                            | 500 ± 113                      | 453 ± 106                      | <0.001  | 453 ± 106                        | 406 ± 98                       | <0.001  | 394 ± 98                        |
| HbA1c (%)                                   | 6.6 ± 1.3                      | 6.3 ± 1.2                      | <0.001  | 6.3 ± 1.2                        | 6.2 ± 1.1                      | <0.001  | 6.0 ± 1.0                       |
Clinical Impact of Initial HDL Level on Clinical Outcomes

As shown in Figure 2A and Table 2, subjects with a higher HDL-C level at admission showed significantly lower rates of 12-month MACEs, particularly cardiac and all-cause mortality. These beneficial effects were observed in the rates of in-hospital mortality and 1-month MACEs during the early period after AMI. The Cox-regression analysis revealed that low HDL-C level at admission was an important predictor for 12-month MACEs before and after adjusting for other important covariates (age, sex, body mass index [BMI], Killip class, hypertension, DM, dyslipidemia, smoking, previous CAD, lipid profile, hsCRP, and NT-proBNP), as shown in Figure 3A. Additionally, older age (≥65 years), poorer Killip class, DM, dyslipidemia, previous CAD, chronic kidney disease, and high levels of hsCRP and NT-proBNP were significant independent predictors for 12-month MACEs (Table 3).

Clinical Impact of Follow-Up Changes in HDL-C

A total of 3,574 patients with lipid profile data at admission and 12 months were stratified into 4 groups according to the follow-up change between the initial and follow-up HDL-C levels using the reference value of 40 mg/dL HDL-C. Group 2 had more females, fewer smokers, and a lower creatinine level than those in group 4 (Table 1). No significant differences in other variables were found, including in-hospital medications or

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**FIGURE 2.** Kaplan–Meier curve of the prevalence of 1-year MACEs. (A) Initial HDL-C. (B) quartiles of HDL-C change at 6 months. HDL-C = high-density lipoprotein cholesterol; MACE = major adverse cardiac event.
TABLE 2. Clinical Outcomes According to HDL Level

| Group | HDL-C ≥40 mg/dL | HDL-C <40 mg/dL |
|-------|----------------|----------------|
| Group 1 | n=1446 | n=7669 |
| Phase 1 Analysis | | |
| Death | 328 (8%) | 383 (5%) |
| Cardiac death | 280 (3%) | 323 (4%) |
| Recurrent MI | 57 (0.5%) | 64 (0.8%) |
| Total MACEs | 13 (0.9%) | 9 (1.5%) |
| Group 2 | n=592 |
| Phase 2 Analysis | | |
| Death | 525 (5%) | 553 (7%) |
| Cardiac death | 396 (4%) | 420 (5%) |
| Recurrent MI | 104 (1.0%) | 97 (1.3%) |
| Total MACEs | 124 (15%) | 136 (16%) |
| Group 3 | n=593 |
| Phase 1 Analysis | | |
| Death | 525 (5%) | 553 (7%) |
| Cardiac death | 396 (4%) | 420 (5%) |
| Recurrent MI | 104 (1.0%) | 97 (1.3%) |
| Total MACEs | 124 (15%) | 136 (16%) |
| Group 4 | n=1033 |
| Phase 2 Analysis | | |
| Death | 525 (5%) | 553 (7%) |
| Cardiac death | 396 (4%) | 420 (5%) |
| Recurrent MI | 104 (1.0%) | 97 (1.3%) |
| Total MACEs | 124 (15%) | 136 (16%) |

The 2013 American College of Cardiology/American Heart Association and 2015 European Society guidelines on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults do not recommend adding therapy to raise low HDL-C in patients who are on maximal statin therapy. These guidelines only focused on LDL-C and do not recommend follow-up HDL-C level checks because some clinical trials have indicated no improvement in clinical outcome despite treatment to increase HDL-C levels.7,8,15 However, it is important to regularly check HDL-C level, take more intensive statin therapy, modify lifestyle, and quit smoking because follow-up HDL level was a surrogate marker for residual CV risk in our data.

The discrepancy between previous studies and ours may be explained by several reasons. First, most of the previous studies

**DISCUSSION**

We showed 2 major findings in unselected consecutive patients with AMI from a large number of different hospitals. First, low initial HDL-C level was a powerful predictor of CV risk, similar to previous clinical research. Second, the decreasing follow-up HDL-C level was associated with the risk of MACES defined as all-cause death, recurrent MI, or any repeat revascularization after 1 year of follow-up in patients with AMI. HDL-C level is an important predictor of coronary events in patients with known CAD across a broad range of LDL-cholesterol levels, as demonstrated in the following post-hoc analyses of some randomized trials. An analysis of 13,173 patients in the LIPID and CARE trials found that low serum HDL-C level is a significantly stronger predictor of CV events in patients with a LDL-C <125 mg/dL compared with those with LDL-C ≥125 mg/dL.10 The event rate for a 10-mg/dL increase in HDL-C decreased 29% in those with LDL-C <125 mg/dL compared with those with LDL-C ≥125 mg/dL. Nearly 10,000 patients with established CAD were treated with either high- or low-dose statin therapy in the Treating to New Targets trial.11 A multivariate analysis revealed that HDL-C level was inversely predictive of the time to first major CV event across a spectrum of LDL-C levels, including patients with treated LDL-C levels <70 mg/dL. The rate of all-cause death or MI was 33% lower in the highest compared with the lowest HDL-C quartile in a post-hoc analysis of 2193 subjects with stable disease who participated in the COURAGE trial for optimal medical therapy.12 This relationship was even stronger among subjects with LDL-C levels <70 mg/dL. These results indicate that HDL-C could be a surrogate marker for residual CV risk in patients taking a statin.

The 2013 American College of Cardiology/American Heart Association and 2015 European Society guidelines on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults do not recommend adding therapy to raise low HDL-C in patients who are on maximal statin therapy.13,14 These guidelines only focused on LDL-C and do not recommend follow-up HDL-C level checks because some clinical trials have indicated no improvement in clinical outcome despite treatment to increase HDL-C levels.7,8,15 However, it is important to regularly check HDL-C level, take more intensive statin therapy, modify lifestyle, and quit smoking because follow-up HDL level was a surrogate marker for residual CV risk in our data.

The discrepancy between previous studies and ours may be explained by several reasons. First, most of the previous studies
were not performed in the setting of AMI unlike ours. Although dal-OUTCOMES trial was researched in Acute coronary syndrome,8 15% of patients were not an initial clinical presentation of AMI. In addition, previous studies only compared the treatment group with the control group by only whether use of HDL raising agent or not. This point would be most important difference between previous studies and this work. Our study population was collected from a nationwide registry with a large sample size, an unselected population and properly adjusted for major potential confounders. Additionally, their cholesterol treatment was not done by HDL-raising agent, but only by statins. In the statin era, we showed the clinical effect of the absolute value of follow-up HDL-C level on CV risk. Group 4 showed a similar rate of 12-month MACEs compared with that of group 2, which is one of the most important findings.

In contrast, recent clinical studies have suggested that the results of previous studies were owing to abnormal HDL-C particle composition and function.16,17,19 The HDL particle has multiple antiatherogenic properties thought to be mediated by its participation in removing cholesterol from macrophages during ‘‘macrophage cholesterol efflux.’’ Furthermore, cholesterol efflux capacity has a strong inverse relationship with CV risk and increasing capacity was expected another therapeutic target.18 In addition, the vascular effects exerted by HDL in patients with a range of CV conditions differ substantially from the properties of HDL in healthy subjects, which have

**TABLE 3.** Univariate and Multivariate Cox Regression Analyses of 1-year MACEs in Patients With AMI (Phase 1)

| Variable                        | Univariate Analysis | Multivariate Analysis |
|---------------------------------|---------------------|-----------------------|
|                                 | \(P\)               | Hazard Ratio (95% CI) | \(P\)               | Hazard Ratio (95% CI) |
| Age \(\geq 65\) y               | <0.001              | 1.78 (1.64–1.94)      | <0.001              | 1.33 (1.19–1.49)      |
| Male                            | <0.001              | 1.41 (1.29–1.53)      | 0.50                | 1.05 (0.91–1.20)      |
| BMI \(\geq 25\) kg/m\(^2\)      | <0.001              | 1.25 (1.14–1.38)      | 0.14                | 1.09 (0.97–1.22)      |
| Killip class (more than II)     | <0.001              | 2.02 (1.86–2.20)      | <0.001              | 1.43 (1.28–1.59)      |
| STEMI                           | 0.11                | 0.97 (0.93–1.01)      | –                   | –                     |
| Hypertension                    | <0.001              | 1.22 (1.13–1.33)      | 0.62                | 1.03 (0.92–1.14)      |
| Diabetes mellitus               | <0.001              | 1.42 (1.31–1.55)      | <0.001              | 1.22 (1.09–1.36)      |
| Dyslipidemia                    | <0.001              | 1.30 (1.14–1.49)      | 0.002               | 1.30 (1.10–1.54)      |
| Smoking                         | <0.001              | 1.34 (1.23–1.45)      | 0.75                | 1.02 (0.90–1.15)      |
| Previous angina                 | <0.001              | 1.32 (1.22–1.44)      | 0.005               | 1.16 (1.05–1.29)      |
| Initial total cholesterol \(\geq 200\) mg/dL | <0.001              | 1.21 (1.11–1.32)      | 0.17                | 1.09 (0.96–1.24)      |
| Initial triglyceride \(\geq 150\) mg/dL | <0.001              | 1.24 (1.13–1.37)      | 0.29                | 1.07 (0.94–1.21)      |
| Initial LDL cholesterol \(\geq 100\) mg/dL | <0.001              | 1.27 (1.17–1.37)      | 0.34                | 1.06 (0.94–1.19)      |
| Initial HDL cholesterol \(< 40\) mg/dL | <0.001              | 1.22 (1.13–1.32)      | 0.01                | 1.14 (1.03–1.27)      |
| Initial creatinine \(\geq 1.5\) mg/dL | <0.001              | 2.72 (2.45–3.01)      | <0.001              | 1.74 (1.48–2.03)      |
| Initial hsCRP \(\geq 1.8\) mg/L | <0.001              | 1.63 (1.49–1.78)      | <0.001              | 1.35 (1.22–1.50)      |
| Initial NT-proBNP \(\geq 400\) pg/mL | <0.001              | 1.30 (1.19–1.42)      | 0.04                | 1.11 (1.02–1.24)      |

AMI = acute myocardial infarction, BMI = body mass index, CI = confidence interval, HDL = high-density lipoprotein, hsCRP = high-sensitivity C-reaction protein, LDL = low-density lipoprotein, MACE = major adverse cardiac event, NT-pro BNP = N-terminal prohormone of brain natriuretic peptide, STEMI = ST-elevation myocardial infarction.
TABLE 4. Univariate and Multivariate Cox Regression Analyses of 1-year MACEs in Patients With AMI

| Variable                        | Univariate Analysis | Multivariate Analysis |
|--------------------------------|---------------------|-----------------------|
|                                | P       | Hazard Ratio (95% CI) | P       | Hazard Ratio (95% CI) |
| Age ≥65 y                       | 0.01   | 1.29 (1.05–1.58)     | 0.09   | 1.22 (0.97–1.55)     |
| Male                           | 0.06   | 1.24 (0.99–1.56)     | 0.12   | 1.28 (0.94–1.74)     |
| BMI ≥25 kg/m²                   | 0.29   | 1.12 (0.91–1.39)     | 0.25   | 1.14 (0.91–1.44)     |
| Killip class (more than II)     | 0.06   | 1.24 (0.99–1.55)     | 0.16   | 1.19 (0.93–1.51)     |
| STEMI                           | 0.13   | 1.08 (0.98–1.19)     | 0.69   | 1.05 (0.84–1.30)     |
| Hypertension                    | 0.81   | 1.02 (0.84–1.26)     | 0.80   | 1.03 (0.82–1.33)     |
| Diabetes                        | 0.65   | 1.05 (0.84–1.32)     | 0.72   | 1.04 (0.82–1.33)     |
| Dyslipidemia                    | 0.15   | 1.27 (0.91–1.77)     | 0.37   | 1.17 (0.83–1.65)     |
| Smoking                         | 0.71   | 1.04 (0.84–1.28)     | 0.64   | 1.06 (0.84–1.34)     |
| Previous angina                 | 0.52   | 1.09 (0.84–1.43)     | 0.33   | 1.15 (0.86–1.54)     |
| ACE inhibitor or ARB therapy    | 0.14   | 0.80 (0.60–1.07)     | 0.37   | 0.86 (0.63–1.19)     |
| Statin therapy                  | 0.30   | 0.89 (0.71–1.11)     | 0.69   | 0.95 (0.74–1.22)     |
| Group 1                          | 0.02   |                       |        |                      |
| Group 2 vs 1                    | 0.01   | 1.26 (1.09–1.43)     | 0.02   | 1.24 (1.07–1.59)     |
| Group 3 vs 1                    | 0.42   | 0.86 (0.61–1.23)     | 0.32   | 0.82 (0.56–1.21)     |
| Group 4 vs 1                    | 0.01   | 1.37 (1.07–1.73)     | 0.02   | 1.37 (1.06–1.78)     |

ACE = angiotensin-converting enzyme, AMI = acute myocardial infarction, ARB = angiotensin-receptor blocker, BMI = body mass index, CI = confidence interval, MACE = major adverse cardiac event, STEMI = ST-elevation myocardial infarction.

been called “HDL dysfunction.” In the absence of inflammation, HDL has a complement of antioxidant enzymes that work to maintain an anti-inflammatory state. In the presence of systemic inflammation, such as in acute coronary syndrome, these antioxidant enzymes are inactivated and HDL accumulates oxidized lipids and proteins, making it proinflammatory. Other study with mendelian randomization has suggested that some genetic mechanisms that raise HDL-C plasma level do not reduce risk of CV events. Some another studies have suggested that measuring the quality and novel functions of HDL could provide an improved means of identifying subjects at increased risk for atherosclerotic events, compared with the current practice of only measuring HDL-C level. Other study with mendelian randomization has suggested that measuring the quality and novel functions of HDL could provide an improved means of identifying subjects at increased risk for atherosclerotic events, compared with the current practice of only measuring HDL-C level. The quality and function of HDL are attractive targets for emerging therapies. And the other recent studies presented that HDL subclass, and function of HDL are attractive targets for emerging therapies. In conclusion, lowering follow-up HDL-C level was an important prognostic factor for 1-year MACEs in patients with AMI.

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