

Lipid Testing and Statin Dosing After Acute Myocardial Infarction

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Background—The 2013 American College of Cardiology/American Heart Association cholesterol guidelines recommend high-intensity statins for patients after myocardial infarction (MI) rather than treating to a low-density lipoprotein cholesterol goal, as the previous ATP III (Adult Treatment Panel third report) guidelines had advised.

Methods and Results—To evaluate the frequency of postdischarge lipid testing and high-intensity statin use among MI patients discharged on a statin during the ATP III guidelines era, we linked ACTION (Acute Coronary Treatment and Intervention Outcomes Network) Registry data to Medicare claims for 11 046 MI patients aged ≥65 years who were discharged alive on a statin from 347 hospitals (2007–2009). Multivariable regression was used to evaluate the association between lipid testing and 1-year high-intensity statin use. Only 21% of MI patients were discharged on a high-intensity statin. By 90 days after MI, 44% of patients discharged on a statin underwent lipid testing (43% on low- or moderate-intensity statins and 49% on high-intensity statins; *P*=0.001). Follow-up lipid testing rates were 47% among patients with in-hospital low-density lipoprotein cholesterol ≥100 mg/dL and 47% among newly prescribed statin recipients. By 1 year, only 14% of patients were on high-intensity statins. Only 4% of patients discharged on low- or moderate-dose statin were uptitrated to high intensity; postdischarge lipid testing was associated with a slightly higher likelihood of high-intensity statin use by 1 year (5.4% versus 2.9%, adjusted odds ratio: 1.92; 95% confidence interval, 1.52–2.41).

Conclusions—Previous guidelines recommended low-density lipoprotein cholesterol goal-directed statin therapy, but lipid testing and high-intensity statin use were infrequent after MI. The American College of Cardiology/American Heart Association guidelines may promote more intensive cardiovascular risk reduction by eliminating treatment dependence on lipid testing. (*J Am Heart Assoc.* 2018;7:e006460. DOI: 10.1161/JAHA.117.006460.)

Key Words: lipid testing • myocardial infarction • statin dosing

ultiple primary^{1–6} and secondary prevention trials^{7–14} have consistently shown a strong, direct correlation between low-density lipoprotein cholesterol (LDL-C) level and atherosclerotic cardiovascular disease risk. As a result, the third report from the National Cholesterol Education Program Adult Treatment Panel (ATP III) promoted cardiovascular risk reduction via treatment to LDL-C targets.¹⁵ The ATP III

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guidelines recommended an LDL-C target of <100 mg/dL for patients with established coronary heart disease and a target of <70 mg/dL for acute coronary syndrome patients. Statins (3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors) are considered a first-line therapy to achieve such reductions in LDL-C levels; however, despite guideline recommendations, the majority of acute coronary syndrome patients failed to achieve their LDL-C goal of <70 mg/dL. 16

In 2013, the American College of Cardiology (ACC) and the American Heart Association (AHA) updated the cholesterol guidelines to recommend high-intensity statin therapy for patients with confirmed atherosclerotic cardiovascular disease, regardless of LDL-C levels, and particularly among those aged <75 years. ¹⁷ Nevertheless, some clinicians continue to advocate for an LDL-C goal-directed strategy to more aggressively reduce risk, and the 2016 European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) guidelines still call for treating patients to designated LDL-C targets. ¹⁸

We hypothesized that under the ATP III guidelines that pursued targeted LDL-C goals, patients were suboptimally treated after myocardial infarction (MI) because of low rates

Clinical Perspective

What Is New?

- Previous guidelines recommended low-density lipoprotein cholesterol goal-directed statin therapy, but lipid testing and high-intensity statin use were infrequent after myocardial infarction.
- High-intensity statin use was infrequently prescribed at discharge and use rates were even lower by 1 year after discharge. Low-density lipoprotein cholesterol levels may be suboptimally reduced in post—myocardial infarction patients as a result of these practices.

What Are the Clinical Implications?

 The 2013 American College of Cardiology/American Heart Association cholesterol guidelines promote more intensive cardiovascular risk reduction by eliminating treatment dependence on lipid testing after myocardial infarction.

of follow-up lipid testing and treatment intensification. The deemphasis of lipid targets in current guidelines may optimize secondary-prevention lipid lowering by removing the reliance of therapeutic titration on follow-up lipid test results. Consequently, we evaluated the frequency and temporal trends of postdischarge lipid testing and 1-year statin treatment intensity among MI patients who were discharged on a statin during the ATP III guidelines era.

Methods

The data, analytic methods, and study materials will not be made available to other researchers for purposes of reproducing the results or replicating the procedure.

Data Sources

The data source for this study was the National Cardiovascular Data Registry's ACTION (Acute Coronary Treatment and Intervention Outcomes Network) Registry, which was linked to the Centers for Medicare and Medicaid Services (CMS) claims database. The ACTION Registry is an ongoing quality-improvement registry that captures detailed clinical information on consecutive patients with a primary diagnosis of either ST-segment—elevation MI (STEMI) or non-STEMI who were treated at >300 participating hospitals across the United States. Using previously described methodology, ¹⁹ we used 5 indirect identifiers in combination (date of birth, sex, hospital identification, date of admission, date of discharge) to link patients from the ACTION Registry who were aged ≥65 years with their respective Medicare claims data. The linkage to CMS claims data provides information on postdischarge lipid

testing rates (Medicare Part B), as well as statin use and dosage (Medicare Part D). This data linkage was supported by a Centers for Education and Research on Therapeutics grant (U19HS021092) from the Agency for Healthcare Research and Quality. The Duke University Medical Center institutional review board granted a waiver of informed consent and authorization for this study.

Study Population

Our study population started with all patients aged ≥65 years in the linked database who were treated for STEMI or non-STEMI at hospitals participating in the ACTION Registry from July 1, 2007, to December 31, 2009, and enrolled in the Part D prescription coverage plan before discharge (n=23 029). We excluded patients who were not discharged to home (n=7809), who were not discharged on a statin (n=3140), or who did not survive to 90 days after discharge (n=552). We also excluded patients who were not enrolled in Medicare Part A (inpatient coverage), Part B (outpatient coverage), and Part D (prescription coverage) in the 90 days after discharge (n=160). Finally, patients were counted once, and subsequent MI hospitalizations in the ACTION Registry were excluded (n=322). After these exclusions, the final analysis population comprised 11 046 patients from 347 United States hospitals.

Data Definitions

Postdischarge lipid testing was ascertained from Medicare Part B carrier files and outpatient revenue center file line items and was defined as any of the following Current Procedural Terminology codes occurring within 90 days after the index MI discharge: 80061, 82465, 83700, 83701, 83704, 93718, 83 721, and 84 478. As expected with administrative claims data, the presence of these codes indicates that the test was performed, but postdischarge laboratory test results are unknown. In-hospital LDL-C levels were captured in the ACTION Registry data collection form. Statin use before the index MI hospitalization was also captured in ACTION Registry data. Postdischarge statin type and dose were ascertained from Medicare Part D prescription fill data. High-intensity statin was defined as atorvastatin ≥40 mg daily or rosuvastatin ≥20 mg daily; moderateintensity statin was defined as a daily dose of atorvastatin 10 to 39 mg, rosuvastatin 5 to 19 mg, simvastatin ≥20 mg, pravastatin ≥40 mg, lovastatin ≥40 mg, fluvastatin ≥80 mg, or pitavastatin ≥2 mg. All other statin type-dose combinations were considered low intensity. Statin use at 1 year was defined based on the dose and supply of the most recent refill before day 365 (with a 7-day grace period) among the 10 099 patients alive at 1 year with continued Part D coverage.

Statistical Analyses

We summarized rates of lipid testing within 90 days after discharge over time and the variation in 90-day testing rates at the hospital level among hospitals with at least 20 patients discharged on a statin. Patients were divided into those with and without follow-up testing within 90 days; patient characteristics, including medical history, risk factors, and in-hospital treatment, were described in the 2 groups. Categorical variables were presented as frequencies (percentages), and continuous variables were presented as median (interquartile range). We also examined rates of lipid testing among patients grouped by age, in-hospital LDL-C levels, statin therapy use before the index MI, and statin intensity at discharge. ¹⁷ Rates of high-intensity statin use at discharge were compared between age groups using a Pearson chi-square test.

A multivariable logistic regression model was used to determine which patient and hospital factors are independently associated with follow-up lipid testing. Variables entered into the model included demographics (age, sex, race), medical history (current/recent tobacco use, hypertension, dyslipidemia, dialysis status, diabetes mellitus, prior MI, prior heart failure, prior percutaneous coronary intervention, prior coronary artery bypass grafting, prior stroke, peripheral arterial disease, Charlson comorbidity index), presentation (STEMI versus non-STEMI, signs of heart failure on arrival, shock on arrival, heart rate on admission, systolic blood pressure on admission, body mass index), statin use before admission, time from arrival to catheterization, in-hospital LDL-C, in-hospital creatinine clearance (Cockcroft-Gault), discharge medications (angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, P2Y₁₂ inhibitors, beta blockers, statin intensity, ezetimibe), hospital characteristics (geographic region, teaching status, total hospital beds, urban or rural location), and community characteristics (median household income, percentage that completed high school, urban or rural location, unemployment rate). Rates of missing data were <1% for all variables except body mass index (1.4% missing), and missing values were imputed using the sex- and STEMI-specific median for patient-level covariates and the overall median for hospital- or community-level covariates. A hierarchical modeling approach was used, with patients clustered within hospitals and hospitals treated as a random effect. This approach considers that a patient's probability of postdischarge lipid testing may be dependent on the hospital at which he or she received care. Hospital effect was assessed with a median odds ratio (OR), which is the median of the distribution of all possible comparisons (ORs) of patients from different hospitals. Because the patient with higher odds is always in the numerator, the median OR is always ≥1, and a larger median OR indicates more betweenhospital variability, 20 suggesting that the treating hospital contributes to individual patient probability of having a lipid test performed. The final model was selected using forward stepwise selection. For variables not included in the final model, significance tests were generated by adding them one at a time to the final model. In addition, a multivariable regression model was used to examine the association of 90-day lipid testing with 1-year statin use and intensity among patients still alive and enrolled in Medicare Part D at 1 year. Variables included in the model were the same as above with the addition of an interaction term between statin intensity at discharge and 90-day lipid testing.

Based on patients' in-hospital LDL-C levels and the intensity of statin therapy that they were prescribed at discharge, we estimated the proportion of patients who would reach the guideline-recommended target of <70 mg/dL by 1 year after discharge, assuming that high-intensity statins decrease LDL-C levels by 50%, moderate-intensity statins decrease LDL-C by 30%, and low-intensity statins decrease LDL-C by 15%. ¹⁷ In addition, we estimated the proportion of patients who would reach the target of <70 mg if all patients were prescribed high-intensity statins at discharge. Finally, although linked CMS data were not available beyond 2014, we presented discharge high-intensity statin use rates among MI patients aged ≥65 years discharged alive in 2014, which was the year after the ACC/AHA guidelines were released.

All analyses were conducted at the Duke Clinical Research Institute using SAS version 9.4 or higher (SAS Institute).

Results

Patterns of Postdischarge Lipid Testing

Among 11 046 MI patients discharged on a statin between July 2007 and December 2009, only 21% were discharged on a high-intensity statin. This rate varied only slightly by patient age; high-intensity statins were used in 24% of patients aged <75 years and in 18% of patients aged ≥ 75 years (P<0.0001).

Lipid testing was performed in 44% of patients within 90 days after discharge. The rate of follow-up lipid testing remained relatively unchanged through our study period (Figure 1). The proportion of patients with lipid testing by 90 days varied across hospitals, ranging between 20% and 70% of discharged patients with a median testing rate of 45% (Figure 2).

Characteristics Associated With Postdischarge Lipid Testing

When we compared patients with and without follow-up lipid testing, no major differences were observed in the location or type of hospitals from which the patients were discharged

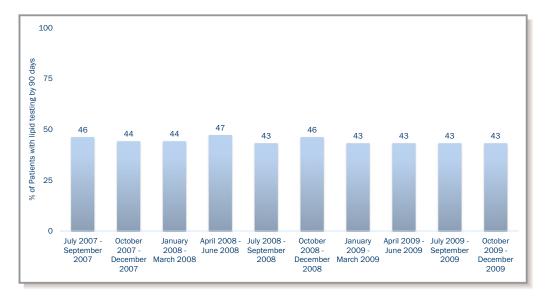


Figure 1. Trend in lipid testing within 90 days after myocardial infarction in the United States from 2007 to 2009.

(rural hospital: 15% versus 14%; teaching hospital: 28% versus 29%; median bed size: 418 versus 418; *P*>0.05 for all). Similarly, we did not observe major differences in the age, sex, race, or socioeconomic status of patients with and without follow-up lipid testing (Table). Patients with follow-up lipid testing were more likely to present with STEMI and less likely to have a history of MI, heart failure, percutaneous coronary intervention, coronary artery bypass grafting, atrial fibrillation/flutter, stroke, or peripheral arterial disease; these patients were as likely to get lipids measured during the initial hospitalization as patients who did not have follow-up testing.

In the multivariable regression model, patient characteristics associated with a greater likelihood of follow-up lipid testing included prior history of dyslipidemia and a high-intensity statin prescribed at discharge, as well as higher body mass index and higher household income (Figure 3). Statin use before admission was associated with a lower likelihood

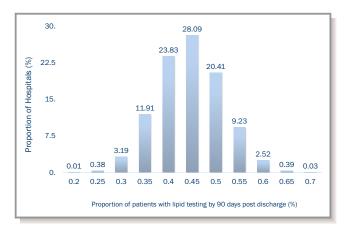


Figure 2. Rates of lipid testing in US hospitals from 2007 to 2009.

of follow-up lipid testing. In addition, older patients, those of nonwhite race, and current/recent smokers were less likely to receive follow-up lipid testing. Patients' probability of undergoing postdischarge lipid testing was also significantly dependent on the hospital at which they were cared for during their index MI (median OR: 1.28; 95% confidence interval, 1.21–1.36).

Patients had a median in-hospital LDL-C level of 90 mg/dL (25th and 75th percentiles: 68 and 115, respectively), and 39% (n=3320) of patients had an in-hospital LDL-C ≥100 mg/ dL. Rates of follow-up lipid testing did not vary significantly based on in-hospital LDL-C levels (47% of patients with inhospital LDL-C ≥100 mg/dL versus 43% of patients with LDL-C <100 mg/dL, P=0.19 when added to the multivariable model). Among patients aged <75 years, the rates of followup testing were 51% in those with in-hospital LDL-C ≥100 mg/dL and 45% in those with LDL-C <100 mg/dL. Rates of follow-up testing were modestly higher among patients newly prescribed a statin compared with those already on a statin before their MI (47% versus 41%, P=0.004 from multivariable model). At discharge, 21% (n=2315) of patients were prescribed a high-intensity statin; follow-up lipid testing was more common among high-intensity statin users than those discharged on lower intensity statins (49% versus 43%, P=0.0005 from multivariable model). Lipid testing rates were 51% versus 47% among patients aged <75 years prescribed high- versus lower intensity statins at discharge.

Postdischarge Lipid Testing and Subsequent Treatment Changes

Rates of overall and high-intensity statin use at 1 year are shown in Figure 4. Patients who had received lipid testing

Table. Patient Characteristics

	Lipid Testing Within 90 Days (n=4884)	No Lipid Testing Within 90 Days (n=6162)
Patient demographics		
Age, y, median (IQR)	74 (69–80)	75 (70–82)
Female, %	49.3	51.9
White race, %	88.9	86.9
Household income (×\$1000), median (IQR)*	45 (40–53)	45 (40–52)
Unemployment rate, %*	6.6	6.8
College degree, %*	23.4	23.4
Medical history		2
Current/recent smoker, %	17.3	18.3
Hypertension, %	78.3	81.5
Dyslipidemia, %	64.8	65.1
Currently on dialysis, %	2.9	2.2
Chronic lung disease, %	23.1	28.3
Diabetes mellitus, %	33.3	34.2
Prior MI, %	23.3	30.1
Prior HF, %	11.2	16.9
Prior CABG, %	16.0	22.1
Prior PCI, %	21.1	27.3
Atrial fibrillation/flutter, %	18.2	21.4
Prior stroke, %	8.5	11.1
Peripheral arterial disease, %	11.9	14.1
Charlson comorbidity index, median (IQR)	2.0 (1.0–4.0)	3.0 (2.0–4.0)
Presentation		
STEMI	36.1	29.7
Signs of HF	15.1	20.2
Cardiogenic shock	2.1	2.0
HR on admission, bpm, median (IQR)	79 (66–93)	80 (68–96)
SBP on admission, mm Hg, median (IQR)	145 (125–165)	144 (124–165)
BMI, kg/m ² , median (IQR)	27.6 (24.5–31.2)	27.1 (23.8–31.1)
Measurements and labs		
Creatinine, mg/dL, median (IQR)	1.1 (0.9–1.3)	1.1 (0.9–1.4)
Hemoglobin, g/dL, median (IQR)	13.5 (12.2–14.67)	13.2 (11.9–14.4)
Lipids measured in hospital, %	79.4	78.9
In-hospital LDL-C, mg/dL, median (IQR)	92 (70–117)	87 (66–113)

Continued

Table. Continued

	Lipid Testing Within 90 Days (n=4884)	No Lipid Testing Within 90 Days (n=6162)	
Discharge medications			
ACEI or ARB	74.9	74.6	
Beta blocker	97.2	96.0	
High-intensity statin	23.4	19.1	

All variables in this table missing in <1% of patients except for BMI, which was missing in 1.4% of patients. ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; CABG, coronary artery bypass grafting; HF, heart failure; HR, heart rate; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; MI, myocardial infarction; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; STEMI, ST-segment-elevation myocardial infarction

within 90 days after discharge were more likely to remain on a statin at 1 year (adjusted OR: 1.17; 95% confidence interval, 1.07-1.29), regardless of whether they were discharged on a high-intensity statin or a lower intensity statin (P_{interaction}=0.29). Among patients discharged on highintensity statins, 51.4% remained on a high-intensity statin by 1 year. For patients prescribed a high-intensity statin at discharge, lipid testing within 90 days was not associated with remaining on a high-intensity statin at 1 year (adjusted OR: 1.07; 95% confidence interval, 0.90–1.28). Among patients who were discharged on a low- or moderateintensity statin, those who received lipid testing within 90 days were nearly twice as likely to be titrated up to a high-intensity statin at 1 year compared with those who did not receive lipid testing (adjusted OR: 1.92; 95% confidence interval, 1.52-2.41), but the rate of high-intensity statin use was still very low (5.4%) in the lipid-tested group.

Guidelines during this study period recommended an LDL goal <70 mg/dL for post-MI patients. Although administrative claims data show performance of lipid testing, measured lipid levels are not available. Based on the patients' in-hospital LDL-C levels and discharge statin therapy intensity and on the assumption that high-intensity statins decrease LDL-C levels by 50%, moderate-intensity statins decrease LDL-C by 30%, and low-intensity statins decrease LDL-C by 15%, 17 we estimate that only \approx 66% of the post-MI patients in our study population would reach LDL-C levels <70 mg/dL. Among patients who were discharged on moderate- and low-intensity statins, 37% and 57%, respectively, would remain undertreated with estimated LDL-C ≥70 mg/dL (Figure 5). The 2013 ACC/AHA guidelines favor consideration of highintensity statin for secondary prevention. If all patients discharged on low- or moderate-intensity statin in our study were instead given high-intensity statins, we estimate that 90% of all MI patients would reach LDL-C <70 mg/dL.

^{*}Census data based on patient ZIP code of residence.

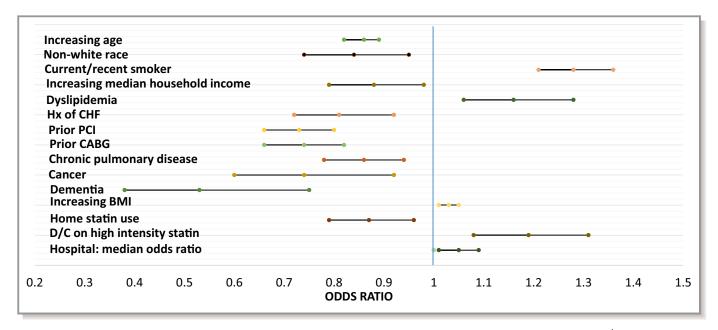


Figure 3. Characteristics associated with postdischarge lipid testing. *For each 5-year increase above age 74 years. [†]For each \$10 000 increase. [‡]For each 1-point increase. [§]The median OR is the median of the distribution of ORs for all possible pair of people with similar covariates but treated at different hospitals. The median OR allows examination of the magnitude of hospital effect relative to the other factors in the multivariable model. BMI indicates body mass index; CABG, coronary artery bypass grafting; CHF, congestive heart failure; CI, confidence interval; D/C, discharged; Hx, history; OR, odds ratio; PCI, percutaneous coronary intervention.

Among patients aged <75 years, an estimated 64% would potentially reach LDL-C levels <70 mg/dL based on inhospital LDL-C levels and discharge statin intensity; 57% and 40% of patients discharged on low- and moderate-intensity statin, respectively, would remain undertreated with estimated LDL-C \geq 70 mg/dL. If these patients were instead given high-intensity statins, we estimate that 90% would reach LDL-C <70 mg/dL. Among MI patients aged \geq 65 years discharged in 2014 in the ACTION Registry, the rate of high-intensity statins prescribed at discharge had increased to 49%; among patients aged between 65 and 75 years, the rate of high-intensity statin use was 54%.

Discussion

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Previous guidelines recommended that statins be dosed for individual patients based on measured LDL-C levels, with the goal of lowering LDL-C to <70 mg/dL after acute MI. In this national study, we showed that this strategy likely failed to optimally lower LDL-C because follow-up lipid testing was performed in only a minority (44%) of post-MI patients, and only 1 in 7 patients was treated with a high-intensity statin at 1 year. Among patients discharged on low- or moderate-intensity statin, follow-up lipid testing was associated with a greater likelihood of high-intensity statin use at 1 year after discharge. If high-intensity statin was initiated in all MI patients at discharge, per current guideline recommendations,

we estimate that a greater proportion of patients would reach LDL-C <70 mg/dL.

Any LDL-C goal-based strategy for lipid-lowering therapy must necessarily rely on lipid testing. Under the ATP III guidelines, lipid testing rates remained consistently in the 40% range, and we did not see evidence that testing was used selectively for patients with greater need of statin uptitration. Even among patients with in-hospital LDL-C levels above conservative guideline-recommended goals (LDL-C≥100 mg/ dL) who would benefit from lipid testing to ensure treatment target reached, less than half (47%) received lipid testing within 90 days following hospital discharge. Similarly, testing rates were low for patients with newly prescribed statin therapy (43%) and among patients discharged on low- or moderate-intensity statins (47%)—groups that may be expected to require active testing and statin intensification. Surprisingly, patients discharged on a high-intensity statin were more likely to undergo lipid testing. Older patients and those with clinical comorbidities were less likely to undergo lipid testing; these may be patients for whom statin dose titration is not a high priority. In addition, we showed that the likelihood of postdischarge lipid testing varies significantly by the hospital at which patients received index MI treatment, suggesting that hospital discharge practices and the coordination of follow-up care within or between healthcare systems may be a driver of postdischarge lipid testing, rather than patient factors. The transition from the hospital to home

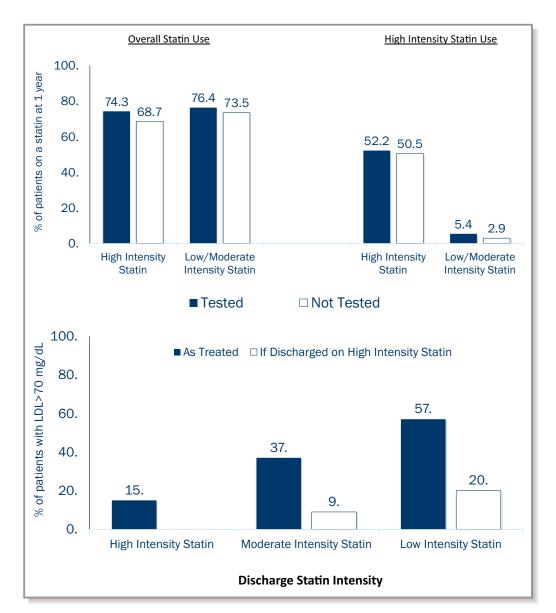


Figure 4. Overall and high-intensity statin use at 1 year after discharge. Overall and high-intensity statin use at 1 year after discharge among patients with and without lipid testing within 90 days.

setting has been identified as an important period that may influence patients' downstream care and risk of adverse cardiovascular events.²¹

Our study shows that the prior LDL-C goal-directed strategy did not yield as high of a proportion of patients on high-intensity statin therapy. By 1 year after MI, only 14% of patients were treated with a high-intensity statin. Of the 79% of post-MI patients discharged on low- or moderate-intensity statin, those with follow-up lipid testing were more likely to be uptitrated than those who did not, but overall, only 4% of these patients were uptitrated to a higher intensity statin by 1 year after MI. Based on in-hospital LDL-C levels and intensity of statins prescribed at discharge, we project that only $\approx\!66\%$ of all patients discharged on a statin in our study population would reach the guideline-recommended LDL-C

goal of <70 mg/dL. The actual rate of achieving LDL-C <70 mg/dL may be lower than this estimate, as this estimate is calculated based on in-hospital LDL-C levels, which are lower during an acute MI.²² Our results suggest that suboptimal lipid-lowering in post-MI patients can be attributed (1) to low use of high-intensity statin at discharge, despite trial evidence suggesting benefit^{7,8,11,23,24}; (2) to underutilization of follow-up lipid testing to guide statin dose titration; and (3) to inertia in uptitrating statin dose to achieve guideline-recommended LDL-C, even when lipid testing was performed.

Targeted lipid management is a multistep process in which the post-MI patient discharged on a statin is required to have their lipid levels followed after a few months of treatment. If LDL-C levels have not reached guideline-recommended targets, then statin intensification would be considered. The

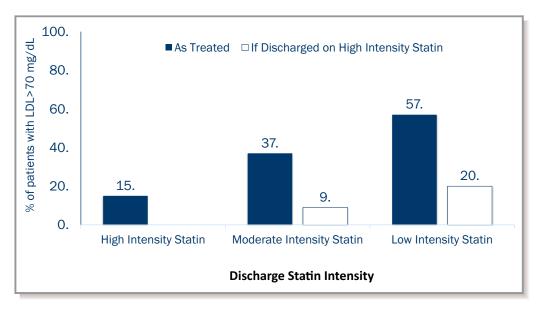


Figure 5. Projected proportion of patients failing to reach guideline-recommended LDL-C <70 mg/dL based on in-hospital LDL-C level and statin intensity prescribed at discharge. *Under the assumption that high-intensity statins decrease LDL-C levels by 50%, moderate-intensity statins decrease LDL-C by 30%, and low-intensity statins decrease LDL-C by 15%. ¹⁸ LDL-C indicates low-density lipoprotein cholesterol.

cycle of lipid remeasurement and dose titration is repeated until the LDL-C goal is reached, which introduces barriers or delays to treatment intensification. In shifting toward a strategy of treating post-MI patient populations uniformly with high-intensity statins, the ACC/AHA guidelines effectively replaced the prior paradigm of tailoring statin dosing to target LDL-C level goals, thereby reducing the reliance of dose titration on follow-up lipid testing. These current guidelines may promote a more aggressive approach to lipid management and cardiovascular risk reduction; our study data project that implementation of these guidelines could substantially raise the likelihood of reaching LDL-C <70 mg/ dL. This simplified treatment strategy also streamlines the transition-of-care process after hospital discharge and reduces potential therapeutic inertia in responding to lipid test results. Examination of MI patients in the year after the 2013 ACC/ AHA guidelines already showed an increase in high-intensity statin use at discharge. Of course, patients discharged on a high-intensity statin may not necessarily continue on a highintensity statin long term. However, prior studies have shown that patients are more likely to be adherent to therapies when these therapies are prescribed at discharge.²⁵

Some clinicians view the current guidelines as a sledge-hammer approach to a nuanced problem. The 2016 ESC/EAS cholesterol guidelines ¹⁸ have inspired new momentum to return to pursuing LDL-C goals, advocating >50% LDL-C reduction for patients with high or very high cardiovascular disease risk. Arguably, the role of lipid-level monitoring extends beyond simply assessing progress toward an LDL-C goal. Regular lipid measurements may help ascertain patient

medication adherence, monitor safety of statin treatment, and offer an opportunity to actively educate and engage post-MI patients in risk factor modification behaviors. Early and routine follow-up outpatient visits, as required with lipid testing, have been associated with improved adherence to lipid-lowering therapy.^{26,27} Recent trials have emerged that suggest improved patient outcomes with targeted lipid management. Results from IMPROVE-IT (Improved Reduction of Outcomes: Vytorin Efficacy International Trial) demonstrate significant reductions in cardiovascular events when ezetimibe was added to statin therapy to lower LDL-C levels below previous targets.²⁸ Novel lipid-lowering therapies, such as PCSK9 inhibitors, have shown substantial LDL-C-lowering capability, with imminent outcomes trial results.²⁹⁻³¹ How these data will influence future guideline updates is unknown, but our results encourage reengineering of clinical processes if we return to a lipid-level-driven treatment strategy, because only a minority of post-MI patients actually completed followup lipid testing in routine clinical practice during the time period when this follow-up was strongly recommended by national guidelines, and the association between lipid testing and therapy intensification was significant but modest.

Study Limitations

This study should be interpreted in the context of a few limitations. First, although the ACTION Registry provides data on LDL-C values from in-hospital lipid testing, the linked administrative data can only assess whether postdischarge lipid tests were performed; they do not provide follow-up lipid

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test results. We used in-hospital LDL-C values to project the proportion of patients with controlled LDL-C levels based on the dose of statin prescribed at discharge; however, this approach may overestimate the proportion of patients with LDL-C <70 mg/dL, because in-hospital LDL-C values may be depressed when measured in the setting of acute coronary syndrome. Second, because statin type and dosing are gathered from Medicare data, our study population was limited to patients aged ≥65 years, and results cannot be generalized to younger patients. Third, the ACTION Registry may represent hospitals with interest in quality improvement and may not be representative of all practices in the United States. Fourth, as in any observational study, there is the potential for unmeasured confounding factors. We cannot causally link patients who had received lipid testing to the use of statins or high-intensity statins downstream. Finally, this analysis examined a study period before the consideration and dissemination of guidelines that changed our lipid management strategy. More recent Medicare data are not currently available to examine lipid testing practices and postdischarge statin use patterns after implementation of the 2013 ACC/AHA guidelines.

Conclusion

Although previous guidelines recommended lipid-lowering targets, follow-up lipid measurements were done in only a minority of post-MI patients in routine clinical practice. High-intensity statin use was infrequently prescribed at discharge, and use rates were even lower by 1 year after discharge. LDL-C levels may be suboptimally reduced in post-MI patients as a result of these practices. The 2013 ACC/AHA cholesterol guidelines promote more intensive cardiovascular risk reduction by eliminating treatment dependence on post-MI lipid testing.

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Disclosures

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