Temporal trends, determinants, and impact of high-intensity statin prescriptions after percutaneous coronary intervention: Results from a large single-center prospective registry

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Background High-intensity statins (HIS) are recommended for secondary prevention following percutaneous coronary intervention (PCI). We aimed to describe temporal trends and determinants of HIS prescriptions after PCI in a usual-care setting.

Methods All patients with age ≤75 years undergoing PCI between January 2011 and May 2016 at an urban, tertiary care center and discharged with available statin dosage data were included. HIS were defined as atorvastatin 40 or 80 mg, rosuvastatin 20 or 40 mg, and simvastatin 80 mg.

Results A total of 10,495 consecutive patients were included. Prevalence of HIS prescriptions nearly doubled from 36.6% in 2011 to 60.9% in 2016 ($P < .001$), with a stepwise increase each year after 2013. Predictors of HIS prescriptions included ST-segment elevation myocardial infarction/non–ST-segment elevation myocardial infarction (odds ratio [OR] 4.60, 95% CI 3.98-5.32, $P < .001$) and unstable angina (OR 1.31, 95% CI 1.19-1.45, $P < .001$) as index event, prior myocardial infarction (OR 1.48, 95% CI 1.34-1.65, $P < .001$), and co-prescription of β-blocker (OR 1.26, 95% CI 1.12-1.43, $P < .001$). Conversely, statin treatment at baseline (OR 0.86, 95% CI 0.77-0.96, $P = .006$), Asian races (OR 0.73, 95% CI 0.65-0.83, $P < .001$), and older age (OR 0.90, 95% CI 0.88-0.92, $P < .001$) were associated with reduced HIS prescriptions. There was no significant association between HIS prescriptions and 1-year rates of death, myocardial infarction, or target-vessel revascularization (adjusted hazard ratio 0.98, 95% CI 0.84-1.15, $P = .84$), although there was a trend toward reduced mortality (adjusted hazard ratio 0.71, 95% CI 0.50-1.00, $P = .05$).

Conclusion Although the rate of HIS prescriptions after PCI has increased in recent years, important heterogeneity remains and should be addressed to improve practices in patients undergoing PCI. (Am Heart J 2019;207:10-18.)

The 2013 American Heart Association/American College of Cardiology (AHA/ACC) guidelines on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults recommend initiating or continuing high-intensity statins (HIS) for patients with clinical atherosclerotic cardiovascular disease who are ≤75 years of age.[1] Consequently, using Medicare and commercial health insurance data, increased HIS prescriptions following hospitalization for myocardial infarction (MI) were reported in the time period following the release of the guidelines.[2,3] Yet, persistent underutilization of HIS remains in the United States.[4,5] In a large registry of 138 cardiology and endocrinology practices in 2015, only 47.3% of patients with atherosclerotic cardiovascular disease were treated with an appropriate statin intensity level for secondary prevention, underscoring the need for additional efforts in this arena.[6]

Currently, the extent of the implementation of the 2013 AHA/ACC guidelines among patients undergoing

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percutaneous coronary intervention (PCI) remains unclear. Determining predictors of HIS prescriptions in usual-care settings could help to improve medical practices by identifying undertreated categories of patients. We therefore aimed to describe temporal trends, determinants, and the clinical impact of HIS prescriptions after PCI with data from a large institutional database reflecting a real-world setting.

Materials and methods

Population

All consecutive patients aged ≤75 years who underwent PCI with balloon angioplasty and/or stent implantation from January 2011 to May 2016 at our center with information available on statin prescriptions at discharge were included in the present study. Patients aged >75 years are the subject of differing and specific recommendations by the 2013 AHA/ACC guidelines and thus were not included in the present analysis. Baseline and procedural characteristics as well as information concerning the lipid-lowering drug(s) prescribed at hospital discharge were obtained through a review of medical records contained in the local PCI registry. Trained research coordinators obtained 1-year outcomes data through clinical visits or telephone follow-up. End points of interest were the composite of all-cause mortality, MI according to the third universal definition,[7] or target-vessel revascularization (TVR), as well as each component within 1 year of the index procedure. Statin intensity was defined according to the definitions used in the AHA/ACC guidelines.[1] High-intensity statins included atorvastatin 40-80 mg, rosuvastatin 20-40 mg, and simvastatin 80 mg. Moderate-intensity statins were defined as atorvastatin 10-20 mg, rosuvastatin 5-10 mg, simvastatin 20-40 mg, pravastatin 40-80 mg; lovastatin 40 mg, fluvastatin 40 mg bid, fluvastatin XL 80 mg, and pitavastatin 2-4 mg. Finally, low-intensity statin was defined as simvastatin 10 mg, pravastatin 10-20 mg, lovastatin 20 mg, fluvastatin 20-40 mg, and pitavastatin 1 mg. The present study was supported by a research grant by Regeneron Pharmaceutical; however, the authors independently performed and are solely responsible for the design and conduct of this study, all study analyses, the drafting and editing of the paper, and its final contents.

Statistical analysis

Descriptive statistics are reported as mean ± SD, median and interquartile range, or number and percentage when appropriate. The χ^2 test was used to compare differences between categorical variables. The independent-samples *t* test was used to compare continuous variables with normal distribution, and the Mann-Whitney test was used to compare continuous variables without a normal distribution. The independent association between baseline and procedural characteristics and HIS prescriptions at hospital discharge was investigated with multivariate logistic regression. Year of index PCI was analyzed with 2011 as the year of reference. Concerning clinical presentation, ST-segment elevation MI (STEMI), non–ST-segment elevation MI (NSTEMI), and unstable angina were compared to stable angina and asymptomatic presentation. Finally, white race was used as the reference when evaluating the impact of race or ethnicities on HIS prescriptions. Multivariate Cox regression model was constructed to investigate the impact of HIS prescriptions on 1-year end points using the following covariates: age, sex, white ethnicity, diabetes mellitus, history of hyperlipidemia or systemic hypertension, smoking status, body mass index, *chronic kidney disease* (defined as estimated glomerular filtration rate <60 mL/min), clinical presentation, prescription of β-blocker at discharge, chronic total occlusion, presence of severe calcification, AHA/ACC lesion classification B2/C, bundle-branch block at electrocardiogram, baseline hemoglobin, and C-reactive protein level at baseline.

Results

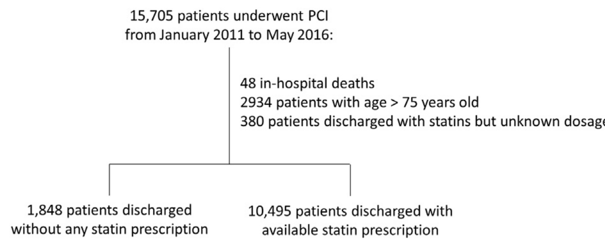
Temporal trend in the prescriptions of high-intensity statins

Between January 2011 and May 2016, 15,705 patients underwent PCI in our center and were screened for inclusion (Figure 1). A total of 1,848 (11.8%) patients overall were discharged without any statin prescription, with a decrease in proportion of patients per year from 35.4% in 2011 to 8.8% in 2016 ($P < .001$) (Figure 2). A total of 10,495 (66.8%) patients with age ≤75 years were discharged with an available statin prescription and therefore included in the analysis. Patients discharged with HIS prescriptions were younger, were more frequently male, were more often hospitalized for an acute coronary syndrome, had higher baseline low-density lipoprotein cholesterol (LDL-C), and had more complex coronary disease (Table I). Overall, rates of high-, moderate-, and low-intensity statin prescriptions at discharge were 4,373 (41.7%), 5,444 (51.9%), and 678 (6.4%), respectively, with statistically significant variations over the study period (Figure 3). Particularly, the prevalence of HIS prescriptions in our cohort nearly doubled from 36.6% in 2011 to 60.9% in 2016 ($P < .001$). Conversely, the concomitant use of other lipid-lowering agents such as niacin or ezetimibe steadily decreased over this period from 101 patients (5.3%) in 2011 to 3 (0.6%) in 2016 ($P < .001$).

Determinants of high-intensity statin prescriptions at discharge

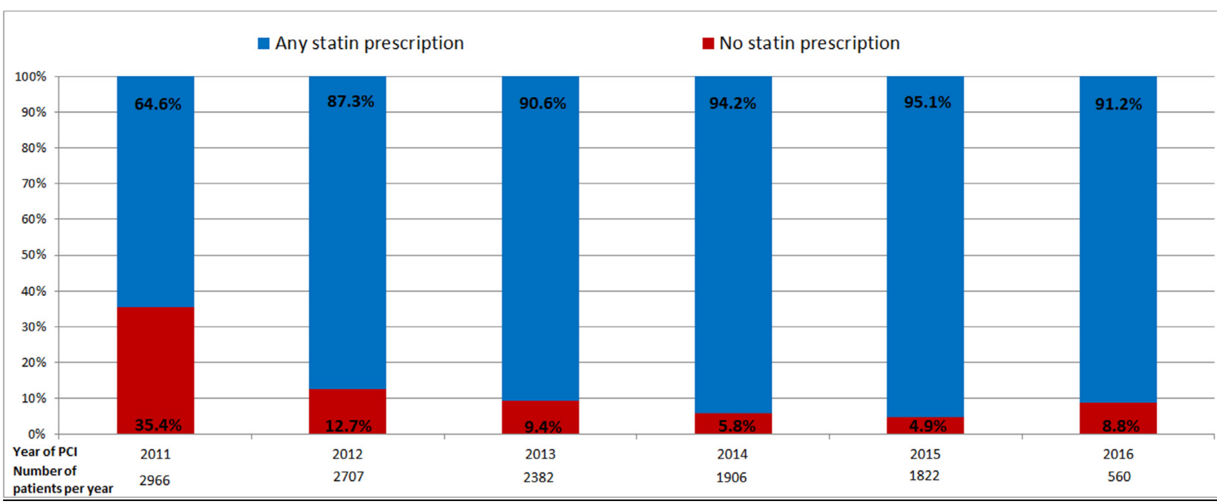
In the multivariate model, there was a significant association between the year of procedure and HIS prescription, with a stepwise increase from 2014 to 2016 compared to 2011 (Table II). A history of prior MI, clinical presentation with STEMI/NSTEMI and unstable angina,

Figure 1



Flowchart.

Figure 2



PCI: percutaneous coronary intervention

Temporal evolution of statin prescriptions following PIC.

higher LDL-C level at baseline, longer total stent length, and concomitant β-blocker prescription at discharge were independently associated with more frequent HIS prescriptions. Conversely, Asian race, older age, and statin treatment at baseline were independently associated with reduced HIS prescriptions at time of discharge.

One-year clinical outcome

Kaplan-Meier estimates of 1-year event rates and survival curves are detailed in Table III and Figure 4. In univariate analysis, the rate of MI within 1 year of the index procedure was significantly higher among patients with HIS prescription at discharge compared to those who were not discharged on HIS. Independent predictors of 1-year major adverse cardiovascular event and its individual components are shown in Table IV. In multivariate analysis, there was no significant association between HIS prescriptions and the composite end point of death, MI, or TVR as well as the individual components, although there was a trend toward reduced mortality. Of note, there was no significant interaction between LDL-C level at baseline and the intensity of statins prescription at discharge on any study end points.

Discussion

In the present analysis comprising 10,495 patients undergoing PCI, we demonstrated a gradual increase in the prescription of HIS at discharge following the publication of the 2013 AHA/ACC guideline on treatment of high blood cholesterol. Despite this improvement in guideline adherence, approximately 40% of patients continue to receive low- or moderate-intensity statins after PCI. Several demographic and clinical

Table I. Baseline and procedural characteristics

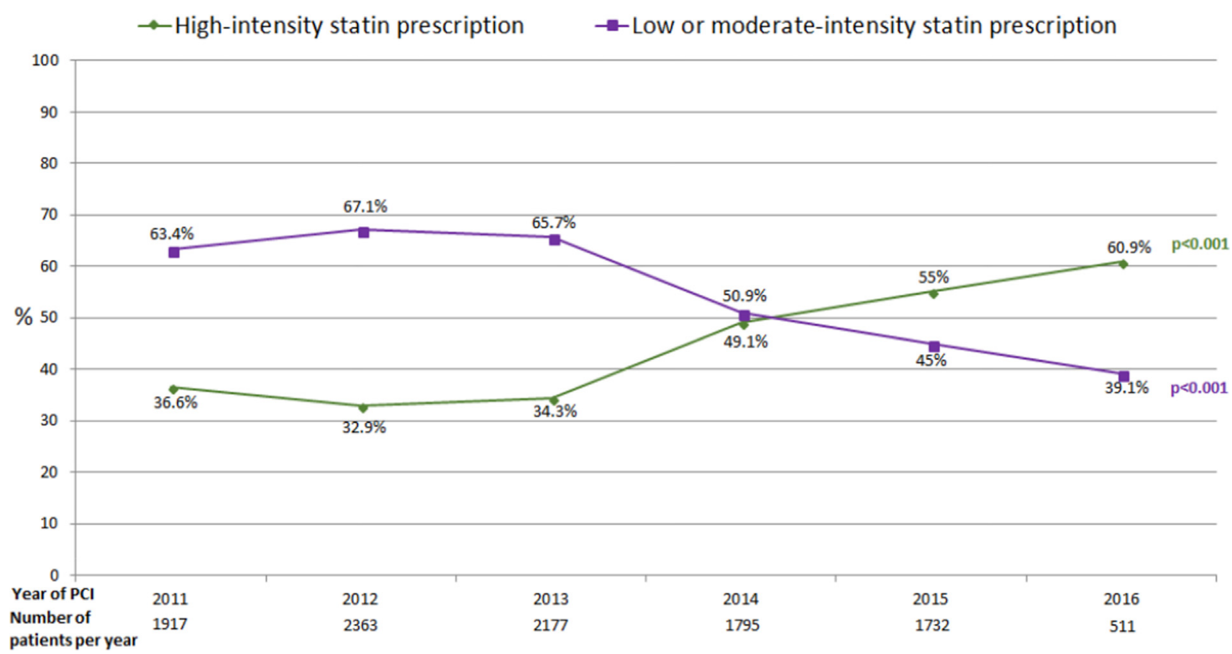
	Overall population (N = 10,495)	High-intensity statin at discharge (n = 4373)	Non–high-intensity statins at discharge (n = 6122)	*P* value
Baseline characteristics				
Age (y)	61.6 ± 9	60.4 ± 9.5	62.4 ± 8.6	<.001
Ethnicity				<.001
White	4045 (38.5%)	1751 (40.0%)	2294 (37.5%)	
Asian	1912 (18.2%)	7141 (6.3%)	1198 (19.6%)	
Black	1196 (11.4%)	512 (11.7%)	684 (11.2%)	
Hispanic	2112 (20.1%)	942 (21.5%)	1170 (19.1%)	
Other	1230 (11.7%)	454 (10.4%)	776 (12.7%)	
Male gender	7633(72.7%)	3228 (73.8%)	4405 (72.0%)	.04
BMI (kg/m^2)	29.5 ± 7.3	29.6 ± 7.8	29.4 ± 6.9	.12
Hyperlipidemia	10,008 (95.4%)	4066 (93.0%)	5942 (97.1%)	<.001
Hypertension	9696 (92.4%)	3943 (90.2%)	5753 (94.0%)	<.001
Diabetes mellitus	5266 (50.2%)	2091 (47.8%)	3175 (51.9%)	<.001
Insulin dependent	1634 (15.6%)	688 (32.9%)	946 (29.8%)	.02
Chronic kidney disease	2341 (23.5%)	909 (22.1%)	1432 (24.5%)	.05
Current smoker	1847 (17.6%)	874 (20.0%)	973 (15.9%)	<.001
LVEF (%)	53.5 ± 12.1	52.3 ± 12.6	54.3 ± 11.6	<.001
Medical history				
Previous myocardial infarction	2488 (23.7%)	1185 (27.1%)	1303 (21.3%)	<.001
Previous CABG	1570 (15.0%)	674 (15.4%)	896 (14.6%)	.27
Peripheric artery disease	915 (8.7%)	389 (8.9%)	526 (8.6%)	.58
Cerebrovascular disease	946 (9.0%)	386 (8.8%)	560 (9.1%)	.57
Chronic lung disease	805 (7.7%)	321 (7.3%)	484 (7.9%)	.28
Clinical presentation				
Asymptomatic	48 (0.6%)	165 (3.8%)	315 (5.1%)	<.001
Stable angina	5001 (47.7%)	1763 (40.3%)	3238 (52.9%)	<.001
Unstable angina	3605 (34.3%)	1435 (32.8%)	2170 (35.4%)	.005
NSTEMI	1099 (10.5%)	748 (17.1%)	35 (15.7%)	<.001
STEMI	292 (2.8%)	256 (5.9%)	36 (0.6%)	<.001
Medications at admission				
Aspirin	9056 (86.3%)	3743 (85.6%)	5313 (86.8%)	.08
Statin	8233 (78.4%)	3288 (75.2%)	4945 (80.8%)	<.001
β-Blockers	7732 (73.7%)	3154 (72.1%)	4578 (74.8%)	.002
Medications at discharge				
Aspirin	10,268 (97.8%)	4288 (98.1%)	5980 (97.7%)	0.19
Statin	10,495 (100.0%)	4373 (100.0%)*	6122 (100.0%)	
Ezetimibe	243 (2.3%)	83 (1.9%)	160 (2.6%)	.016
Niacin	13 (0.1%)	0	13 (0.2%)	.002
β-Blockers	8844 (84.3%)	3799 (86.9%)	5045 (82.4%)	<.001
Clopidogrel	7781 (74.1%)	3020 (69.1%)	4761 (77.8%)	<.001
Dual antiplatelet therapy	10,161 (96.8%)	4241 (97.0%)	5920 (96.7%)	.42
Oral anticoagulant	522 (0.5%)	215 (4.9%)	307 (5.0%)	.82
ECG at admission				
Bundle-branch block	1042 (9.9%)	389 (8.9%)	653 (10.7%)	.003
Biological results at admission				
Hemoglobin (g/dL)	13 ± 1.7	13.1 ± 1.8	13. ± 1.7	.11
Serum creatinine (mg/dL)	1.0 [0.8-1.2]	1.0 [0.8-1.2]	1.0 [0.8-1.2]	.008
Total cholesterol (mg/dL)	144.5 ± 47.1	147.6 ± 55	142.3 ± 40.4	<.001
LDL-C (mg/dL)	80.2 ± 34.1	83.3 ± 37.8	78.1 ± 31.1	<.001
HDL-C (mg/dL)	41.2 ± 12.7	40.6 ± 12.4	41.7 ± 12.9	.01
C-reactive protein (mg/dL)	2.00 [0.8-5.1]	2.10 [0.8-5.7]	1.90 [0.8-4.7]	<.001
Platelets (/mm^3)	206.7 ± 69.7	211.2 ± 73.2	203.4 ± 66.9	<.001
Procedural characteristics				
Lesion length (mm)	28.7 ± 20.9	29.3 ± 21	28.2 ± 20.8	.01
Chronic total occlusion	934 (8.9%)	418 (9.6%)	516 (8.4%)	.045
Severe coronary calcification	834 (7.9%)	346 (7.9%)	488 (8.0%)	.21
AHA/ACC class B2 or C lesion	7695 (73.3%)	3300 (75.5%)	4395 (71.8%)	<.001
Stent length (mm)	32 ± 19.7	32.9 ± 19.8	31.5 ± 19.7	<.001
Stent maximal diameter (mm)	3.1 ± 0.5	3.1 ± 0.5	3.1 ± 0.5	<.001

Baseline characteristics were defined according to the National Cardiovascular Data Registry criteria. Coronary artery calcification was assessed based on fluoroscopic appearance on the diagnostic angiogram during the index procedure by consensus agreement of, at minimum, a senior interventional fellow and an experienced interventional cardiologist. Results are presented as mean ± SD or median [interquartile 25%-75%].

LVEF, left ventricular ejection fraction; *CABG*, coronary artery bypass graft; *ECG*, electrocardiogram.

*Including 116 patients discharged with simvastatin 80 mg.

Figure 3



PCI: Percutaneous coronary intervention

Temporal evolution of statin intensity prescriptions following PCI.

Table II. Multivariate analysis for determinants of high-intensity statin prescriptions

Variables	Odds Ratio	95% CI	*P* value
Year of index procedure (vs 2011)			
2012	0.89	0.77-1.02	.10
2013	1.02	0.88-1.18	.80
2014	1.93	1.65-2.25	<.001
2015	2.35	2.01-2.74	<.001
2016	3.10	2.48-3.88	<.001
Race and ethnicity (vs white)			
Asian	0.75	0.66-0.85	<.001
African American	0.88	0.76-1.03	.10
Hispanic	1.06	0.94-1.19	.35
Other races	0.85	0.73-0.99	.043
Clinical presentation (vs stable angina)			
STEMI/NSTEMI	4.56	3.94-5.27	<.001
Unstable angina	1.30	1.18-1.43	<.001
History of MI	1.49	1.34-1.65	<.001
Co-prescription of β-blocker at discharge	1.27	1.12-1.43	<.001
Baseline LDL-C level (per 10-mg/dL increase)	1.02	1.00-1.03	.009
Stent length (per 10-mm increase)	1.03	1.01-1.05	.008
Age (per 5-y increase)	0.90	0.88-0.93	<.001
Already on statin at baseline	0.89	0.79-0.99	.036

characteristics, such as younger age, acute presentation, and more extensive coronary artery disease (CAD), were associated with the prescription of HIS at discharge. After accounting for baseline imbalances, we observed no significant differences in the 1-year cumulative risk of death, MI, or TVR among patients treated with versus without HIS, although there was a trend for reduced mortality in favor of HIS.

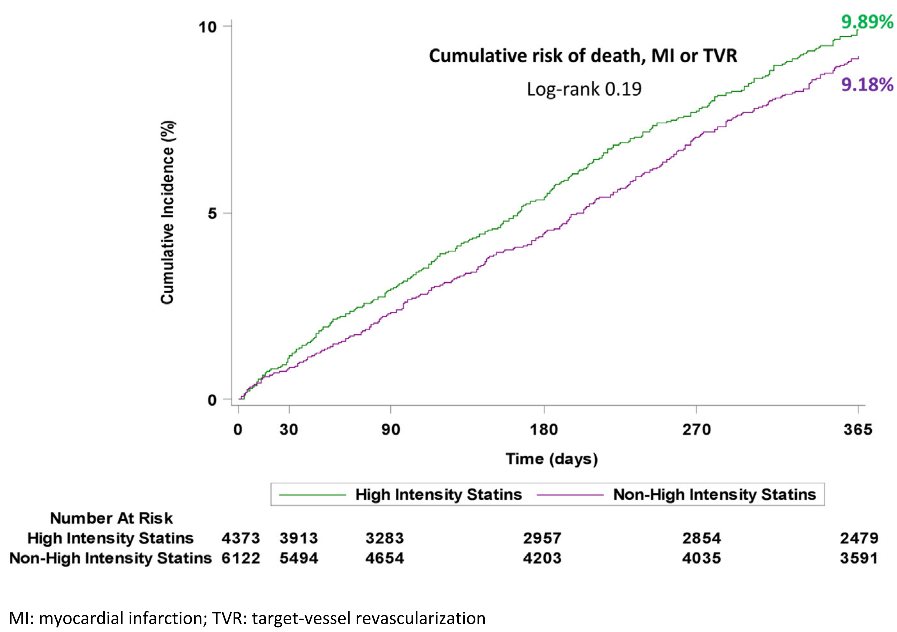
Our study is illustrative of the impact of the 2013 AHA/ACC guideline recommendations on clinical practice

Table III. Univariate analysis of 1-year clinical outcomes according to statin prescriptions at discharge

	High-intensity statins prescription (n = 4373)	Non–high-intensity statins prescription (n = 6122)	*P* value
Death, MI, or TVR	9.89%	9.18%	.19
Death	1.72%	2.15%	.26
MI	2.67%	1.88%	.014
TVR	8.41%	7.65%	.15

Data are shown as Kaplan-Meier estimate.

Figure 4



Kaplan-Meier survival curve without event (death, MI, or TVR) according to the intensity of statin prescription.

with a clear stepwise increase in the rate of HIS prescriptions between 2014 and 2016 compared with 2011-2013. These results are in line with a previous report in patients discharged after MI.[2] Conversely, rates of HIS prescriptions after 2013 were relatively high in our population compared to the results reported by Navar et al on patients with atherosclerotic cardiovascular disease.[6] This may be explained by further implementation of the 2013 AHA/ACC guidelines and by the fact that CAD has been associated with increased use of statins compared to other localization of atherosclerosis, particularly peripheral vascular disease.[3,6,8,9] In the present study, a statin treatment at baseline was an independent risk factor of reduced HIS prescriptions. Reluctance to change chronic lipid-lowering medication by the patient, particularly in case of prior statin-associated symptoms with higher dosages, or clinical inertia by the physician even after an acute coronary event may explain this association.[10] However, as the need for PCI represents an adverse evolution of atherosclerosis, these patients could still benefit from intensification of lipid-lowering treatment.[11-17]

Our study included a broad, ethnically diverse patient population with associated discrepancies regarding HIS prescriptions. Racial disparities surrounding the use of statins for secondary prevention of CAD have been previously described, with white race being associated with increased rates of HIS prescriptions.[18-20] Consistently, we found Asian ethnicity to be associated with reduced prescriptions of HIS compared with white patients. These results may be explained by socioeconomic factors and/or issues with statin intolerance, the

Table IV. Multivariate analysis of determinants of 1-year outcomes

	Adjusted hazard ratio	95% CI	*P* value
Composite of death, MI, or TVR			
High-intensity statins	0.98	0.84-1.15	.81
Female sex	0.81	0.68-0.97	.02
Diabetes mellitus	1.34	1.14-1.59	<.001
History of MI	1.51	1.29-1.78	<.001
Severe coronary calcification	1.65	1.30-2.09	<.001
STEMI/NSTEMI as indication of PCI	1.72	1.37-2.17	<.001
Unstable angina as indication of PCI	1.32	1.11-1.56	.001
C-reactive protein >2 mg at baseline	1.49	1.26-1.75	<.001
Hemoglobin at baseline (per 1-g/dL increase)	0.86	0.82-0.91	<.001
Death			
High-intensity statins	0.71	0.50-1.00	.052
Diabetes mellitus	1.87	1.29-2.73	.001
History of chronic kidney disease	2.03	1.42-2.92	.0001
History of MI	1.82	1.30-2.54	.0005
Severe coronary calcification	1.80	1.13-2.85	.013
STEMI/NSTEMI as indication of PCI	1.85	1.17-2.90	.008
C-reactive protein >2 mg at baseline	4.37	2.77-6.89	<.001
Hemoglobin at baseline (per 1-g/dL increase)	0.81	0.73-0.89	<.001
Body mass index (per 1-kg/m^2 increase)	0.96	0.94-0.99	.012
MI			
High-intensity statins	1.12	0.81-1.54	.49
Diabetes mellitus	1.85	1.30-2.62	<.001
History of MI	1.64	1.19-2.27	.003
STEMI/NSTEMI as indication of PCI	3.66	2.41-5.55	<.001
Unstable angina as indication of PCI	1.48	1.02-2.16	.04
C-reactive protein >2 mg at baseline	1.89	1.31-2.73	<.001
Hemoglobin at baseline (per 1-g/dL increase)	0.79	0.73-0.87	<.001
TVR			
High-intensity statins	1.06	0.89-1.26	.51
Diabetes mellitus	1.27	1.06-1.52	.01
History of MI	1.29	1.07-1.55	.007
Severe coronary calcification	1.42	1.07-1.88	.02
STEMI/NSTEMI as indication of PCI	1.37	1.03-1.81	.029
Unstable angina as indication of PCI	1.46	1.22-1.75	<.001
Hemoglobin at baseline (per 1-g/dL increase)	0.94	0.89-0.99	.03
Older age (per 1-y increase)	0.99	0.98-1.00	.007

latter being particularly prevalent among Asian patients.[21] In particular, an adjustment of the dose of rosuvastatin is recommended by the federal Food and Drug Administration in Asian patients, as it may come with a nearly 2-fold increase in exposure time compared to white patients.[22] Interestingly, we showed that patients who could not be categorized as African American, Hispanic, Asian, or Caucasian were undertreated in term in statins intensity after PCI. In the present studies, African American and Hispanic ethnicities were not significantly associated with reduced HIS statins prescription compared to white patients. These results differ from recent studies which described undertreatment regarding statin intensity with these patients.[19,23] Finally, sex was not an independent risk factor of statin prescriptions in the present analysis, although sex-based differences as they relate to the prescription of statins for secondary prevention have been recently described.[23-28]

Overall, we found that HIS prescriptions were highly correlated with the risk profile of patients. History of MI, acute coronary syndrome as the clinical presentation for the index PCI, increased stent length, and higher baseline LDL-C have been commonly described as risk factors for ischemic recurrence following PCI and were associated with increased HIS prescriptions.[29,30] We observed a higher risk of MI within 1 year of PCI among patients discharged with HIS in univariate analysis, probably reflecting the increased risk profile of these patients. This association was no longer significant after adjusting for baseline and procedural characteristics, although we found a trend toward reduced mortality with HIS prescription.

Our study was not without limitations. First, our observational study design precludes us from making any causal inferences. Second, this was a single-center study, and our results may not be generalizable to patients undergoing PCI at other centers. Nonetheless, we attempted to mitigate this limitation by including a large number of patients in our study. Third, LDL-C values at baseline were lower in the present study than in previous randomized control trials,[11,12,31] which might be explained by the large overall prevalence of statin prescriptions at baseline in our study (78.4%). Hence, HIS prescriptions might have resulted in a relatively modest LDL-C decrease post-PCI. As a result, the present study may have been underpowered to demonstrate the benefits of HIS prescriptions in these patients. Moreover, the follow-up duration of 1 year in the present study may have been too short to evaluate the longer-term effects of high-intensity statins to become evident. The intensity of statin therapy before PCI, any prior intolerance to statins, or the types of insurance coverage, which may have a confounding effect on a patient's likelihood of being discharged on a high-intensity statin, were not available. Although the Food and Drug Administration recommended against the initiation or the titration of simvastatin 80 mg, we included the 116 patients discharged with such dosage in the analysis, as it was previously evaluated in randomized trial as an HIS. Finally, we did not include patients older than 75 years, as the level of evidence supporting the use of high-intensity statins in this population is less clear.[1,32]

Conclusion

Although HIS prescriptions following PCI have increased among patients aged ≤75 years since the introduction of the 2013 AHA/ACC guideline, 40% of patients continue to receive low- or moderate-intensity statins following PCI. Targeting independent predictors of limited HIS prescriptions may improve practices according to 2013 AHA/ACC guidelines and most importantly patient outcomes.

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Disclosures

All authors have materially participated in the research and/or article preparation and approved the final article.

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Dr Sharma reports industry-sponsored lectures for Abbott Laboratories; AngioScore, Inc; Boston Scientific Corporation; Cardiovascular Systems, Inc; Daiichi-Sankyo Co Ltd/Eli Lilly and Company Partnership; Medtronic, Inc; and The Medicines Company and being in the Scientific Advisory Board of Cardiovascular Systems, Inc.

Dr Dangas reports receiving consulting fees from Boston Scientific.

References

1. Stone NJ, Robinson JG, Lichtenstein AH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation 2014;129(25 Suppl 2):S1-45.
2. Rosenson RS, Farkouh ME, Mefford M, et al. Trends in use of high-intensity statin therapy after myocardial infarction, 2011 to 2014. J Am Coll Cardiol 2017;69(22):2696-706.
3. Valentino M, Al Danaf J, Panakos A, et al. Impact of the 2013 American College of Cardiology/American Heart Association cholesterol guidelines on the prescription of high-intensity statins in patients hospitalized for acute coronary syndrome or stroke. Am Heart J 2016;181:130-6.
4. Bellows BK, Olsen CJ, Voelker J, et al. Antihyperlipidemic medication treatment patterns and statin adherence among patients with ASCVD in a managed care plan after release of the 2013 ACC/AHA guideline on the treatment of blood cholesterol. J Manag Care Spec Pharm 2016;22(8):892-900.
5. Virani SS, Pokharel Y, Steinberg L, et al. Provider understanding of the 2013 ACC/AHA cholesterol guideline. J Clin Lipidol 2016;10(3):497-504.e4.
6. Navar AM, Wang TY, Li S, et al. Lipid management in contemporary community practice: results from the Provider Assessment of Lipid Management (PALM) Registry. Am Heart J 2017;193:84-92.
7. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Circulation 2012;126(16):2020-35.
8. Steen DL, Khan I, Becker L, et al. Patterns and predictors of lipid-lowering therapy in patients with atherosclerotic cardiovascular disease and/or diabetes mellitus in 2014: insights from a large US managed-care population. Clin Cardiol 2017;40(3):155-62.
9. Mukherjee D, Lingam P, Chetcuti S, et al. Missed opportunities to treat atherosclerosis in patients undergoing peripheral vascular interventions: insights from the University of Michigan Peripheral Vascular Disease Quality Improvement Initiative (PVD-QI2). Circulation 2002;106(15):1909-12.
10. Phillips LS, Branch WT, Cook CB, et al. Clinical inertia. Ann Intern Med 2001;135(9):825-34.
11. Cannon CP, Braunwald E, McCabe CH, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. N Engl J Med 2004;350(15):1495-504.
12. Cholesterol Treatment Trialists' (CTT) Collaboration. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170 000 participants in 26 randomised trials. Lancet 2010;376(9753):1670-81.
13. de Lemos JA, Blazing MA, Wiviott SD, et al. Early intensive vs a delayed conservative simvastatin strategy in patients with acute

coronary syndromes: phase Z of the A to Z Trial. JAMA 2004;292(11): 1307-16.
14. Schwartz GG, Olsson AG, Ezekowitz MD, et al. Effects of atorvastatin on early recurrent ischemic events in acute coronary syndromes: the MIRACL study: a randomized controlled trial. JAMA 2001;285(13): 1711-8.
15. Rodriguez F, Maron DJ, Knowles JW, et al. Association between intensity of statin therapy and mortality in patients with atherosclerotic cardiovascular disease. JAMA Cardiol 2017;2(1):47-54.
16. Ray KK, Cannon CP, McCabe CH, et al. Early and late benefits of high-dose atorvastatin in patients with acute coronary syndromes: results from the PROVE IT-TIMI 22 trial. J Am Coll Cardiol 2005;46(8): 1405-10.
17. Sakamoto T, Kojima S, Ogawa H, et al. Effects of early statin treatment on symptomatic heart failure and ischemic events after acute myocardial infarction in Japanese. Am J Cardiol 2006;97(8):1165-71.
18. Lauffenburger JC, Robinson JG, Oramasionwu C, et al. Racial/ethnic and gender gaps in the use of and adherence to evidence-based preventive therapies among elderly Medicare Part D beneficiaries after acute myocardial infarction. Circulation 2014;129(7):754-63.
19. Johansen ME, Hefner JL, Foraker RE. Antiplatelet and statin use in us patients with coronary artery disease categorized by race/ethnicity and gender, 2003 to 2012. Am J Cardiol 2015;115(11):1507-12.
20. Schroff P, Gamboa CM, Durant RW, et al. Vulnerabilities to health disparities and statin use in the REGARDS (Reasons for Geographic and Racial Differences in Stroke) study. J Am Heart Assoc 2017;6(9), https://doi.org/10.1161/JAHA.116.005449. pii: e005449.
21. Dai W, Huang X, Zhao S. No evidence to support high-intensity statin in Chinese patients with coronary heart disease. Int J Cardiol 2016;204:57-8.
22. Lee E, Ryan S, Birmingham B, et al. Rosuvastatin pharmacokinetics and pharmacogenetics in white and Asian subjects residing in the same environment. Clin Pharmacol Ther 2005;78(4):330-41.
23. Gamboa CM, Colantonio LD, Brown TM, et al. Race-sex differences in statin use and low-density lipoprotein cholesterol control among people with diabetes mellitus in the Reasons for Geographic and Racial Differences in Stroke Study. J Am Heart Assoc 2017;6(5), https://doi.org/10.1161/JAHA.116.004264. pii: e004264.
24. Albright KC, Howard VJ, Howard G, et al. Age and sex disparities in discharge statin prescribing in the stroke belt: evidence from the Reasons for Geographic and Racial Differences in Stroke Study. J Am Heart Assoc 2017;6(8), https://doi.org/10.1161/JAHA.117.005523. pii: e005523.
25. Peters SAE, Colantonio LD, Zhao H, et al. Sex differences in high-intensity statin use following myocardial infarction in the United States. J Am Coll Cardiol 2018;71(16):1729-37.
26. Lu Y, Zhou S, Dreyer RP, et al. Sex differences in lipid profiles and treatment utilization among young adults with acute myocardial infarction: results from the VIRGO study. Am Heart J 2017;183: 74-84.
27. Rodriguez F, Lin S, Maron DJ, et al. Use of high-intensity statins for patients with atherosclerotic cardiovascular disease in the Veterans Affairs Health System: practice impact of the new cholesterol guidelines. Am Heart J 2016;182:97-102.
28. Wei J, Mehta PK, Grey E, et al. Sex-based differences in quality of care and outcomes in a health system using a standardized STEMI protocol. Am Heart J 2017;191:30-6.
29. Levine GN, Bates ER, Bittl JA, et al. 2016 ACC/AHA guideline focused update on duration of dual antiplatelet therapy in patients with coronary artery disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines: an update of the 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention, 2011 ACCF/AHA guideline for coronary artery bypass graft surgery, 2012 ACC/AHA/ACP/AATS/PCNA/SCAI/STS guideline for the diagnosis and management of patients with stable ischemic heart disease, 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction, 2014 AHA/ACC guideline for the management of patients with non–ST-elevation acute coronary syndromes, and 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery. Circulation 2016;134(10):e123-55.
30. Chandrasekhar J, Baber U, Sartori S, et al. Effect of increasing stent length on 3-year clinical outcomes in women undergoing percutaneous coronary intervention with new-generation drug-eluting stents: patient-level pooled analysis of randomized trials from the WIN-DES Initiative. JACC Cardiovasc Interv 2018;11(1): 53-65.
31. LaRosa JC, Grundy SM, Waters DD, et al. Intensive lipid lowering with atorvastatin in patients with stable coronary disease. N Engl J Med 2005;352(14):1425-35.
32. O'Brien EC, Wu J, Schulte PJ, et al. Statin use, intensity, and 3-year clinical outcomes among older patients with coronary artery disease. Am Heart J 2016;173:27-34.