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Beneficial effects of statins after percutaneous coronary intervention

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Abstract

Background—Evidence about the efficacy of statin treatment among patients after percutaneous coronary intervention (PCI) is very limited. The rapid advancement in PCI technology and near universal use of adjunctive cardioprotective medications make it necessary to formally assess the effect of statin therapy on cardiac events after PCI.

Design—This was a multicenter prospective cohort study

Methods—Patients who received stent implantation and survived to hospital discharge from the National Heart, Lung, and Blood Institute Dynamic Registry from 2004 to 2006 formed the study cohort. Patients with cardiogenic shock, in-hospital adverse events [including myocardial infarction and coronary artery bypass graft surgery (CABG)], liver disease, renal disease, alcoholism, or drug abuse were excluded. The occurrences of death, CABG, and repeat PCI, and repeat revascularization were collected over 1-year follow-up.

Results—Of the 3227 patients evaluated, 2737 (85%) were prescribed a statin at discharge. By 1-year follow-up, incident events were 98 deaths, 44 CABG, 290 repeat PCI procedures, and 328 repeat revascularizations. After propensity score adjustment, postdischarge statin therapy was associated with lower risks of death [hazard ratio (HR) λ = λ 0.58, 95% confidence interval (CI): 0.36–0.93, $P\lambda$ = λ 0.02], CABG (HR λ = λ 0.49, 95% CI: 0.24–1.00, $P\lambda$ = λ 0.05), and repeat revascularization (HR λ = λ 0.74, 95% CI: 0.56–1.00, $P\lambda$ = λ 0.05).

Conclusion—These results support the routine use of statin therapy after PCI.

Keywords

mortality; propensity score; repeat revascularization; stent

Introduction

Secondary prevention trials of statins (HMG-CoA reductase inhibitors) [1–3] have shown a 25–30% reduction in ischemic events among patients with coronary artery disease. However, data about the effects of statins on outcomes for patients after percutaneous coronary intervention (PCI) are limited. Several observational studies, but few clinical trials, have shown that statin use after PCI improves safety [4,5] and effectiveness [4,6]. A recent meta-analysis of six randomized clinical trials [7] concluded that statin therapy reduces the risk of myocardial infarction (MI) after elective PCI, but not necessarily mortality or repeat revascularization. The applicability of these studies to current practice is limited, however, as drug-eluting stents were not routinely available, other concomitant cardiac medications were not in use or the analyses were limited to selected patient subgroups. With these considerations in mind, we evaluated the effect of postdischarge statin therapy on clinical outcomes in PCI patients treated with one or more stents in the National Heart, Lung, and Blood Institute Dynamic Registry from 2004 to 2006.

Methods

Study design and patient selection

The National Heart, Lung, and Blood Institute Dynamic Registry is a prospective, multicenter observational study. It is designed to collect demographic, clinical, angiographic, and procedural data on the consecutive patients undergoing PCI at selected clinical centers in North America during prespecified time intervals or ‘waves’ [wave 1: July 1997 to February 1998 ($n=2524$ patients); wave 2: February 1999 to June 1999 ($n=2105$ patients); wave 3: October 2001 to March 2002 ($n=2047$ patients); wave 4: February 2004 to May 2004 ($n=2112$ patients); and wave 5: February 2006 to July 2006 ($n=2174$ patients)]. Trained on-site research coordinators were guided by a manual of operations. The study was approved by the institutional review board of each clinical site. All participants gave written informed consent.

For this analysis, we evaluated patients from the two most recent recruitment waves (wave 4 and wave 5), because the PCI procedures performed and postdischarge medical therapy reflect current clinical practice the most. Patients who received one or more stents and survived to hospital discharge were included. Patients with cardiogenic shock, in-hospital adverse events [including MI or coronary artery bypass graft surgery (CABG)], liver disease, renal disease, alcoholism, or drug abuse were excluded, resulting in a total of 3227 patients for the final analysis. After discharge, patients were contacted by telephone 30 days, 6 months, and annually thereafter to assess vital status as well as to collect information on new hospitalizations and the need for repeat revascularization procedures.

Death is defined as all-cause mortality. Repeat PCI includes both target and nontarget vessel interventions. Target vessel revascularization was defined as a repeat revascularization procedure involving a previously treated artery. Repeat revascularization includes both repeat PCI and CABG.

The follow-up for each patient started from the date of hospital discharge. Patients were censored at the last known date of contact or at 1-year of follow-up, whichever was earlier.

Statistical methods

Patients were categorized into two groups according to whether or not statin therapy was prescribed at discharge post-PCI. Intergroup differences of continuous variables were analyzed with Student’s *t*-test or a Wilcoxon’s nonparametric test. Intergroup differences of categorical variables were analyzed with χ^2 tests or Fisher’s exact tests. Logistic regression

was used to develop a propensity score model to adjust for factors associated with the decision to prescribe postdischarge statin therapy [8,9]. A propensity score is the conditional probability of 'being assigned' to a particular exposure group, given a set of observed characteristics. Patients with the same propensity score have equal estimated probabilities to 'be assigned' to each exposure group and the same conditional distribution of the observed characteristics. Therefore, it is akin to randomized clinical trials, which achieve balance of confounders between the exposure groups through randomization. A candidate list of demographic variables, disease history, procedural characteristics, and medications (other than statins) at discharge was considered. Age, sex, race, and enrolment wave were included in the model. A backward selection approach was used to choose other predictors, using a *P* value of 0.40. Covariate balance between statin-treated patients and nonstatin-treated patients was assessed after adjustment for the propensity score as a continuous covariate, using logistic regression for categorical covariates and linear regression for continuous covariates.

Time to adverse events was summarized by the Kaplan–Meier estimates and the corresponding curves were compared by log-rank tests. Cox proportional hazards regression was used to estimate 1-year hazard ratios for adverse events. In addition to adjusting for a continuous propensity score, we also considered alternative adjustments for selection bias by using dummy variable adjustment for quartiles of the propensity score, and by weighting the Cox model by the inverse probability, which is the inverse of the probability of receiving the observed exposure conditional on observed covariates [10].

The proportional hazards assumption of constant relative risk was evaluated by testing the interaction between time and the primary exposure and found to hold. All statistical tests were performed using the SAS program, Version 9.1 (SAS Institute, Cary, NC). *P* values ≤ 0.05 were considered to be statistically significant.

Results

Of the 3227 patients included in this analysis, 2737 (85%) were taking a statin after PCI discharge and the remaining 490 patients (15%) were not treated with a statin. The site-specific proportions of patients who were taking a statin after PCI discharge in 17 clinical sites ranged from 71.7 to 91.7% and at one site all five patients were discharged on a statin after PCI procedure.

Statin-treated patients were more likely to be of younger age or to have private insurance, a history of MI, chest pain at admission, or a history of hypercholesterolemia (Table 1). Statin-treated patients were also less likely to be female, or to have a history of congestive heart failure or congestive heart failure at admission.

Procedural indications, lesion and procedural characteristics and medications prescribed at discharge are shown in Table 2. Statin-treated patients were more likely than nonstatin-treated patients to present with acute MI or urgent/emergent acuity, and receive periprocedural glycoprotein IIb/IIIa inhibitors. Treated lesions in statin-treated patients were more likely to have evidence of thrombus. As expected, statin-treated patients were less likely to receive cholesterol-lowering drug other than statins, but they were also less likely to receive only angiotensin-converting enzyme (ACE) inhibitors or only calcium channel blockers. Statin-treated patients were more likely to receive aspirin.

The probability density functions of the propensity score among the statin-treated and nonstatin-treated patients are shown in Fig. 1. Although the vast majority of statin-treated patients had a very-high predicted probability of receiving statin therapy, the figure illustrates adequate overlap of propensity scores between the treated and untreated patients

for the purposes of treatment comparisons. None of the significant differences in either Table 1 or Table 2 remained statistically significant after propensity score adjustment. The use of the propensity score as a covariate balanced the statin-treated and untreated patients in terms of observed demographic, clinical, and procedural characteristics.

Incident events (first occurrence) included 98 deaths, 290 repeat PCI procedures, 44 CABG surgeries, and 328 repeat revascularizations. Unadjusted cumulative incidence rates (expressed as percentages) for each adverse event at 1 year are shown in Table 3. Compared with nonstatin-treated patients, statin-treated patients had significantly lower cumulative incidence rates of death, and a borderline significantly lower rate of CABG. The estimated Kaplan–Meier survival curves of death and CABG are shown in Fig. 2. Each of these curves shows consistent differences over time with no significant departure from proportional hazards.

Estimated adjusted hazard ratios from the Cox proportional hazard models are shown in Table 4. After adjustment with the propensity score as a continuous covariate, postdischarge statin treatment was associated with significant reductions of 42% in the risk of death, 51% in the risk of CABG, and 26% in the risk of repeat revascularization. The reduction of 21% in need for repeat PCI was not statistically significant. Similar effect estimates and conclusions were obtained using the alternative methods of adjustment for selection bias (results not shown).

Discussion

This study evaluated whether planned statin treatment after hospital discharge was associated with lower risks of adverse cardiac events among PCI treated patients, the majority of whom were simultaneously receiving other adjunctive medical therapy. Our analyses were adjusted for factors associated with statin treatment; our use of the propensity score as a covariate balanced the statin-treated and untreated patients in terms of demographic, clinical, and procedural characteristics. Our data indicate a beneficial effect of statins on death, CABG, and repeat revascularization, which occurs independently of other routinely prescribed cardiac medications, and thus support the routine use of statin therapy after PCI.

Our study showed an estimated 42% reduction in the adjusted risk of death at 1 year associated with statin therapy used after PCI. The magnitude of this effect is comparable with other observational studies investigating effect of periprocedural statins on mortality [4,5,11]. The Fluvastatin Angiographic Restenosis trial [12] observed a lower rate of the composite endpoint of death or MI for periprocedural statin therapy (1.4 vs. 4.0%, $P\lambda=0.025$), although this particular analysis was not prespecified. In contrast, several clinical trials [13–15] did not find a statistically significant association between periprocedural statin use and mortality risk. Importantly, mortality was not the primary endpoint in these negative trials, and some of the sample sizes were small to modest. From a mechanistic standpoint, statins reduce blood cholesterol levels [1–3]. Beyond that, statins also have favorable effects on platelet adhesion [16], risk of thrombosis [17], endothelial function [18], plaque stability [19], inflammation [20], and atherosclerosis regression [21]. All of these effects may contribute to a potential mortality benefit after coronary revascularization with PCI. Further investigations that are warranted, but not possible using our data, might focus on identifying the mechanism(s) by which statins lower the risk of mortality after PCI.

Earlier laboratory experiments [22,23] and human studies [24,25] have shown that statins inhibit smooth muscle proliferation. Consistent with this mechanism, we observed a

reduction of 26% in risk of repeat revascularization. This may be a result of less restenosis among the statin-treated patients. Since a small study in 1991 [26] suggested a potential beneficial effect of statin on restenosis, several additional studies [4,6] have reported similar findings in PCI patients. Moreover, a clinical trial of post-PCI patients [27] reported significantly slower plaque growth in the statin treatment arm compared with usual care. In contrast, other trials [12–14] have failed to show that periprocedural statins reduce the risk of restenosis. However, these latter trials were designed to assess restenosis in the setting of balloon angioplasty, thereby limiting their relevance to current PCI practice. In our study, the majority of both statin-treated and nonstatin-treated patients (83%) received at least one drug-eluting stent.

There are limitations to our study. First, although we adjusted for a wide array of factors associated with statin prescription with a propensity score model that eliminated observed baseline imbalances, the apparent beneficial effect of statin may be the result of residual confounding. We do not know the number of patients who did not take statin because of intolerance. However, there is little evidence that intolerance to statins is associated with the prognosis of coronary artery disease. We did not adjust for left ventricular ejection fraction because of the large amount of missing data. Among the patients with this information available, there was no evidence that left ventricular ejection fraction differed between statin-treated and nonstatin-treated patients. We did not collect blood cholesterol values at discharge, which is an indication for statin treatment. However, there is compelling evidence [4–6,15] that coronary artery disease patients with either high or low baseline low-density lipoprotein benefit similarly from statins. We did not adjust for statin use at other times (i.e. during or before the PCI procedure) because of the lack of information for patients from wave 4. However, among the 1652 patients from wave 5, the associations between statin prescription at discharge and statin use at other time was low. Second, the prescription information that was collected did not take into account patient compliance throughout the follow-up. However, we have no reason to believe such misclassification would be differential, in which case the potential net effect would likely be bias toward the null (i.e. underestimation of true beneficial effect of statin therapy).

Overall, our study provides evidence that statin therapy after contemporary PCI is associated with reduced risks of mortality, CABG and need for repeat revascularization, and that this lower risk occurs independently of adjunctive cardiac medical therapy. These results support the routine use of statin therapy after PCI.

Acknowledgments

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Appendix

Variables used in propensity model adjustment

Sex, age, enrolment wave, body mass index, history of prior percutaneous coronary intervention, coronary artery bypass graft surgery, myocardial infarction, congestive heart failure, diabetes, hypertension, hypercholesterolemia, procedure indications, procedure circumstance, vessel disease, number of lesions, left anterior descending artery $\geq 50\%$ stenosis, right coronary artery $\geq 50\%$ stenosis, left circumflex $\geq 50\%$ stenosis, other atherectomy attempt, balloon attempt, local drug delivery, renal-protective medications before procedure, periprocedural glycoprotein IIb/IIIa inhibitor, heparin use during procedure, medication use at discharge: aspirin, thienopyridines, cholesterol-lowering drug other than statins, calcium channel blockers, angiotensin-converting enzyme inhibitor, β -blocker, and two two-way and one three-way interaction terms between calcium channel blocker, angiotensin-converting enzyme inhibitor, and β -blocker.

Fig. 1.

Probability density functions of the propensity score among the 490 nonstatin-treated patients and the 2737 statin-treated patients in Dynamic Registry from 2004 to 2006. The comparison of propensity scores distribution among statin-treated patients and nonstatin-treated patients. The solid line depicts the propensity score among statin-treated patients and the dotted line depicts the propensity score among nonstatin-treated patients.

Fig. 2. The Kaplan Meier survival curves for (a) death; (b) coronary artery bypass graft (CABG) surgery among the 490 nonstatin-treated patients and the 2737 statin-treated patients in Dynamic Registry from 2004 to 2006. The comparison of survival among statin-treated and nonstatin-treated patients. (a) The endpoint of death and (b) the endpoint of CABG surgery. The solid line represents the survival of statin-treated patients and the dotted line stands for the survival of nonstatin-treated patients.

Table 1

Demographic and disease history characteristics for non-statin-treated and statin-treated patients

Characteristic	No statin (<i>n</i> =1490)	Statin (<i>n</i> =12737)	<i>P</i> *	<i>P</i> **
Patient demographics				
Female (%)	38.8	31.7	<0.01	0.67
Age mean (SD) (years)	65.8 (11.9)	63.4 (11.9)	<0.0001	0.89
Race (%)			0.04	0.25
White	81.4	80.1		
Black	13.9	15.1		
Asian	1.4	3.0		
Other	3.3	1.8		
Insurance (%)			0.03	0.52
Medicare	39.0	34.6		
Other public	19.3	16.8		
Private	39.6	45.1		
None/self-pay	2.1	3.5		
BMI (kg/m ²) (%)			0.39	1.00
<25	23.5	20.7		
25–30	36.1	39.1		
30–35	25.5	24.4		
35–40	8.4	9.9		
>40	6.5	5.8		
Enrolled at wave 5 (%)	50.4	51.4	0.71	0.94
Disease history				
Prior PCI (%)	34.1	32.1	0.39	0.99
Prior CABG (%)	19.0	17.6	0.45	0.99
Prior MI (%)	20.2	24.6	0.04	0.70
History of diabetes (%)	37.4	33.1	0.07	0.81
History of chest pain (%)	52.7	54.7	0.44	0.70
Chest pain at admission (%)	66.5	72.2	0.01	0.91
History of CHF (%)	13.3	8.6	0.001	0.87
CHF at admission (%)	7.6	5.3	0.05	0.81
Severe noncardiac disease (%)	38.3	34.1	0.07	0.99
History of hypertension (%)	79.6	76.9	0.19	0.85
History of hypercholesterolemia (%)	66.5	78.9	<0.0001	0.56

Categorical variables are presented as percentages, continuous variables as mean (SD).

BMI, body mass index; CABG, coronary artery bypass graft surgery; CHF, congestive heart failure; MI myocardial infarction; PCI, percutaneous coronary intervention.

* *P* value of tests for covariate balance between statin-treated and nonstatin-treated patients before propensity score adjustment.

** *P* value of tests for covariate balance between statin-treated and nonstatin-treated patients after adjustment for the propensity score as a continuous covariate.

Table 2

Procedure and lesion characteristics and medication at discharge among the nonstatin-treated patients and statin-treated patients

Characteristic	No statin (<i>n</i> =1490)	Statin (<i>n</i> =12737)	<i>P</i> value *	<i>P</i> value **
λProcedure characteristics				
νProcedure indication (%)			0.001	0.99
ννAcute MI	22.9	28.4		
ννUnstable angina	33.5	34.0		
ννStable angina	20.2	21.4		
ννAsymptomatic CAD	16.1	11.9		
ννOther	7.4	4.3		
νProcedure circumstance (%)			0.001	0.80
ννElective	66.9	57.9		
ννUrgent	25.9	31.5		
ννEmergent	7.1	10.6		
νUse of drug-eluting stent (%)	82.0	83.0	0.60	0.81
νLeft ventricular ejection fraction, mean (SD) (%)	51.8 (13.7)	52.4 (13.3)	0.46	N/A ^a
νPeriprocedural glycoprotein IIb/IIIa inhibitor (%)	30.6	37.2	0.01	0.68
νVessel disease (%)			0.48	0.77
ννSingle	38.2	35.9		
ννDouble	29.7	32.3		
ννTriple	32.2	31.8		
Lesion characteristic				
νNumber of lesions (%)			0.33	0.95
νν1	73.5	73.9		
νν2	22.7	20.8		
νν3 or more	3.9	5.3		
νAny lesion >30λmm (%)	9.0	7.9	0.41	0.60
νAny evidence of thrombus (%)	12.5	17.3	0.01	0.21
νAny calcification (%)	31.6	29.9	0.44	0.54
νAny ulcerated lesion (%)	13.9	15.0	0.50	0.84
νAny ostial lesion (%)	9.4	9.2	0.89	0.87
νAny tortuous lesion (%)	27.8	25.2	0.24	0.22
νAny lesion receives collateral (%)	10.8	10.7	0.96	0.56
νAngiographic success (%)			0.44	0.48
ννYes	98.2	98.4		
ννPartial	1.0	1.2		
ννNo	0.8	0.4		
Medications at discharge				
νCholesterol-lowering drug other than statins (%)	22.5	11.9	<0.0001	0.67
νLong-acting nitrates (%)	15.1	13.8	0.43	0.87
νThienopyridines (%)	97.6	98.4	0.21	0.93

Characteristic	No statin (<i>n</i> =1490)	Statin (<i>n</i> =12737)	<i>P</i> value [*]	<i>P</i> value ^{**}
vAspirin (%)	94.5	98.1	<0.0001	0.73
vOther concomitant cardiac medications (%)			<0.0001	1.00
vvNone	7.1	6.4		
vvOnly ACE	35.1	30.7		
vvOnly β -blocker	3.5	2.3		
vvOnly calcium channel blocker	28.0	40.2		
vvACE inhibitors+ β -blocker	3.1	1.5		
vvACE inhibitors+calcium channel blocker	7.4	5.7		
vvBeta-blocker+calcium channel blocker	5.7	6.0		
vvACE inhibitors+ β -blocker+calcium channel blocker	10.2	7.2		

Categorical variables are presented as column percentages, continuous variables as mean (SD). ACE, angiotensin-converting enzyme; CAD, coronary artery disease; MI, myocardial infarction.

^a Owing to 30% of missing cases, left ventricular ejection fraction is not included as a predictor in the propensity score model.

^{*} *P* value of tests for covariate balance between statin-treated and nonstatin-treated patients before propensity score adjustment.

^{**} *P* value of tests for covariate balance between statin-treated and nonstatin-treated patients after adjustment for the propensity score as a continuous covariate.

Unadjusted 1-year cumulative incident proportions of adverse events among the nonstatin-treated and statin-treated patients

Table 3

Event	No statin (N1=1490)		Statin (N2=12737)		P value
	n	(%)	n	(%)	
λDeath	27	5.7	71	2.7	0.001
Repeat PCI	51	10.8	239	9.1	0.20
CABG surgery	11	2.4	33	1.3	0.06
Repeat revascularization	60	12.8	268	10.2	0.09

CABG, coronary artery bypass graft; PCI, percutaneous coronary intervention.
P values are for log-rank tests comparing unadjusted 1-year cumulative incident proportions.

Table 4

Estimated hazard ratios and 95% confidence intervals of adverse events associated with statin treatment at discharge after PCI, based on the Cox proportional hazards models adjusted for the propensity score as a continuous covariate

Event	Hazard ratio	95% Confidence intervals	P value
λDeath	0.58	0.36–0.93	0.02
Repeat PCI	0.79	0.58–1.08	0.14
CABG surgery	0.49	0.24–1.00	0.05
Repeat revascularizations	0.74	0.56–1.00	0.05

CABG, coronary artery bypass graft; PCI, percutaneous coronary intervention.