

ORIGINAL ARTICLES

Undertreatment of Hyperlipidemia in the Secondary Prevention of Coronary Artery Disease

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OBJECTIVES: To determine adherence to national guidelines for the secondary prevention of coronary artery disease (CAD) using lipid-lowering drugs (LLDs), by studying the rate of use of LLDs, predictors of use, and the rate of achieving lipid goals, among eligible patients recently hospitalized with acute myocardial infarction.

DESIGN: Cross-sectional analysis of 2,938 medical records, collected from July 1995 to May 1996.

SETTING: Thirty-seven community-based hospitals in Minnesota.

PATIENTS: The 622 patients had previously established CAD and hyperlipidemia (total cholesterol >200 mg/dL or currently using LLDs), and were eligible for LLDs according to the National Cholesterol Education Program II (NCEP II) Guidelines.

MEASUREMENTS: The use of LLDs in eligible patients (primary outcome) and successful achievement of NCEP II goals (total cholesterol <160 mg/dL) among treated patients (secondary outcome).

MAIN RESULTS: Only 230 (37%) of 622 eligible patients received LLDs. In multivariate logistic regression, factors independently related to LLD use included age greater than 74 years (adjusted odds ratio [AOR] 0.55; 95% confidence interval [CI] 0.35, 0.88) and severe comorbidity (AOR 0.60; 95% CI 0.38, 0.95), managed care enrollee (AOR 1.56; 95% CI 1.02, 2.39), past smoker (AOR 1.72; 95% CI 0.98, 3.01), prior revascularization (AOR 2.31; 95% CI 1.51, 3.53), and the use of aspirin (AOR 1.59; 95% CI 1.07, 2.38) or ≥ 4 medications (AOR 2.89; 95% CI 2.19, 3.84). Of the treated patients who had lipid levels measured ($n = 149$), 15% achieved the recommended goal of a total cholesterol below 160 mg/dL. Of the untreated patients ($n = 392$), 89% were discharged from hospital without a LLD prescription.

CONCLUSIONS: Lipid-lowering drugs, although proven effective for the secondary prevention of CAD, were used by only

one third of eligible patients. Among patients receiving LLDs, few achieved recommended lipid goals. Directed quality improvement interventions, such as starting LLDs during hospitalization, may have the potential to substantially reduce CAD morbidity and mortality in this vulnerable population.

KEY WORDS: hyperlipidemia; treatment; guidelines; secondary prevention; coronary artery disease.

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Many factors influence whether a new drug will be widely prescribed by physicians, including product characteristics (such as novelty, comparative efficacy, safety, tolerability, and cost), marketing intensity, media awareness, patient demands and expectations, and social influences.^{1,2} Based on such factors, the use of lipid-lowering drugs (LLDs) for the secondary prevention of coronary artery disease (CAD) should be high.

Observational studies and meta-analyses have long suggested aggressive lipid lowering, for secondary prevention, should be effective for decreasing morbidity and mortality.³⁻⁶ In 1987, new drugs became available (the 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors, as a class referred to as the "statins"), which were potent, easy to use, better tolerated than older drugs, had few adverse effects, and were heavily marketed.^{5,7} In 1993, widely disseminated revised guidelines (The National Cholesterol Education Program-Adult Treatment Panel II [NCEP II]) recommended aggressive lipid lowering for patients with established CAD.⁸ And in 1994, the large Scandinavian Simvastatin Survival Study, a secondary prevention trial, reported lipid-lowering therapy could reduce total mortality, as well as cardiovascular morbidity, with benefits of the magnitude predicted by the epidemiologic literature.^{4,9}

Given these influences to change prescribing behavior, we sought to investigate the extent to which LLDs would be used in eligible patients, and when used, to determine whether treatment goals would be achieved. We hypothesized that elderly patients and women would be at particular risk of underuse, as previously reported for other effective cardiovascular drugs^{10,11} and procedures.¹² We also hypothesized that clinical factors, in particular the presence of unrelated comorbidity,¹³ would be associated with undertreatment. To study these questions, we performed a cross-sectional analysis of population-based data collected in the context of a trial to improve the quality of the treatment of acute myocardial infarction (AMI)

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through the use of local opinion leaders.¹⁴ The data were collected between 1995 and 1996, well after all of the described prescribing influences had occurred.

METHODS

Patients and Setting

The data for the present study were drawn from the medical records of patients admitted with AMI to 37 Minnesota hospitals during the period July 1995 to May 1996.¹⁴ Although study hospitals volunteered to be part of our quality improvement program, they represented more than 80% of all community hospital beds and more than half of all AMIs in the state. Two hospitals were academic centers, and 17 were located in rural communities. Nineteen hospitals had fewer than 100 beds, and only two had more than 500 beds.

Over a 10-month period, data were collected on 2,938 patients admitted with a diagnosis of acute or suspected myocardial infarction who met at least two of the following criteria: (1) clinical symptoms typical of AMI; (2) compatible electrocardiographic findings as documented in the medical record by a physician; and (3) elevated serum creatine kinase and MB fractions.¹⁴ Patients were excluded if they died before admission, were transferred from a nonstudy hospital, or had suffered an AMI in the previous 2 weeks. For the present study, we identified a subgroup comprising 1,206 patients (41%) with previously established CAD, based on the presence of chronic angina, prior AMI, or prior revascularization (percutaneous transluminal coronary angioplasty [PTCA] or coronary artery bypass grafting [CABG]) documented at admission. We considered this subgroup to have been eligible, prior to their admission, for interventions aimed at the secondary prevention of CAD.

Of these 1,206 patients, we identified 622 patients (52%) who were eligible for the treatment of hyperlipidemia, according to the NCEP II Guidelines for Secondary Prevention,⁸ including those already treated with LLDs at admission ($n = 230$) or patients with a total cholesterol value of 200 mg/dL or greater ($n = 392$) on the day of admission. Total cholesterol values, collected within 24 hours of admission, have previously been shown to reliably reflect a patient's usual serum cholesterol status.¹⁵⁻¹⁷ We did not have information regarding fasting status, specific lipid subfractions, or the laboratory methods used to determine lipid levels. We excluded patients with measured total cholesterol less than 200 mg/dL ($n = 418$), no serum lipid measurements ($n = 147$), missing data ($n = 13$), or known contraindications to LLDs (hepato-biliary disease, $n = 6$).

Patient Outcomes

We classified each LLD as a statin, fibrate, niacin, resin, or any combination thereof. We collapsed these classes into a binary variable (using LLDs or not using LLDs at the time of admission) that was our primary out-

come of interest. Our secondary outcome was the successful achievement of NCEP II cholesterol goals (a total cholesterol <160 mg/dL, approximately equivalent to a low-density lipoprotein level <100 mg/dL)⁸ among patients using LLDs. In addition, we studied the achievement of a less strict goal, a total cholesterol <200 mg/dL (approximately a low-density lipoprotein level <130 mg/dL). All categorizations of total cholesterol values, and approximate equivalencies to low-density lipoprotein levels, were based on the NCEP II Guidelines.⁸

Patient Variables

Nonclinical variables included age (<55, 55-64, 65-74, or >74 years), gender, race (white or nonwhite), marital status (married/common-law or alone), residence (urban or rural), employment (employed, unemployed, or retired), and health insurance arrangement (fee for service including Medicaid, Blue Cross, Medicare, or other commercial coverage; or managed care including Medicare HMO).

Clinical variables documented at admission included a history of angina, AMI, PTCA, CABG, congestive heart failure, hypertension, diabetes mellitus, smoking status (never, current, or past smoker), body mass index (<24, 24-27, 28-31, or >31 kg/m²), and the number of traditional cardiac risk factors (0-4 factors including smoking, hypertension, obesity [body mass index >28 kg/m²], and diabetes mellitus). Comorbidity was assessed by both the number of noncardiac morbidities (0, 1, 2, or >2) and the use of Greenfield's Index of Co-Existent Disease (ICED).¹³ This index has components for coexistent diseases and their severity based on physical impairment and functional status. We graded each component on an ordinal scale and then combined them into a final 4-point scale, ranging from no (ICED = 0) to severe (ICED = 3) comorbidity.^{13,18} On the basis of previous work,¹³ we dichotomized this scale into the absence (ICED = 0, 1, 2) or presence (ICED = 3) of severe comorbidity. Drug variables included the names and total number of medications at admission (<4, 4-6, or >6, less LLDs).

Data Collection and Integrity

Nurses experienced in the care of AMI patients collected data from medical records using a standardized abstraction instrument. Abstractors were required to demonstrate initial and ongoing interrater agreement with a criterion review of 95% or higher.^{11,14} Retrospective audits of a random sample of 10% of each abstractor's completed cases were conducted to ensure that each abstractor met and maintained the data quality standard of 95% all-item agreement with a study auditor.^{11,14}

Statistical Analysis

We classified eligible study patients as either using LLDs or not using LLDs. Potential categorical predictors were

examined using χ^2 statistics. Age, gender, and univariate predictors with $p < .2$ were considered for further analysis. Logistic regression models were used to determine the predictors independently associated with LLD use. Models initially included terms for patient age, gender, marital status, location of residence, health insurance, number/type of traditional cardiac risk factors, prior revascularization (PTCA or CABG), the number/severity of comorbidities, and medications at admission (aspirin, β -blockers, and total number).

Final models, using patients with complete data (90%), were constructed with stepwise logistic regression available in SAS.¹⁹ Potential confounding was addressed by serially introducing each nonsignificant univariate predictor back into the final model and assessing for any important change (greater than 10%) in the β -coefficients of model terms. The final model was subjected to a bootstrap analysis (1,000 cycles, with replacement) that revealed no evidence of overfitting.²⁰

Because so few patients using LLDs achieved a total cholesterol level below 160 mg/dL (our secondary outcome), there was insufficient statistical power for multivariate analysis. Thus, we report only frequencies and significant univariate associations.

RESULTS

Patient Sample

For the 622 study patients, the mean age was 66.4 years, 37% were female, and most (88%) were white (Table 1). At the time of admission, 29% of patients had severe comorbidities. Only 37% of patients ($n = 230$) were using LLDs at the time of admission, primarily monotherapy with a statin. Twenty-four percent of treated patients, and 40% of untreated patients, were frankly hyperlipidemic (total cholesterol >240 mg/dL). Cholesterol profiles stratified by treatment status and details of lipid-lowering treatments are presented in Table 2.

Univariate Analysis

In univariate analysis, both nonclinical (Table 3) and clinical (Table 4) variables were significantly associated with the use of LLDs. The relation between LLD use and age took the shape of an inverted U; use was lowest among those younger than 55 years (34%) and those older than 74 years (31%), in contrast to patients aged 55 to 64 years (39%) or aged 65 to 74 years (45%). Women were as likely to be treated as men (33% compared with 39%; odds ratio [OR] 0.76; 95% confidence interval [CI] 0.54, 1.10). LLDs were used more often by patients who belonged to managed care plans than fee-for-service patients (43% compared with 35%; OR 1.42; 95% CI 1.00, 2.03).

Significant clinical variables are presented in Table 4. No single cardiac risk factor was significantly associated with the use of LLDs, except for smoking status; compared with current smokers or never smokers, past smokers were more likely to use LLDs. Patients who used other

Table 1. Characteristics of Patients Eligible for Secondary Prevention of Coronary Artery Disease Using Lipid-Lowering Drugs

Variable	Number (%) ($n = 622$)
Nonclinical	
Age, years	
<55	138 (22)
55–64	132 (21)
65–74	168 (27)
>74	184 (30)
Female	230 (37)
Age >64 years	
Female	177 (28)
Male	175 (28)
Age >74 years	
Female	115 (18)
Male	69 (11)
White	454 (88)
Married or common-law	445 (72)
Urban resident	454 (73)
Retired	303 (49)
Managed care	177 (28)
Clinical	
Prior myocardial infarction	282 (45)
Prior PTCA or CABG*	200 (32)
Congestive heart failure	94 (15)
Severe comorbidity present†	178 (29)
Hypertension	392 (63)
Diabetes mellitus	152 (24)
Mean body mass index, kg/m ² (\pm SD)	28.6 (\pm 5.1)
Obesity (body mass index >28 kg/m ²)	412 (66)
Current smoker	155 (25)
Past smoker	367 (59)
Mean number of risk factors (\pm SD)‡	2.3 (\pm 1.0)
Aspirin use	285 (46)
β -Blocker use	141 (23)
Lipid-lowering drug use	230 (37)
Mean number of medications (\pm SD)	3.8 (\pm 2.8)

*PTCA indicates percutaneous transluminal coronary angioplasty; CABG, coronary artery bypass grafting.

†Based on Greenfield's Index of Coexistent Disease.^{13,18}

‡Risk factors are hypertension, diabetes, obesity, or smoking (from 0 to 4).

medications for secondary prevention, such as aspirin or β -blockers, or who used four or more medications at the time of admission, were more likely to use a LLD. Although a greater number of comorbidities was associated with LLD use, in univariate analysis the presence of severe comorbidity was not ($p = .97$).

Of the 230 patients who used LLDs, 149 (65%) also had serum lipids measured. Only 15% of these patients achieved the NCEP II goal of a total cholesterol level below 160 mg/dL, and 18% needed combination therapy to do so (Table 2). Patients who used two or more LLDs were more likely to achieve NCEP II goals than patients who used only one LLD (31% successful compared with 14%; OR 2.91; 95% CI 0.81, 10.48). Even with a less strict target

Table 2. Characteristics of Hyperlipidemia in Patients Eligible for Secondary Prevention, Stratified by Lipid-Lowering Treatment*

Variable	Untreated Number (%) (n = 392)		Treated Number (%) (n = 149)	
Total cholesterol >240 mg/dL	155	(40)	36	(24)
Total cholesterol >200 mg/dL	392	(100) [†]	73	(49)
Mean total cholesterol (±SD)	242.0 (±40.5)		209.1 (±50.4)	
Median total cholesterol	233		201	
25th, 75th percentile	214	255	178	238
10th, 90th percentile	206	292	154	279
Lipid-lowering drugs used	0	(0) [†]	149	(100)
Statins			109	(73)
Fibrates			15	(10)
Niacin			11	(7)
Resins			1	(1)
Any combination			13	(9)
Total cholesterol <200 mg/dL achieved	0	(0) [†]	76	(51)
Statins			55	(37)
Fibrates			8	(5)
Niacin			6	(4)
Resins			1	(1)
Any combination			6	(4)
NCEP II goals achieved [‡] (total cholesterol <160 mg/dL)	0	(0) [†]	22	(15)
Statins			15	(10)
Fibrates			1	(1)
Niacin			2	(1)
Resins			0	(0)
Any combination			4	(3)

*Patients who used lipid-lowering drugs (n = 230) and had lipids measured (n = 149).

[†]By study design.

[‡]NCEP II indicates National Cholesterol Education Program-Adult Treatment Panel II.⁸

(total cholesterol <200 mg/dL), only half the patients were successful in achieving lipid goals (Table 2).

Although 392 patients were identified, during hospitalization, as needing their serum lipids lowered (622 patients eligible, less the 230 patients using LLDs), at the time of discharge only 43 (11%) were prescribed LLDs.

Multivariate Analysis

In multivariate analysis (Table 5), patients older than 74 years were less likely to use LLDs than younger patients (adjusted OR 0.55; 95% CI 0.35, 0.88). The association remained significant after adjustment for all terms in the model, including female gender and the presence of severe comorbidity. Patients who were members of a man-

Table 3. Nonclinical Variables Associated with Using Lipid-Lowering Drugs (Univariate Analysis)

Variable	Number (%) (n = 230)	p (χ ²)
Age, years		.037
<55	47 (34)	
55-64	51 (39)	
65-74	75 (45)	
>74	57 (31)	
Gender		.11
Male	154 (39)	
Female	76 (33)	
Race		.30
White	208 (38)	
Nonwhite	5 (31)	
Unknown or missing	17 (28)	
Marital status		.035
Married or common-law	176 (40)	
Alone	54 (31)	
Residence		.13
Urban	176 (39)	
Rural	54 (32)	
Insurance arrangement		.052
Fee-for-service	134 (35)	
Managed care	76 (43)	
Unknown or missing	20 (31)	

aged care plan were more likely to use LLDs than similar fee-for-service patients.

Among the clinical variables, prior revascularization, aspirin use, and the use of four or more medications were positively associated with the use of LLDs (Table 4). Although the number of noncardiac comorbidities was not significant in multivariate analysis, after controlling for age, gender, and medication use, the presence of severe comorbidity was associated with LLD use (adjusted OR 0.60; 95% CI 0.38, 0.95).

In fuller exploratory models, we found no evidence of confounding by, or interaction with, race, marital status, employment, residence, diabetes, obesity, hypertension, number of cardiac risk factors, prior myocardial infarction, congestive heart failure, or the number of comorbidities.

DISCUSSION

Patients with both established CAD and lipid abnormalities have a mortality risk 10 times greater than that of patients without overt disease who have similar lipid abnormalities.²¹ Despite these widely documented risks, our study found that one third of eligible patients were using LLDs at the time of admission for AMI. Of those treated, only a minority actually achieved recommended lipid goals. Elderly patients, independent of associated comorbidities, were at particular risk of undertreatment. Women were as likely to be treated as men, but patients enrolled in managed care plans were more likely to be

Table 4. Clinical Variables Associated with Using Lipid-Lowering Drugs (Univariate Analysis)

Variable	Number (%) (n = 230)	p (χ^2)
Cigarette use		.011
Never	33 (33)	
Past smoker	153 (42)	
Current smoker	44 (28)	
Number of risk factors*		.081
<2	49 (35)	
2	72 (34)	
3	76 (38)	
4	33 (49)	
Prior PTCA or CABG [†]		.001
Absent	115 (27)	
Present	115 (56)	
Aspirin		.001
Absent	92 (27)	
Present	138 (48)	
β -Blockers		.001
Absent	158 (33)	
Present	72 (51)	
Number of medications		.001
<4	53 (19)	
4-6	99 (48)	
>6	78 (60)	
Number of comorbidities [‡]		.001
0	17 (15)	
1	76 (37)	
2	79 (47)	
>2	58 (41)	
Severe comorbidity (ICED) [‡]		.97
Absent	164 (37)	
Present	66 (37)	

*Risk factors = smoking, diabetes, obesity, or hypertension (from 0 to 4).

[†]PTCA indicates percutaneous transluminal coronary angioplasty; CABG, coronary artery bypass grafting.

[‡]Based on Greenfield's Index of Co-existent Disease.^{13,18}

treated than fee-for-service patients. Overall, if all of the eligible but untreated patients had used LLDs for 5 years before admission, approximately 25 AMIs or cardiovascular deaths might have been prevented.²²

Others have previously documented similar underuse of different proven effective therapies, such as aspirin and β -blockers, for secondary prevention.²³⁻²⁷ Using a variety of data sources, investigators have estimated that between 30% and 40% of CAD patients eligible for LLDs are actually using them.^{6,21,23-26,28} Our study supports this magnitude of undertreatment, but also allows us to examine some of the clinical and nonclinical correlates of treatment with LLDs.

Furthermore, in this study, treatment with LLDs did not guarantee adequate control of hyperlipidemia. Only 15% of patients using LLDs had actually achieved recommended NCEP II secondary prevention goals at the time of their hospitalization. This is equivalent to the success rate achieved

Table 5. Variables Independently Associated with Using Lipid-Lowering Drugs (Multiple Logistic Regression)

Variable	Odds Ratio	(95% Confidence Interval)	p (χ^2)
Age >74 years	0.55	(0.35, 0.88)	.013
Female	0.88	(0.57, 1.37)	.58
Managed care*	1.56	(1.02, 2.39)	.043
Past smoker [†]	1.72	(0.98, 3.01)	.058
Current smoker [†]	0.91	(0.46, 1.81)	.78
Prior PTCA or CABG [‡]	2.31	(1.51, 3.53)	<.0001
Aspirin use	1.59	(1.07, 2.38)	.023
Number of medications [§]	2.89	(2.19, 3.84)	<.0001
Severe comorbidity	0.60	(0.38, 0.95)	.028

*Referent group is fee-for-service.

[†]Referent group is never smoker.

[‡]PTCA indicates percutaneous transluminal coronary angioplasty; CABG, coronary artery bypass grafting.

[§]Referent group is <4 medications.

in the control arm ("usual care") of a controlled trial of case management for risk factor modification after AMI,²⁹ and comparable to other low reported rates of success in achieving NCEP II goals.^{25,26,30,31} Nevertheless, although recommended goals are rarely achieved, any lowering of serum cholesterol in this population appears beneficial.⁶

Advanced age (>74 years) was a significant risk factor for undertreatment. Although there is some controversy about the strength of the association between elevated cholesterol and the risk of CAD in elderly patients,^{3,32-34} the main cause of death in older patients is still CAD. Their 1-year mortality following AMI is about 30%, not the commonly acknowledged 5% mortality of younger patients enrolled in clinical trials.³⁵ With such a high baseline mortality risk, the absolute benefit of lowering cholesterol in these older patients should be equal to, if not greater than, that for younger patients. For example, a recent study of 665 elderly patients (mean age, 72 years) found a significant (32%) reduction in cardiovascular events in those with known CAD who were treated with standard doses, compared with fixed low doses, of pravastatin.³⁶ In fact, the NCEP II Guidelines suggest extrapolation of the current evidence to the elderly,⁸ until proof of benefit is established. The fact that older age continues to be such a strong predictor of underuse, even after multivariate adjustment, implies that some of the decision not to use LLDs may be related to the age of the patient, in and of itself. We do not know if the undertreatment of hyperlipidemia in older patients is due to decisions by patients or by their physicians. However, patient demand is a powerful determinant of physician treatment decisions, and preventive services are provided less often than patients prefer or experts recommend.^{37,38} Even after controlling for available indicators of health-seeking behavior (number of medications and illnesses,³⁸ and smoking cessation), older patients were undertreated.

We found patients with severe comorbidity were less likely to use LLDs for the prevention of chronic CAD. We previously reported patients with severe comorbidity were less likely to receive AMI treatments, such as aspirin and thrombolytic therapy.¹³ In both studies, patients with mild and moderate comorbidity were indistinguishable from those without comorbidity. Thus, it appears that only high levels of concomitant illness influence physician decision making. This association has been previously noted in the contexts of forgoing or withdrawing life-sustaining therapy in critically ill patients,^{39,40} decreased use of screening for cancer in patients with significant comorbidity,⁴¹ and undertreatment of unrelated disorders in patients with chronic illnesses.⁴²

Patients enrolled in some form of capitated managed care plan (primarily network models) were more likely than similar fee-for-service patients to use LLDs. This is consistent with previous studies that suggest managed care plans may provide more comprehensive preventive services.^{43,44} Possible mechanisms for this effect are better continuity of care, easier access to primary care physicians or specialists, use of information systems and treatment protocols, prescription drug benefits, or self-selection of health-seeking and compliant patients into managed care plans.

Our study has several limitations. Most importantly, we do not know if patients were offered and declined, or started and discontinued, LLDs as outpatients. Although we are unaware of any data addressing the rate of patient refusal of LLDs when offered, long-term rates of compliance to statin drug therapy have been reported to be 65% to 85% in observational studies^{45,46} and as high as 89% to 94% in clinical trials.^{9,47,48} In addition, we based our definition of hyperlipidemia (and achievement of NCEP II goals) on single values of total cholesterol, rather than multiple determinations of lipoprotein subfractions. Because lipid subfractions were not available, we may have misclassified a few patients; those with elevated low-density lipoprotein levels who had a total cholesterol level less than 200 mg/dL and patients with a total cholesterol level greater than 200 mg/dL but decreased low-density lipoproteins and elevated high-density lipoprotein levels. Also, we had no reliable dose or compliance data. Thus, we do not know if the inability to achieve lipid goals was due to inadequate doses of single drugs, underuse of multiple drugs, or noncompliance. Finally, we do not know if we can generalize these results obtained from patients admitted to 37 community hospitals in one state. We have no reason to believe that our findings are not representative of current patterns of practice in the United States^{25,26,28}; in addition, the rates of use of effective therapies for AMI in this population were comparable to those reported from similar populations in recent studies.^{10,35}

In conclusion, LLDs for the secondary prevention of CAD were underused in this at-risk population. Older patients were at particular risk of undertreatment, but other nonclinical and clinical variables were important determi-

nants of treatment. Even when used, nationally recommended lipid goals were rarely achieved. Consistent with the NCEP II guidelines, newer studies published since these data were collected have demonstrated the benefit of aggressive lipid-lowering for secondary prevention.^{47,48}

The method we used to identify patients with hyperlipidemia—namely, a single routine measurement of total cholesterol at admission—could be easily used for all AMI patients.^{15–17} Because diet alone is unlikely to achieve recommended lipid goals in this population,^{49,50} initiating treatment with LLDs during hospitalization may be justified.^{5,6,8} This may help maintain continuity of care and continuity of therapeutic intent, particularly when a different physician is responsible for care after discharge from the hospital. All of the patients we studied might be considered “failures” of secondary prevention—each of them had known CAD and hyperlipidemia in the community, and then had another CAD-related event requiring hospitalization. It is sobering to note that of the 392 easily identified high-risk patients with untreated hyperlipidemia, only a tenth were actually discharged with a prescription for LLDs. In addition to prompt diet and behavior modification, we believe starting treatment with LLDs before discharge may be a simple and effective intervention with the potential to substantially reduce cardiovascular morbidity and mortality.

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REFERENCES

1. Soumerai SB. Factors influencing prescribing. *Aust J Hosp Pharm.* 1988;18(3):9–16.
2. Lamas GA, Pfeffer MA, Hamm P, et al. Do the results of randomized trials of cardiovascular drugs influence medical practice? *N Engl J Med.* 1992;327:241–7.
3. La Rosa JC, Hunninghake D, Bush D, et al. The cholesterol facts—a summary of the evidence relating dietary fats, serum cholesterol, and coronary heart disease. *Circulation.* 1990;81:1721–33.
4. Gaziano JM, Hebert PR, Hennekens CH. Cholesterol reduction: weighing the benefits and risks. *Ann Intern Med.* 1996;124:914–8.
5. Kjekshus J, Pedersen TR, Tobert JA. Lipid lowering therapy for patients with or at risk of coronary artery disease. *Curr Opin Cardiol.* 1996;11:418–27.
6. Gotto AM Jr. Cholesterol management in theory and practice. *Circulation.* 1997;96:4424–30.
7. Davignon J, Montigny M, Dufour R. HMG CoA reductase inhibitors: a look back and a look ahead. *Can J Cardiol.* 1992;8:843–88.
8. The Expert Panel. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel II). *JAMA.* 1993;269:3015–23.

9. Scandinavian Simvastatin Survival Study Group. Randomized trial of cholesterol lowering in 4444 with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet*. 1994; 344:1383-9.
10. Tsuyuki RT, Teo KK, Ikuta RM, Bay KS, Greenwood PV, Montague TJ. Mortality risk and patterns of practice in 2,070 patients with acute myocardial infarction, 1987-92. *Chest*. 1994;105:1687-92.
11. McLaughlin TJ, Soumerai SB, Willison DJ, et al. Adherence to national guidelines for drug treatment of suspected acute myocardial infarction. *Arch Intern Med*. 1996;156:799-805.
12. Ayanian JZ, Epstein AM. Differences in the use of procedures between women and men hospitalized for coronary heart disease. *N Engl J Med*. 1991;325:221-5.
13. McLaughlin TJ, Soumerai SB, Willison DJ, et al. The effect of comorbidity on use of thrombolysis or aspirin in acute myocardial infarction patients eligible for treatment. *J Gen Intern Med*. 1997;12:1-6.
14. Soumerai SB, McLaughlin TJ, Gurwitz JH, et al. Effect of local medical opinion leaders on quality of care for acute myocardial infarction: a randomized controlled trial. *JAMA*. 1998;279:1358-63.
15. Gore JM, Goldberg RJ, Matsumoto AS, Castelli WP, McNamara PM, Dalen JE. Validity of serum total cholesterol level obtained within 24 hours of acute myocardial infarction. *Am J Cardiol*. 1984;54:722-5.
16. Ryder REJ, Hayes TM, Mulligan IP, Kingswood JC, Williams S, Owens DR. How soon after myocardial infarction should plasma lipid values be assessed? *BMJ*. 1984;289:1651-3.
17. Ahnve S, Angelin B, Edhag O, Berglund L. Early determination of serum lipids and apolipoproteins in acute myocardial infarction: possibility for immediate intervention. *J Intern Med*. 1989;226: 297-301.
18. Greenfield S, Apolone G, McNeil BJ, Cleary PD. The importance of coexistent disease in the occurrence of postoperative complications and one-year recovery in patients undergoing total hip replacement: comorbidity and outcomes after hip replacement. *Med Care*. 1993;31:141-54.
19. SAS Institute. SAS/STAT User's Guide. Version 6. 4th ed. Vol. 2. Cary, NC: SAS Institute; 1989.
20. Efron B, Gong G. A leisurely look at the bootstrap, the jackknife, and cross-validation. *Am Statistician*. 1983;37:36-48.
21. Brown BG, Zhao X, Bardsley J, Albers JJ. Secondary prevention of heart disease amongst patients with lipid abnormalities: practice and trends in the United States. *J Intern Med*. 1997;241:283-94.
22. Rembold CM. Number-needed-to-treat analysis of the prevention of myocardial infarction and death by antidiabetic therapy. *J Fam Pract*. 1996;42:577-86.
23. Vogel RA. Risk factor intervention and coronary artery disease: clinical strategies. *Coron Artery Dis*. 1995;6:466-71.
24. ASPIRE Steering Group. A British Cardiac Society survey of the potential for the secondary prevention of coronary disease: ASPIRE (Action on Secondary Prevention through Intervention to Reduce Events), principal results. *Heart*. 1996;75:334-42.
25. Pearson TA, Peters TD, Feury D. Comprehensive risk reduction in coronary patients: attainment of goals of the AHA guidelines in U.S. patients. *Circulation*. 1997;96 (suppl):I-733.
26. McBride P, Schrott HG, Plane MB, Underbakke G, Brown RL. Primary care practice adherence to National Cholesterol Education Program Guidelines for patients with coronary heart disease. *Arch Intern Med*. 1998;158:1238-44.
27. Soumerai SB, McLaughlin TJ, Spiegelman D, Hertzmark E, Thi-beault G, Goldman L. Adverse outcomes of underuse of beta-blockers in elderly survivors of acute myocardial infarction. *JAMA*. 1997;277:115-21.
28. Stafford RS, Blumenthal D, Pasternak RC. Variations in cholesterol management practices of U.S. physicians. *J Am Coll Cardiol*. 1997;29:139-46.
29. Debusk RF, Houston-Miller N, Superko R, et al. A case-management system for coronary risk factor modification after acute myocardial infarction. *Ann Intern Med*. 1994;120:721-9.
30. Schrott HG, Bittner V, Vittinghoff E, et al. Adherence to National Cholesterol Education Program treatment goals in postmenopausal women with heart disease. *JAMA*. 1997;277:1281-6.
31. Marcelino JJ, Feingold KR. Inadequate treatment with HMG CoA reductase inhibitors by health care providers. *Am J Med*. 1996; 100:605-10.
32. Krumholz HM, Seeman TE, Merrill SS, et al. Lack of association between cholesterol and coronary heart disease mortality and morbidity and all-cause mortality in persons older than 70 years. *JAMA*. 1994;272:1335-40.
33. Hulley SB, Newman TB. Cholesterol in the elderly: is it important? *JAMA*. 1994;272:1372-4.
34. Corti MC, Guralnik JM, Salive ME, et al. Clarifying the direct relation between total cholesterol and death from coronary heart disease in older persons. *Ann Intern Med*. 1997;126:753-60.
35. Marciniak TA, Ellerbeck EF, Radford MJ, et al. Improving the quality of care for Medicare patients with acute myocardial infarction: results from the Cooperative Cardiovascular Project. *JAMA*. 1998;279:1351-7.
36. Ouchi Y, Ohashi Y, Ito H, et al. Serum cholesterol lowering by pravastatin reduces cardiovascular events in the elderly with hypercholesterolemia: overall and age-related analyses of the results from the PATE study. *Circulation*. 1997;96 (suppl):I-66.
37. Pearson TA, McBride PE, Houston-Miller N, Smith SC Jr. Organization of preventive cardiology service. *J Am Coll Cardiol*. 1996; 27:1035-47.
38. Kottke TE, Blackburn H, Brekke ML, Solberg LI. Making time for preventive services. *Mayo Clin Proc*. 1993;68:785-91.
39. Smedire NG, Evans BH, Grais LS, et al. Withholding and withdrawal of life support from the critically ill. *N Engl J Med*. 1990; 322:309-15.
40. Hanson LC, Danis M. Use of life-sustaining care for the elderly. *J Am Geriatr Soc*. 1991;39:772-7.
41. Fontana SA, Baumann LC, Helberg C, Love RR. The delivery of preventive services in primary care practices according to chronic disease status. *Am J Public Health*. 1997;87:1190-6.
42. Redelmeier DA, Tan SH, Booth GL. The treatment of unrelated disorders in patients with chronic medical diseases. *N Engl J Med*. 1998;338:1516-20.
43. Berwick DM. Payment by capitation and the quality of care. *N Engl J Med*. 1996;335:1227-31.
44. Emanuel EJ, Dubler NN. Preserving the physician-patient relationship in the era of managed care. *JAMA*. 1995;273:323-9.
45. Avorn J, Monette J, Lacour A, et al. Persistence of use of lipid-lowering medications. *JAMA*. 1998;279:1458-62.
46. Andrade SE, Walker AM, Gottlieb LK, et al. Discontinuation of antihyperlipidemic drugs—do rates reported in clinical trials reflect rates in primary care settings? *N Engl J Med*. 1995;332: 1125-31.
47. Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. *N Engl J Med*. 1996;335:1001-9.
48. LIPID Study Group. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. *N Engl J Med*. 1998;339: 1349-57.
49. Kannel WB. Preventive efficacy of nutritional counseling. *Arch Intern Med*. 1996;156:1138-9.
50. Hunninghake DB, Stein EA, Dujovne CA, et al. The efficacy of intensive dietary therapy alone or combined with lovastatin in outpatients with hypercholesterolemia. *N Engl J Med*. 1993;328: 1213-9.