Improving Lipid Evaluation and Management in Medicare Patients Hospitalized for Acute Myocardial Infarction

Monte Malach, MD, MACP, FACC; John Quinley, MD, MPH; Pascal James Imperato, MD, MACP, MPH & TM; Marcia Wallen, MPH

Background: The control of low-density lipoprotein cholesterol (LDL-C) levels in patients with known coronary artery disease, particularly in those with acute myocardial infarction, has been shown to reduce the rates of disease progression, recurrent events, and mortality.

Objectives: To evaluate and improve hospital-based processes for measuring and treating, when necessary, LDL-C levels above 3.36 mmol/L (130 mg/dL) in patients with an acute myocardial infarction.

Design: A nonrandomized retrospective baseline study followed by a collaborative educational intervention with participating hospitals and a second nonrandomized postintervention study.

Patients: Four hundred six preintervention patients discharged from the hospital alive after a confirmed acute myocardial infarction in 1996, and 498 postintervention patients discharged from the hospital in 1999.

Interventions: Performance of lipid profiles on admission to the hospital and during hospitalization and drug and dietary interventions.

Results: The measurement of LDL-C level on admission to the hospital increased from 8% preintervention in 1996 to 32% postintervention in 1999. The measurement during hospitalization increased from 14% preintervention to 48% postintervention. Hospitals that initiated programs to ensure early lipid evaluations through preprinted orders and policy changes achieved an average patient LDL-C measurement rate of 70% in 1999. Hospitals lacking standard policies averaged only 23% at the same time. Of the patients with a measured LDL-C level greater than 3.36 mmol/L (130 mg/dL) who were not undergoing drug therapy on admission to the hospital, 46% were given lipid-lowering agents by discharge from the hospital during the postintervention period. During this same period, only 11% of the patients were prescribed this therapy if they had either a lower measured level or no LDL-C measurement at all.

Conclusion: Active hospital-based programs to ensure routine LDL-C measurements in patients admitted for acute myocardial infarction increased the use of appropriate lipid-lowering therapy in these high-risk individuals and could contribute to reducing the incidence of recurrent coronary artery disease.

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A acute myocardial infarction (AMI) is a leading cause of morbidity and mortality among Medicare patients. In recent years, the management of patients with AMI has emphasized the use of aspirin, β-blockers, and thrombolytic agents or percutaneous transluminal coronary angioplasty during the first hours of onset. Despite the existence of clear guidelines, not all hospitals have processes in place to ensure their timely implementation. Lowering serum lipid levels to normal levels has been shown to be a major contributor to reducing the rate of disease progression, recurrent events, and mortality among patients with documented coronary artery disease (CAD). Practice guidelines that address the management of lipids in these patients have been established by the American College of Cardiology/American Heart Association and by the National Institutes of Health’s National Cholesterol Education Program. Screening and intervention for the secondary prevention of recurrent coronary events, including dietary education and therapy for lipemia, can significantly improve the long-term prognosis for patients with an AMI. It is suggested that these measures be implemented during the predischarge phase of hospitalization. Despite such recommendations, patients with an AMI often do not undergo lipid screening and appropriate interventions while hospitalized. Frolkis recently reported that at 1

From IPRO, Lake Success, NY (Drs Malach, Quinley, and Imperato and Ms Wallen); the Department of Medicine, New York University Medical Center, New York (Dr Malach); and the Departments of Medicine (Dr Malach) and Preventive Medicine and Community Health (Dr Imperato), State University of New York, Health Science Center at Brooklyn.
PATIENTS AND METHODS

Hospitals in the state of New York interested in improving the level of measurement and management of elevated lipid levels in Medicare patients who experienced an AMI were recruited into this study during the second quarter of 1998. Twenty hospitals eventually agreed to participate.

Four hundred twenty-eight Medicare patients from the 20 participating hospitals were included in the baseline phase of the study. These patients had been discharged alive from these hospitals in July, August, November, and December of 1996. Discontinuous months were chosen to account for possible variations in seasonality for AMI. The medical records of these patients had been included in the Health Care Financing Administration’s Cooperative Cardiovascular Project.

These participants also had to be discharged alive, as part of the study. These patients had been discharged alive from 20 participating hospitals in the state of New York interested in improving care financing. Consecutive months were used for the remeasurement group since no seasonal variation was found on the baseline medical records.

Eligible participants for this study included Medicare patients discharged from the hospital with a principal diagnosis of AMI (International Classification of Diseases, Ninth Revision [ICD-9]), code 410.x1) in whom the AMI was confirmed on medical record review by an elevated creatine phosphokinase of muscle band or an elevated lactate dehydrogenase isoenzyme LD2>LD1 or by 2 of the following criteria: chest pain, a 2-fold elevation of the creatine phosphokinase level, or evidence of an AMI on an electrocardiogram. These participants also had to be discharged alive, as part of the Cooperative Cardiovascular Project.

The quality indicators chosen for this study are presented in the Table. These indicators were based on existing national guidelines, which stress the importance of total cholesterol and LDL-C measurements to assess lipid status. Because of an anticipated initial decrease in cholesterol and LDL-C levels associated with an AMI, separate measurement and therapy and increase participation by the key participants.

The total cholesterol level has been shown to be a risk factor for older patients (aged ≥65 years) with CAD, even after adjustment for other comorbid conditions. Despite this evidence, Medicare patients who experience an AMI and have lipemia are as a group undertreated for the latter. Overall, there is significant evidence that more patients with AMI and lipemia are not achieving ideal lipid levels as recommended by the National Cholesterol Education Program guidelines.
Randomized clinical trials, a meta-analysis of previous clinical trials, and angiographic studies have documented the benefits of LDL-C lowering in patients with CAD.9 The Scandinavian Simvastatin Survival Study10 found that, compared with patients who received routine medical care, patients who received a cholesterol-lowering agent had a nearly 70% reduction in major cardiac events. A meta-analysis8 of secondary cholesterol intervention trials found that aggressive cholesterol lowering resulted in a 31% reduction in rates of fatal and non-fatal reinfarction and a 21% reduction in all causes of mortality. Thus, it is clear that secondary prevention strategies can reduce morbidity and mortality. Despite the evidence supporting secondary prevention, it is often not implemented. A recent cross-sectional analysis by Majumdar et al11 demonstrated that lipid-lowering drugs were used in only 37% of patients with established CAD and lipemia who were hospitalized with an AMI.

Given the importance of evaluating lipid levels in patients hospitalized with an AMI and of initiating appropriate therapy when indicated, we sought to assess hospital-based processes for achieving these goals. We also implemented a collaborative educational intervention program with 20 hospitals to improve these processes and conducted a postintervention assessment to measure its outcomes. This project was undertaken by IPRO, the peer review agent for Medicare in the state of New York, as part of the federal Health Care Financing Administration's Health Care Quality Improvement Program.

### RESULTS

The baseline study demonstrated that relatively few patients had an LDL-C determination either at the time of admission to the hospital or during their hospital stay (Table). Only 2 of the 20 participating hospitals had processes in place for routine LDL-C measurements during hospitalization at the time of the baseline study. The rates documented for these hospitals were 48% and 79%. Of the remaining 18 hospitals that did not have such routine processes in place, none exceeded an LDL-C measurement rate of 30%.

Low-density lipoprotein cholesterol measurements on admission to the hospital and during hospitalization significantly increased to 32% and 48%, respectively, during the postintervention period, from baseline levels of 8% and 14%, respectively (Table). Despite these significant improvements, rates varied greatly between hospitals (Figure).

Each participating hospital described the type of improvement program implemented by the time of follow-up. Five hospitals (25%) either implemented preprinted orders for LDL-C level in the cardiac care unit or emergency department or had a strong program engaging patients in tracking their own LDL-C levels. Eight hospitals (40%) reported adopting a lipid measurement policy, protocol, or clinical pathway and educating staff to order lipid levels, but without preprinted orders or a patient self-monitoring program. Seven hospitals (35%) reported that they were not able to adopt any substantial program to improve the ordering of the LDL-C level. The rates of LDL-C measurement during hospitalization of these 3 groups were 70%, 53%, and 23% (P < .01 for each comparison).

The rate of total cholesterol measurement on admission to the hospital was high (72%) at baseline, but declined in the postintervention period to 54%. The rate of total cholesterol measurement during hospitalization was 78% at baseline, but also declined to a lower level (66%) in the postintervention period.

As shown in the Table, 15% of the patients in the baseline study were taking lipid-lowering agents at the time of admission to the hospital. This increased to 17% in the postintervention period, a difference that is not statistically significant.

Among patients who had LDL-C levels greater than 3.36 mmol/L (≥130 mg/dL), none were prescribed lipid-lowering agents in the baseline period. However, this significantly increased to 46% during the postintervention period.
Lipid-lowering therapy before admission to the hospital was much more common for patients with a history of CAD. Of 498 patients in the postintervention sample, 20% with a history of CAD were taking lipid-lowering medication on admission to the hospital compared with 8% of the other patients. Therapy was also strongly influenced by age, with 22% of the patients younger than 75 years undergoing therapy vs 13% of those aged 75 to 84 years; only 4% of those older than 85 years were undergoing therapy. This pattern persisted at the time of discharge from the hospital.

The results of this study demonstrated that hospitals that have a plan that includes an easy-to-use protocol, a care map, and/or preprinted order sheets are able to improve the rate at which the LDL-C level is measured in Medicare patients with an AMI. Those hospitals that did not adopt plans cited either internal opposition to routine LDL-C measurements in this setting or insufficient implementation time. The fact that hospitals without plans evidenced little change in rates between the baseline and postintervention periods speaks against these trends being due to a secular trend.

Based on the information provided by participating hospitals, it appears that most total cholesterol measurements during the baseline period were made as part of multichannel chemistry panels. During the intervening years, measurement of the total cholesterol level was removed from many admission panels, which may explain the decline observed in the postintervention period.

The rate of dietary therapy counseling varied considerably among hospitals. However, no appreciable change was found for this indicator in the postintervention period. Participating hospitals believed that dietary education was poorly captured in medical records, and none reported an active intervention to improve performance in this area.

Lipid-lowering therapy was higher in the postintervention period for patients with a history of CAD. Also, more patients with such a history were already taking lipid-lowering medication on admission to the hospital compared with other patients. Age was an important determinant of such therapy, with rates being higher for those younger than 75 years.

The lay public’s knowledge of cholesterol screening, hypercholesterolemia, and lipid-lowering treatment greatly increased from 1980 to 1992. Unfortunately, most dyslipemic patients receiving lipid-lowering therapy are not achieving National Cholesterol Education Program LDL-C target levels. Thus, more aggressive therapy is necessary to attain those goals, as demonstrated by the baseline findings of this study.

There still appears to be a sex disparity in the use of lipid-lowering therapy in patients with CAD, despite the greater attention that has been recently focused on this issue. The data from 16 academic centers in the United States and Canada over 3 years showed that lipid-lowering therapy increased in men by 55% vs 35% in women (P = .04) and that the target LDL-C goal of less than 3.36 mmol/L (<130 mg/dL) was reached in 31% of men but in only 12% of women (P = .001).

Published data have clearly shown that total cholesterol level is an independent risk factor in patients with CAD and that lowering it reduces the risk for subsequent AMI and death. Indeed, the Cholesterol and Recurrent Events (CARE) Study, the Long-Term Intervention With Pravastatin in Ischaemic Disease Study, and the Air Force/Texas Coronary Atherosclerosis Prevention Study found a significant reduction in recurring coronary events, AMI, and death. In the West of Scotland Coronary Prevention Study of 6595 men with an elevated total cholesterol level, but no clinical evidence of heart disease, pravastatin reduced the risk of a first heart attack by 31% and the need for revascularization by 37%.

The CARE study and the Air Force/Texas Coronary Atherosclerosis Prevention Study found reduced coronary events in patients with normal cholesterol levels. Age is no longer considered a reason to ignore total cholesterol levels.

The National Cholesterol Education Program report suggests that patients with established CAD or AMI should have the LDL-C level decreased to less than 2.59 mmol/L (<100 mg/dL). Furthermore, managed care organizations must have a Health Plan Employer Data Information Set performance measurement goal of an LDL-C level of less than 3.36 mmol/L (<130 mg/dL) to achieve National Committee for Quality Assurance endorsement.

Individuals with diabetes have a 2- to 10-fold increased risk of coronary events compared with those without diabetes; this risk is even higher in women with diabetes, according to the Framingham Study. Aggressive lipid-lowering therapy in diabetic patients lowered the risk of death and major coronary events in the Scandinavian Simvastatin Survival Study and in the CARE Study by 55% and 25%, respectively, compared with 23% in nondiabetic patients. Thus, it appears that the prevention of major CAD events in diabetic persons without evidence of CAD is even greater than that in nondiabetic persons with CAD.

Perhaps of greatest importance are the other, multiple benefits of statin therapy that are unrelated to lipid lowering. These benefits significantly enhance lipid lowering and further emphasize the importance of the evaluation of lipid levels in all patients after AMI, which is the subject of this report. The non–lipid-lowering actions include (1) plaque stability, (2) enhanced endothelial activity, (3) antiplatelet and antiatherothrombotic activity, and (4) anti-inflammatory and antimacrophage activity. Thus, there is an increasing and evolving role of statins in the management of atherosclerosis.

Rupture of a vulnerable coronary artery plaque is the most frequent cause of sudden unstable angina and AMI. A thin-walled fibrous cap over a liquid lipid core is subject to mechanical stress and infiltraton with inflammatory cells. Most acute coronary artery occlu-
sions occur in vessels with less than 50% stenosis or with noncritical stenoses.

Loss of endothelial activity and smooth muscle cell function results in stasis. Statins have been shown to improve or restore endothelial function and smooth muscle cell activity, unrelated to the effect on lipid levels. This activity translates into a direct clinical benefit. Improvement in coronary vasomotor function is seen within 6 months of therapy, and improved forearm blood flow is seen in hypercholesterolemic patients treated with statins for only 4 weeks.

Antiplatelet and antiatherothrombotic properties of some of the statins have been described. The prothrombotic factors affected by statins include plasma factor VII and cellular receptor VIIa, platelet aggregation, fibrinogen level, plasma viscosity, and a fibrinolytic factor. Platelets from patients with an elevated LDL-C level are more sensitive to aggregation than are platelets from normolipemic patients and patients receiving statin therapy.

Anti-inflammatory antimacrophage activity is promoted by the statins. Oxidized LDL-C attracts macrophages directly and stimulates binding to the endothelium via adhesion molecules. The statins simvastatin and lovastatin inhibit oxidation of LDL-C and reduce uptake by macrophages, thus blunting atheroma formation. Invasive macrophages promote plaque rupture by the release of enzymes (metalloproteinases), which digest and weaken the atheroma cap.

Aggressive medical therapy with atorvastatin (lowering the LDL-C level to a mean of 1.99 mmol/L [77 mg/dL]) compared with percutaneous transluminal coronary angioplasty, with or without stent use, reduced the incidence of ischemic events, overall mortality, and the need for intervention revascularizations. Statin monotherapy has also been shown to reduce the incidence of stroke in patients with CAD. Stroke or transient ischemic attack was prevented by 24% in the Scandinavian Simvastatin Survival Study, 31% in the CARE Study, and 19% in the Long-term Intervention With Pravastatin in Ischaemic Disease Study.

The present study demonstrated that active hospital policies to ensure the routine measurement and management of lipids in Medicare patients with an AMI may result in a significant reduction in coronary events, mortality, and stroke. It is essential to identify physician champions for the lipid program who set examples and who, by virtue of the respect with which they are held by the medical staff, can promote implementation. Those hospitals with the best practices included lipid panels on routine cardiac care unit admission for AMI and for chest pain protocols. These hospitals also have educational programs in place for physicians, nursing staff, and patients. The best-practice hospitals in this study performed ongoing monitoring of patients.

The value of benefits from the use of statins, other than lipid lowering, has been noted. This significantly emphasizes the need to determine lipid values for all patients with an AMI and a coronary event. The evidence that supports aggressive lipid modification in the secondary prevention of CAD morbidity and mortality is compelling. It is distressing to note the failure to use therapy that has been well documented to benefit patients with acute coronary syndromes and AMI. This is most particularly true in the more vulnerable Medicare population. The opportunity to dramatically affect the natural history of CAD demands the aggressive pursuit of interventions such as lipid determinations and lipid lowering. Indeed, it may be well stated that an individual with acute CAD categorically needs to have a lower LDL-C level.

Attention to initiating and sustaining appropriate lipid-lowering therapy can improve the health and longevity of the Medicare population.

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Corresponding author and reprints: Monte Malach, MD, MACP, FACC, IPRO, 1979 Marcus Ave, Lake Success, NY 11042 (e-mail: nypro.mmalach@sdps.org).

REFERENCES

2. Malach M, Quinley J, Rojas M. ABC (aspirin, beta blocker, clot buster thrombolytic or PTCA) for day one of acute myocardial infarction in Medicare patients [abstract]. J Am Coll Cardiol. 1999;33(suppl):338A.


