Measurement of low-density lipoprotein cholesterol levels in primary and secondary prevention patients: Insights from the PALM registry

Angela M. Lowenstern
*Duke University*

Shuang Li
*Duke University*

Ann Marie Navar
*Duke University*

Veronique L. Roger
*Mayo Clinic*

Jennifer G. Robinson
*University of Iowa*

See next page for additional authors

Follow this and additional works at: https://digitalcommons.wustl.edu/open_access_pubs

Please let us know how this document benefits you.

**Recommended Citation**


https://digitalcommons.wustl.edu/open_access_pubs/7387

This Open Access Publication is brought to you for free and open access by Digital Commons@Becker. It has been accepted for inclusion in Open Access Publications by an authorized administrator of Digital Commons@Becker. For more information, please contact vanam@wustl.edu.
Authors
Angela M. Lowenstern, Shuang Li, Ann Marie Navar, Veronique L. Roger, Jennifer G. Robinson, Anne C. Goldberg, Salim S. Virani, L. Veronica Lee, Peter W.F. Wilson, Michael J. Louie, Eric D. Peterson, and Tracy Y. Wang

This open access publication is available at Digital Commons@Becker: https://digitalcommons.wustl.edu/open_access_pubs/7387
Measurement of Low-Density Lipoprotein Cholesterol Levels in Primary and Secondary Prevention Patients: Insights From the PALM Registry

Angela M. Lowenstern, MD; Shuang Li, MS; Ann Marie Navar, MD, PhD; Veronique L. Roger, MD, MPH; Jennifer G. Robinson, MD, MPH; Anne C. Goldberg, MD; Salim S. Virani, MD, PhD; L. Veronica Lee, MD; Peter W. F. Wilson, MD; Michael J. Louie, MD, MPH, MSc; Eric D. Peterson, MD, MPH; Tracy Y. Wang, MD, MHS, MSc

Background—The 2013 American College of Cardiology/American Heart Association Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults recommended testing low-density lipoprotein cholesterol (LDL-C) to identify untreated patients with LDL-C \( \geq 190 \) mg/dL, assess lipid-lowering therapy adherence, and consider nonstatin therapy. We sought to determine whether clinician lipid testing practices were consistent with these guidelines.

Methods and Results—The PALM (Patient and Provider Assessment of Lipid Management) registry enrolled primary and secondary prevention patients from 140 US cardiology, endocrinology, and primary care offices in 2015 and captured demographic data, lipid treatment history, and the highest LDL-C level in the past 2 years. Core laboratory lipid levels were drawn at enrollment. Among 7627 patients, 2787 (36.5%) had no LDL-C levels measured in the 2 years before enrollment. Patients without chart-documented LDL-C levels were more often women, nonwhite, uninsured, and non–college graduates (all \( P < 0.01 \)). Patients without prior lipid testing were less likely to receive statin treatment (72.6% versus 76.0%; \( P = 0.0034 \)), a high-intensity statin (21.5% versus 24.3%; \( P = 0.016 \)), nonstatin lipid-lowering therapy (24.8% versus 27.3%; \( P = 0.037 \)), and had higher core laboratory LDL-C levels at enrollment (median 97 versus 92 mg/dL; \( P = 0.0001 \)) than patients with prior LDL-C testing. Of 166 individuals with core laboratory LDL-C levels \( \geq 190 \) mg/dL, 36.1% had no LDL-C measurement in the prior 2 years, and 57.2% were not on a statin at the time of enrollment.

Conclusions—in routine clinical practice, LDL-C testing is associated with higher-intensity lipid-lowering treatment and lower achieved LDL-C levels. (J Am Heart Assoc. 2018;7:e009251. DOI: 10.1161/JAHA.118.009251.)

Key Words: clinician lipid testing practices • guideline adherence • low-density lipoprotein cholesterol
Clinical Perspective

What Is New?

- The 2013 American College of Cardiology/American Heart Association (ACC/AHA) Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Risk in Adults transitioned from a focus on achieving an low-density lipoprotein cholesterol target to treatment based on a patient’s overall atherosclerotic disease risk.
- This study explores clinician use of blood lipid level evaluation following the release of the 2013 ACC/AHA guidelines.
- Over a third of patients did not have low-density lipoprotein cholesterol testing in the 2 years before enrollment.
- Patients without low-density lipoprotein cholesterol testing were less likely to be treated with a statin medication, a high-intensity statin, and nonstatin lipid-lowering therapy.

What Are the Clinical Implications?

- Within the context of the 2013 ACC/AHA guideline, lipid testing may help to optimize guideline-recommended statin use and dosing.

ACC/AHA cholesterol guideline. This nationwide, cross-sectional registry allowed us to examine (1) the prevalence of LDL-C testing among primary and secondary prevention patients, (2) provider and patient characteristics associated with lipid testing, (3) whether lipid testing was associated with greater adherence to ACC/AHA guideline–recommended lipid-lowering therapy use, and (4) the association between lipid testing and achievement of lower LDL-C levels. We hypothesized that lipid testing would be associated with greater adherence to ACC/AHA guideline–recommended therapy and lower LDL-C levels.

Methods

Study Population

The PALM registry enrolled 7938 adults on statins, adults at high risk of ASCVD, and adults with prior ASCVD from 140 cardiology, primary care, and endocrinology practices in the United States. Detailed design, rationale, inclusion, and exclusion criteria for the PALM registry have been published previously. The data, analytic methods, and study materials will not be made available to other researchers for purposes of reproducing the results or replicating the procedure. Enrollment was conducted between May 27, 2015, and November 12, 2015. All participants provided signed informed consent to participate. Each site obtained institutional review board approval for participation.

Chart abstractions and core laboratory lipid panels were conducted to assess current lipid management. For this analysis, participants were included if they underwent phlebotomy for core laboratory lipid measurement at enrollment in the study (n=7722). Subjects were excluded if they were missing valid lipid core laboratory data (n=91) or data on prior ASCVD history (n=4). This resulted in a final study population of 7627 primary and secondary patients enrolled at 140 practices.

Data Collection and Definitions

At study initiation and before any patient enrollment, providers at each participating site completed surveys that collected provider and practice characteristics. For each enrolled patient, sites retrospectively reviewed medical records to determine whether lipid testing was previously performed. Patients were defined as having clinician-ordered lipid testing if they had LDL-C test results documented in the medical record within the 2 years before enrollment, regardless of whether these tests were performed at the study site or elsewhere, with results available to the treating physician. Data for the most recent LDL-C measurement, as well as the highest LDL-C measurement in the past 2 years, were entered into the data collection form. Additionally, chart review collected detailed sociodemographic and medical history for each enrolled patient, as well as current statin use and dosing intensity. Enrolled patients were asked if they knew their cholesterol level. On the day of enrollment, each patient underwent phlebotomy to analyze total cholesterol, direct LDL-C, HDL-C, and triglyceride levels that were performed by a central core laboratory.

Treatment with guideline-recommended statin dosing intensity was defined by certain criteria. A high-intensity statin was recommended for patients with at least 1 of the following criteria: (1) known ASCVD and aged ≤75 years; (2) LDL-C ≥190 mg/dL; or (3) diabetes mellitus with estimated 10-year ASCVD risk ≥7.5%, aged 40–75 years, and LDL-C ≥70 mg/dL. A statin of at least moderate intensity was recommended for patients without indication for high-intensity statin and (1) aged 40 to 75 years, diabetes mellitus, and estimated 10-year ASCVD risk <7.5% with LDL-C ≥70 mg/dL or LDL-C <70 mg/dL currently on statin therapy; (2) clinical ASCVD and aged >75; or (3) no diabetes mellitus and 10-year ASCVD risk ≥7.5% with LDL-C ≥70 mg/dL or LDL-C <70 mg/dL currently on statin therapy.

Statistical Analysis

Patients were categorized based on the presence or absence of medically documented lipid testing with at least 1 LDL-C value and the highest LDL-C value in the 2 years before study.
enrollment. The percentages of patients with a documented LDL-C level were presented among all patients, primary prevention patients, and secondary prevention patients, respectively. The percentages of primary and secondary prevention patients with LDL-C documented were compared using the chi-square test.

Patient, clinician, and practice characteristics (including subspecialty, guideline adoption, and practice location), core laboratory lipid measurements, and statin usage were summarized using percentage for categorical variables and median (first quartile, third quartile) for continuous variables and compared between patients with and without a documented LDL-C using the chi-square test for all categorical variables and the Wilcoxon rank-sum test for continuous variables. The lipid measurements were also presented and compared among primary and secondary prevention patients, respectively.

We then assessed the association between documented LDL-C and statin use (yes or no) at the visit among patients with a guideline indication for statin treatment using a generalized estimating equations logistic regression model. This was implemented with a compound symmetric working correlation matrix and empirical (sandwich) standard error estimates, adjusting for clustering of observations from the same hospital. The covariates for risk adjustment consisted of age fitted with restricted cubic spline terms with 3 knots at 10%, 50%, and 90% of the empirical distribution; sex; race (black versus nonblack); body mass index with restricted cubic spline terms with three knots at 10%, 50%, and 90% of the empirical distribution; smoking status (current, quit within past year, quit >1 year ago; or never smoker); hypertension; diabetes mellitus (diabetes mellitus requiring insulin, diabetes mellitus without insulin, and no diabetes mellitus); prior ASCVD; family history of ASCVD; chronic kidney disease; and insurance status (private, government versus none). The percentage of missing values are rare, <2.5% for all variables in the multivariable model, with most of the variables for the risk adjustment missing less than 0.5%. For continuous variables such as age and body mass index, we imputed the missing values to the median value. For categorical variables, the missing values were imputed using mode values.

A P value of <0.05 was considered significant. All statistical analyses were performed using SAS version 9.4 (SAS Institute, Inc, Cary, NC).

**Results**

**Prevalence of Lipid Testing**

Among the final study population of 7627 patients, 4840 (63.5%) had a documented measurement of LDL-C in the medical record in the 2 years before enrollment. Preenrollment LDL-C testing rates were lower among primary prevention than secondary prevention patients (61.8% versus 65.6%; P<0.001). Age was not significantly different between those with and without lipid testing, but patients without LDL-C measurements were more often women, nonwhite, uninsured, and non–college graduates compared with patients who had known LDL-C results (Table 1). Tested patients were more likely to have chronic kidney disease (10.8% versus 7.1%; P<0.001) but less likely to have other cardiovascular risk factors such as diabetes mellitus (37.7% versus 40.0%; P=0.04), hypertension (76.3% versus 79.3%; P=0.002), and habitual smoking (10.6% versus 18.3%; P<0.001) than patients without lipid testing (Table 1). As expected, patients with LDL-C testing documented in the chart were more likely to report knowing their cholesterol level (77.0% versus 64.2%; P<0.001).

**Provider Characteristics Associated With Testing**

Provider characteristics were also associated with a patient’s likelihood for LDL-C testing. Patients seen by endocrinologists had the highest rate of recent lipid testing (74.4%), followed by cardiologists (67.2%), then primary care physicians (64.7%) (Figure 1; P for trend=0.008). Patients seen at practices in urban locations were more likely to undergo lipid testing than at practices in rural locations (65.5% versus 44.5%; P<0.001). Patients seen by providers who reported primarily using the 2013 ACC/AHA guidelines to guide lipid management were more likely to have had lipid testing than those seen by providers who did not (68.2% versus 60.6%; P<0.001).

**Association Between Testing and Statin Use**

Among the 5909 patients with a guideline indication for statin treatment, those with LDL-C testing in the past 2 years were more likely to be taking a statin at the time of their visit (76.0% versus 72.6%; P=0.003). This association persisted after multivariable adjustment: odds ratio, 1.23; 95% confidence interval, 1.01–1.50; P=0.043). Among patients without a chart-documented LDL-C measurement who were indicated for but not on statin treatment, only 91 (15.4%) were previously treated with a statin. Among patients indicated for statin therapy, patients with prior LDL-C testing were more likely to be treated with a high-intensity statin (24.3% versus 21.5%; P=0.016) or a nonstatin lipid-lowering therapy (27.3% versus 24.8%; P=0.037) (Figure 2).

A total of 2627 patients met a guideline indication for primary prevention statin therapy, and 3282 patients met an indication for secondary prevention statin therapy. Among patients indicated for primary prevention statin therapy, 955 (36.4%) were not on a statin at the time of enrollment (Table 2), and of the patients on a statin, 713 (42.6%) were prescribed a
statin at a dose lower than the guideline-recommended intensity. More than one third of patients who were either not on a statin or underdosed (38.0%) had no clinician-ordered LDL-C measures in the past 2 years. Among patients indicated for secondary prevention statin therapy, 536 (16.3%) were not on a statin at the time of enrollment (Table 3), and 43.1% of those prescribed a statin were underdosed, according to the 2013 ACC/AHA guideline recommendations. Again, more than one third of secondary prevention patients who were either not on a statin or underdosed (34.3%) had no LDL-C measures in the past 2 years.

### Association of Lipid Testing With LDL-C Levels

Core laboratory testing of patients enrolled in the PALM registry revealed 166 individuals (2.2% of the overall study population) with LDL-C levels \( \geq 190 \) mg/dL. Of these patients, 36.1% had no LDL-C measurement in the prior 2 years, and 57.2% were not on a statin at the time of enrollment.

In patients with a guideline indication for statin therapy, those with chart-documented LDL-C testing in the prior 2 years had lower core laboratory LDL-C levels at the time of enrollment than those without (median 92 versus 97 mg/dL; \( P<0.001 \)). When subset to patients on statin therapy, those with prior LDL-C testing had lower core laboratory LDL-C, non-HDL, and triglyceride levels at enrollment compared with those without lipid testing, regardless of primary prevention indication (Table 2) or secondary prevention indication (Table 3). Among adults on statins for secondary prevention, 1897 (69.1%) and 715 (26.0%) still had core laboratory LDL-C levels \( \geq 70 \) and 100 mg/dL at the

### Table 1. Patient Characteristics and Lipid Testing Before Enrollment

<table>
<thead>
<tr>
<th>Patients With a Medical Record-Documented LDL-C in Past 2 Years</th>
<th>Yes n=4840 (63.5%)</th>
<th>No n=2787 (36.5%)</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age: median (Q1, Q3)</td>
<td>68 (60, 74)</td>
<td>68 (59, 75)</td>
<td>0.73</td>
</tr>
<tr>
<td>Age ( \geq 75 ) y</td>
<td>25.0%</td>
<td>25.9%</td>
<td>0.35</td>
</tr>
<tr>
<td>Female</td>
<td>46.2%</td>
<td>49.5%</td>
<td>0.006</td>
</tr>
<tr>
<td>Nonwhite race</td>
<td>11.2%</td>
<td>21.7%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>Private insurance</td>
<td>64.4%</td>
<td>47.1%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>Uninsured</td>
<td>1.4%</td>
<td>3.8%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>College graduate or higher</td>
<td>38.4%</td>
<td>32.1%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td><strong>Medical history</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior ASCVD*</td>
<td>44.5%</td>
<td>40.5%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>Prior CAD†</td>
<td>36.8%</td>
<td>33.6%</td>
<td>0.005</td>
</tr>
<tr>
<td>Prior PAD‡</td>
<td>8.8%</td>
<td>8.1%</td>
<td>0.31</td>
</tr>
<tr>
<td>Cerebrovascular disease§</td>
<td>13.3%</td>
<td>12.5%</td>
<td>0.29</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>37.7%</td>
<td>40.0%</td>
<td>0.04</td>
</tr>
<tr>
<td>Hypertension</td>
<td>76.3%</td>
<td>79.3%</td>
<td>0.002</td>
</tr>
<tr>
<td>Current/recent smoker</td>
<td>10.6%</td>
<td>18.3%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>10.8%</td>
<td>7.1%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>Thyroid disease( ^k )</td>
<td>15.5%</td>
<td>12.6%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>BMI: median (Q1, Q3)</td>
<td>29.7 (26.1, 34.1)</td>
<td>29.8 (26.1, 34.7)</td>
<td>0.32</td>
</tr>
<tr>
<td>Family history of premature ASCVD</td>
<td>10.6%</td>
<td>7.1%</td>
<td>( &lt;0.001 )</td>
</tr>
<tr>
<td>Predicted 10 y ASCVD risk (among primary prevention patients)</td>
<td>11.7 (5.7, 20.9)</td>
<td>13.8 (7.1, 23.6)</td>
<td>( &lt;0.001 )</td>
</tr>
</tbody>
</table>

\( P \) values are based on chi-square tests for all categorical variables and Wilcoxon rank-sum tests for all continuous variables. ASCVD indicates atherosclerotic cardiovascular disease; BMI, body mass index; CAD, coronary artery disease; LDL-C, low-density lipoprotein cholesterol; PAD, peripheral artery disease; Q1, quarter 1; Q3, quarter 3.

*ASCVD: defined as patients with prior CABG, PCI, myocardial infarction, CAD, stroke, carotid artery stenosis, abdominal aortic aneurysm, PAD or non-coronary revascularization.

†CAD: defined as patients with prior CABG, PCI, history of myocardial infarction, or known CAD.

‡PAD: including PAD, noncoronary arterial revascularization, and abdominal aortic aneurysm.

§Cerebrovascular disease: including carotid artery stenosis, stroke, or transient ischemic attack.

\( ^k \)Thyroid disease: includes both hypothyroid and hyperthyroid disease.
time of enrollment, respectively. Of the patients with LDL-C \( \geq 100 \) mg/dL, 36.1% had not undergone lipid testing in the past 2 years.

**Discussion**

This analysis of the PALM registry aimed to evaluate current LDL-C testing patterns in patients treated at cardiology, endocrinology, and primary care practices across the United States. We observed that (1) more than one third of patients in community practice had no LDL-C levels measured in the past 2 years; (2) rates of LDL-C testing varied as a function of patient race, sex, and socioeconomic status, as well as by whether the patient was seen by a specialist or primary care provider; (3) among patients recommended for but either not on a statin or prescribed a statin at less than guideline-recommended intensity, more than one third had no LDL-C testing in the prior 2 years; and (4) patients with recent lipid testing were more likely to be treated with statin therapy and had lower LDL-C and non–HDL-C levels.

The 2013 ACC/AHA cholesterol guidelines moved away from achieving target LDL-C levels toward a focus on implementing the appropriate intensity of statin therapy on the basis of underlying cardiovascular disease risk. Recommendations for LDL-C testing remain in these guidelines to screen for familial hypercholesterolemia, determine response to statin therapy, and assess adherence. Prior studies demonstrated a misconception that clinicians following the 2013 ACC/AHA guidelines no longer needed to measure lipid
levels given the shift away from LDL-C treatment targets.12,13 Our study showed that many primary and secondary prevention patients were managed without recent LDL-C testing, which may be a reflection of misinterpretation of the 2013 ACC/AHA guidelines. Endocrinologists had the highest rate of LDL-C testing, followed by cardiologists and then primary care providers. Clinicians who reported primarily following the 2013 ACC/AHA cholesterol guidelines were more likely to test LDL-C levels.

More than one third of patients in our study of community practice had no evidence of recent LDL-C testing. We also observed disparities in lipid testing: Patients without lipid testing were more often women, nonwhite, uninsured, or less educated than those with testing. While all patients in our study were seen in an outpatient clinic, lower socioeconomic status, barriers to routine health maintenance care, lack of access to specialty care, and lower health literacy may explain these findings.14 Disparities in testing may also contribute to undertreatment in these sociodemographic subgroups.

With the release of the 2013 ACC/AHA guideline, a larger population of patients in the United States are now indicated for statin treatment,15–17 but prior studies show a considerable number of patients who were either untreated or underdosed.18,19 Core laboratory results for the PALM registry identified a group of adults with LDL-C ≥190 mg/dL who may have been undiagnosed and untreated because LDL-C levels had not been measured. More than one third of primary and secondary prevention patients who were indicated for statin therapy but were either untreated or treated below the guideline-recommended dose did not have LDL-C testing in the past 2 years. These groups of patients may have benefited from lipid testing to assess candidacy for statin treatment, appropriateness of statin dosing, adherence to treatment, and potential eligibility for nonstatin lipid-lowering

### Table 2. Core Laboratory Measurements Among Patients Indicated for Primary Prevention Statin Therapy

<table>
<thead>
<tr>
<th>Statin Use</th>
<th>Indicated for Primary Prevention Statin Therapy (n=2627)</th>
<th>Not on Statin (n=955)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>On Statin (n=1672)</td>
<td>Not on Statin (n=275)</td>
</tr>
<tr>
<td>Prior LDL-C Testing</td>
<td>Yes (n=1043)</td>
<td>No (n=629)</td>
</tr>
<tr>
<td>Core laboratory LDL-C ≥100 mg/dL at enrollment</td>
<td>420 (40.3%)</td>
<td>302 (48.0%)</td>
</tr>
<tr>
<td>Median LDL-C (Q1, Q3)</td>
<td>92 (73, 114)</td>
<td>98 (79, 122)</td>
</tr>
<tr>
<td>Median HDL-C (Q1, Q3)</td>
<td>51 (43, 63)</td>
<td>52 (42, 63)</td>
</tr>
<tr>
<td>Median triglycerides (Q1, Q3)</td>
<td>136 (96, 195)</td>
<td>144 (103, 212)</td>
</tr>
<tr>
<td>Median non–HDL-C (Q1, Q3)</td>
<td>112 (89, 135)</td>
<td>120 (98, 148)</td>
</tr>
</tbody>
</table>

P values are based on chi-square tests for all categorical variables and Wilcoxon rank-sum tests for all continuous variables. LDL-C indicates low-density lipoprotein cholesterol; Q1, quarter 1; Q3, quarter 3.

### Table 3. Core Laboratory Measurements Among Patients Indicated for Secondary Prevention Statin Therapy

<table>
<thead>
<tr>
<th>Statin Use</th>
<th>Patients With Prior ASCVD (n=3282)</th>
<th>Not on Statin (n=536)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>On Statin (n=2746)</td>
<td>Not on Statin (n=206)</td>
</tr>
<tr>
<td>Prior LDL-C Testing</td>
<td>Yes (n=1823)</td>
<td>No (n=923)</td>
</tr>
<tr>
<td>Core laboratory LDL-C ≥100 mg/dL at enrollment</td>
<td>457 (25.1%)</td>
<td>258 (28.0%)</td>
</tr>
<tr>
<td>Core laboratory LDL-C ≥130 mg/dL at enrollment</td>
<td>123 (6.7%)</td>
<td>93 (19.1%)</td>
</tr>
<tr>
<td>Median LDL-C (Q1, Q3)</td>
<td>80 (65, 100)</td>
<td>83 (67, 103)</td>
</tr>
<tr>
<td>Median HDL-C (Q1, Q3)</td>
<td>49 (42, 59)</td>
<td>50 (40, 61)</td>
</tr>
<tr>
<td>Median triglycerides (Q1, Q3)</td>
<td>131 (93, 184)</td>
<td>140 (101, 198)</td>
</tr>
<tr>
<td>Median non–HDL-C (Q1, Q3)</td>
<td>99 (81, 122)</td>
<td>102 (84, 128)</td>
</tr>
</tbody>
</table>

P values are based on chi-square tests for all categorical variables and Wilcoxon rank-sum tests for all continuous variables. ASCVD indicates atherosclerotic cardiovascular disease; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Q1, quarter 1; Q3, quarter 3.
therapy. Additionally, prior studies have suggested that LDL-C measurement is associated with higher provider adherence to evidence-based statin therapies.\textsuperscript{20,21}

Measuring lipids may also improve patient receptiveness and adherence to statin therapy.\textsuperscript{22} LDL-C measurements provide a more objective basis for patient-provider conversations on the need for statin therapy and patient education on ASCVD risk. As expected, patients with chart-documented LDL-C testing were more likely to report awareness of their cholesterol levels. Among primary prevention patients at high predicted risk of ASCVD, prior lipid testing was also associated with higher likelihood of concern about their own ASCVD risk. These patient insights may contribute to the larger proportion of patients treated with statin therapy that results in lower LDL-C, non–HDL-C, and triglyceride levels when LDL-C testing is performed. Additionally, clinicians can use this information to further educate on the importance of statin adherence and tolerance for minor side effects, as well as encourage improvement in nonpharmacologic therapies such as diet and exercise.

There are several limitations in our study. First, site study personnel were instructed to abstract prior lipid testing from the medical record, including laboratory results and clinic notes. Lipids may have been tested (eg, by another provider), but not have had test results recorded in the chart. Second, the frequency of lipid testing is not clearly recommended in the ACC/AHA guidelines.\textsuperscript{23} Third, because some patients may visit their physician only annually, the PALM study collected data on lipid testing performed in the 2 years before enrollment. A 2-year look-back period for lipid testing was also selected because statin decision making is unlikely to be guided by older lipid test results, and because many physician practices have transitioned to electronic health records, older data may be less accessible. Fourth, the rationale for physician-ordered lipid testing, as well as other details related to prior physician-ordered lipid testing, such as fasting versus nonfasting, direct versus calculated LDL-C measurement, and statin therapy use/dosing at the time of lipid testing, were not collected. Finally, because this was a cross-sectional study, we could not examine the impact of the 2013 ACC/AHA guidelines on testing trends, and given the limited number of sites, further study is necessary for additional generalizability.

Conclusion

More than one third of patients with or at risk for ASCVD are treated in contemporary practice without provider knowledge of LDL-C levels within the previous 2 years. Knowledge of LDL-C test results is associated with increased guideline adherence to statin therapy and better lipid control in both primary and secondary prevention populations. Lipid testing may help to optimize adherence to guideline-recommended statin use and dosing, as well as identify patients who are nonadherent to treatment or who need more intense lipid-lowering therapy.

Acknowledgments

We thank Erin Campbell, MS, for her editorial contributions to this article. Campbell did not receive compensation for her assistance, apart from her employment at the institution where this study was conducted.

Sources of Funding

This study was supported by Sanofi Pharmaceuticals and Regeneron Pharmaceuticals.

Disclosures

Dr Lowenstern reports funding through NIH T-32 training grant #5 T32 HL069749-14. Dr Navar is supported by the NIH, NHLBI K01HL133416-01 and reports research support from Amgen, Sanofi, and Regeneron; and consulting fees from Amgen and Sanofi. Dr Robinson reports research support from AstraZeneca, Amgen, Astra-Zeneca, Eli Lilly, Esai, Glaxo-Smith Kline, Merck, Pfizer, Regeneron/Sanoﬁ, Takeda; and consulting fees from Amgen, Eli Lilly, Merck, Pfizer, and Regeneron/Sanoﬁ. Dr Goldberg reports research support from Amarin, Amgen, Astra-Zeneca, Eli Lilly, Esai, Glaxo-Smith Kline, Merck, Pfizer, Regeneron/Sanoﬁ, IONIS, Genzyme/ Isis, Regeneron, Madrigal, and Arisaph; consulting fees from Optum Rx, Regeneron/Sanoﬁ, and Esperion; and honorarium for editorial work on Merck Manual. Dr Virani reports research support from ADA/AHA/VA and honorarium from ACC as the Associate Editor for Innovations, ACC.org. Dr Lee reports employment with Sanofi. Dr Louie reports employment with Regeneron Pharmaceuticals, Inc and ownership interest in Regeneron Pharmaceuticals, Inc. Dr Peterson reports research support from Eli Lilly, Janssen, Merck; and consulting fees from AstraZeneca, Bayer, Boehringer Ingelheim, Genentech, Janssen, Merck, and Sanofi Aventis. Dr Wang reports research support from AstraZeneca, Daiichi Sankyo, Eli Lilly, Gilead, Glaxo SmithKline, Regeneron, Sanofi; and consultant/advisory/education from Bristol Myers Squibb, AstraZeneca, Eli Lilly, and Premier, Inc. The remaining authors have no disclosures to report.

References


Insights From the PALM Registry  Lowenstein et al


