Improving Lipid Outcomes for VA Patients Taking Nonformulary Statins

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Previous studies have demonstrated advantages for pharmacist-driven clinics over more traditional primary care models with regard to dyslipidemia outcomes. These researchers examined the specific impact such clinics can have on patients who require one of the more expensive antilipemic agents that have been designated as nonformulary by the VHA.

Cardiovascular disease (CVD) is a common yet devastating condition that contributes significantly to morbidity and mortality, as well as to the financial burden of the U.S. population. Since 1900, CVD has been the top cause of mortality every year except 1918. In 2004, CVD was responsible for nearly 40% of all deaths in the United States. And each day, nearly 2,400 Americans die of CVD—an average of one death every 37 seconds.[1]

A causal link has been established between elevated lipid levels and the development of coronary heart disease (CHD). In the 1970s, the Framingham study confirmed the increased risk of developing CVD in conjunction with elevated low-density lipoprotein cholesterol (LDL-C) levels.[2] Since then, several studies have confirmed the benefit of employing lipid lowering therapy to reduce the risk of CVD.[3–5] Based upon these and other studies in the accumulating medical literature, the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) cites LDL-C as the primary target for lipid lowering therapy.[6]

In terms of lowering LDL-C levels, the most widely used and most effective agents are the 3-hydroxy-3-methyl-glutaryl–coenzyme A reductase inhibitors, commonly known as statins. Within the statin class, there is some variation in potency and pharmacokinetic properties—and in price.[7] The VHA, like many other large, integrated health care institutions, uses a closed, centralized formulary to direct prescribing behavior toward selected agents that have been determined to be clinically efficacious and cost conscious for the institution. Accordingly, providers at VHA facilities are encouraged to prescribe formulary statins whenever possible and to reserve use of the nonformulary statins for cases in which the patient does not respond adequately to or cannot tolerate the formulary agents. Although many patients in the general population may achieve their lipid goals through the use of formulary statins, the characteristics of the overall veteran population (including advanced age and the prevalence of multiple comorbidities) unfortunately predispose these patients to refractory dyslipidemia that may require use of more potent, nonformulary statins.

A growing body of research has demonstrated favorable patient outcomes when lipid lowering therapy is overseen by specialized pharmacist-managed clinics rather than by nonpharmacist practitioners within the primary care setting (defined here as "usual care").[8–10] We are unaware of any published studies, however, that specifically compare the pharmacist-managed lipid clinic and usual care models in terms of attainment of lipid goals using nonformulary statins. If research were to demonstrate substantial outcome advantages with the pharmacist-managed lipid clinic model, it might be reasonable to restrict management of high-cost, nonformulary antilipemic therapies to these types of clinics.

The purpose of the current, retrospective study, therefore, was to assess lipid outcomes for patients of

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one VA medical center who were receiving monotherapy with a nonformulary statin and whose follow-up care was provided through either a pharmacist-managed lipid clinic or a usual care model. The primary efficacy lipid outcome was the percentage of patients in each group who achieved their LDL-C goals, which were determined individually according to NCEP guidelines published within the ATP III report.[6] Secondary lipid outcomes included changes in overall 10-year CHD risk; absolute changes in total cholesterol, triglyceride, LDL-C, and high-density lipoprotein cholesterol (HDL-C) levels; and percent changes in these components of the lipid profile. Additionally, we examined the time between nonformulary statin approval and achievement of the LDL-C goal (when applicable), as well as the number of follow-up visits, telephone consultations, and medication adjustments (including changes in dosages and in agents) made during this time.

METHODS

Study setting

In the fall of 2003, the Louis Stokes Cleveland VA Medical Center (LSCVAMC) in Cleveland, OH implemented a pharmacist-managed lipid clinic. This clinic, which is staffed by one full-time pharmacist, provides services to patients who receive their primary care from the community-based outpatient clinics affiliated with the LSCVAMC. Prescribers from these clinics submit electronic consults to the pharmacist-managed lipid clinic for the following: (1) approval of nonformulary antilipemic medications, (2) pharmacist consultation regarding patient-specific drug therapy decisions, or (3) patient referral for complete management of dyslipidemia. Regardless of who provides subsequent follow-up care, the lipid clinic pharmacist must approve all requests for nonformulary agents adhering to a preestablished protocol. At the time of this study, fluvastatin, lovastatin, and simvastatin were on the VHA national formulary, while atorvastatin, pravastatin, and rosuvastatin were designated nonformulary agents and required approval for use.

Participants

The LSCVAMC corporate database was used to identify patients with a pharmacist-managed lipid clinic progress note entered between September 1, 2004 and April 30, 2005. The initial progress note signified response by the pharmacist-managed lipid clinic to an electronic consult sent from a provider requesting either nonformulary medication approval, complete patient management, or both. From this list, every third patient was reviewed for possible inclusion into either the lipid clinic cohort or the usual care cohort. Over the study period, most cases in which a nonformulary antilipemic agent was requested did not involve a request for complete management of the patient's care by the pharmacist-managed lipid clinic but instead involved the provision of follow-up care through the usual care model. For this reason, the majority of consults reviewed for this study were for nonformulary agent approval only.

To be included in the study, patients had to be between 18 and 89 years of age at the time of approval of the nonformulary statin. For patients to be enrolled in the usual care cohort, the nonformulary statin must have been approved by the lipid clinic pharmacist, and any subsequent lipid management must have been performed exclusively by non–lipid clinic practitioners. Conversely, for a patient to be included in the lipid clinic cohort, the nonformulary statin must have been approved by the lipid clinic pharmacist, and all subsequent lipid management must have been performed exclusively by the pharmacist. Patients were excluded from the study if any of the following occurred:

- the baseline serum triglyceride level exceeded 500 mg/dL,
- the nonformulary statin was approved by any practitioner outside of the pharmacist-managed lipid clinic,
- lipid follow-up care was provided by another clinical pharmacist in the general medicine outpatient clinics at the LSCVAMC, or
- there were other, concomitant antilipemic agents on the patient's medication profile at the time of nonformulary statin approval (including cholestyramine, colesevelam, colestipol, gemfibrozil, fenofibrate, fish oil, ezetimibe, or niacin).

Data collection and analysis

For each patient included in the study, baseline demographic information was collected from the medical record. Initial 10-year CHD risk was calculated using the Framingham tables in the ATP III report.[6] Patients were considered to have a comorbid factor only if an International Classification of Diseases, Ninth Edition (ICD-9) code was listed in the active problem list within the patient's medical record. Diagnoses of interest included HIV/AIDS, organ transplant recipient, nephrotic syndrome, hepatic insufficiency, diabetes mellitus, coronary artery disease, and hypothyroidism.

Results of lipid panels performed at six time points were recorded, if available: (1) baseline, (2) first follow-up, (3) most current panel as of data collection, (4) the panel at which the LDL-C goal was achieved

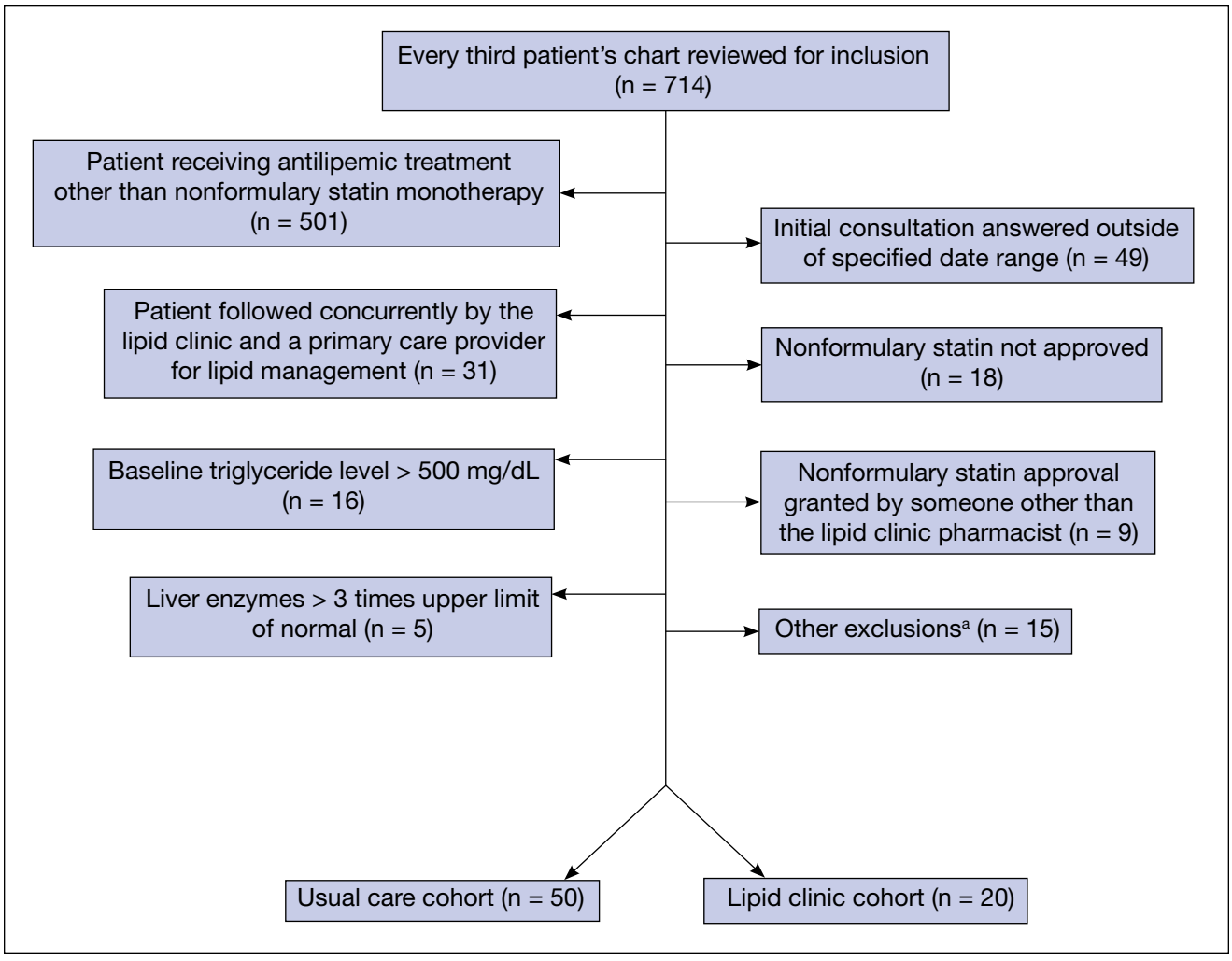

Figure. Flowchart for patient selection in the current study. [a]Other exclusions consisted of the following: patient never returned the initial telephone call made from the lipid clinic (n = 7), patient's follow-up care was managed by a non–lipid clinic provider (n = 3), patient died before the nonformulary statin was initiated (n = 1), patient self-discontinued the nonformulary agent shortly before approval (n = 2), patient's dyslipidemia was managed by a clinical pharmacist in a general medicine clinic (n = 1), patient moved to another state shortly after approval (n = 1).

(if applicable), (5) three-month follow-up panel, and (6) six-month follow-up panel (three- and six-month follow-up panels must have been obtained at 10 to 14 weeks and 22 to 26 weeks, respectively, from the baseline panel). The following additional data also were recorded: number of telephone contacts or follow-up visits conducted, time between approval of nonformulary statin medication and achievement of LDL-C goal (if applicable), and number of medication adjustments made from the time of nonformulary statin approval to achievement of LDL-C goal (if applicable). Patients were included in the efficacy analysis even if the nonformulary statin was discontinued due to adverse effects or lack of efficacy. If other agents were added to the antilipemic regimen to achieve lipid goals, this was considered a medication adjustment.

The data were entered into Microsoft Excel (Microsoft Corporation, Seattle, WA) and analyzed using Primer of Biostatistics Statistical Software Program, version 5.0 (McGraw-Hill Medical, New York, NY). Differences in all nominal data were assessed using the chi-square test. A two-tailed unpaired or paired *t* test was used to

Table 1. Demographics of study participants

Demographic variable[a]	All participants (N = 70)	Usual care cohort (n = 50)	Lipid clinic cohort (n = 20)
Gender, no. (%)			
Male	70 (100.0)	50 (100.0)	20 (100.0)
Female	0 (0.0)	0 (0.0)	0 (0.0)
Age in years, mean (SD)	65.3 (10.6)	64.7 (10.4)	66.7 (11.2)
Age group, no. (%)			
< 50 years	10 (14.3)	8 (16.0)	2 (10.0)
≥ 50 years	60 (85.7)	42 (84.0)	18 (90.0)
Ethnicity, no. (%)			
White	47 (67.1)	35 (70.0)	12 (60.0)
African American	6 (8.6)	4 (8.0)	2 (10.0)
Hispanic	0 (0.0)	0 (0.0)	0 (0.0)
Other	0 (0.0)	0 (0.0)	0 (0.0)
Not documented	17 (24.2)	11 (22.0)	6 (30.0)
Total CHD[b] risk factors, no. (%)			
0	1 (1.4)	1 (2.0)	0 (0.0)
1	10 (14.3)	8 (16.0)	2 (10.0)
2	39 (55.7)	27 (54.0)	12 (60.0)
≥ 3	20 (28.6)	14 (28.0)	6 (3.0)
Presence of specific CHD risk factors, no. (%)			
Age	68 (97.1)	49 (98.0)	19 (95.0)
History of hypertension	54 (77.1)	38 (76.0)	16 (80.0)
Active smoker	11 (15.7)	8 (16.0)	3 (15.0)
Low HDL-C[c] levels	17 (24.3)	11 (22.0)	6 (30.0)
Family history of CHD	3 (4.3)	1 (2.0)	2 (10.0)
Comorbid factors, no. (%)			
CAD[d]/CHD/stroke/MI[e]	43 (61.4)	29 (58.0)	14 (70.0)
Diabetes	24 (34.3)	20 (40.0)	4 (20.0)
Hypothyroidism	1 (1.4)	1 (2.0)	0 (0.0)
Nephrotic syndrome	3 (4.2)	2 (4.0)	1 (5.0)
None	12 (17.1)	8 (16.0)	4 (20.0)
Overall Framingham 10-year CHD risk %, mean (SD)	15.97 (7.7)	15.26 (7.7)	17.8 (7.8)
Framingham 10-year CHD risk % groups, no. (%)			
< 10%	12 (17.1)	9 (18.0)	3 (15.0)
10%–20%	14 (20.0)	9 (18.0)	5 (25.0)
> 20%	44 (62.9)	32 (64.0)	12 (60.0)

[a]Demographic variables were not significantly different between the cohorts ($P > .05$ for all comparisons). [b]CHD = coronary heart disease. [c]HDL-C = high-density lipoprotein cholesterol. [d]CAD = coronary artery disease. [e]MI = myocardial infarction.

assess differences in lipid panel results within each cohort. One-way analysis of variance (ANOVA) with Bonferroni's *t* test correction was used to assess differences in lipid panel results between the cohorts at various follow-up points. In determining statistical significance, the alpha level was set at .05.

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The current study was reviewed and approved by the LSCVAMC Institutional Review Board Committee. No funding was received for this study.

RESULTS

Patient cohorts

A total of 714 patients were included in the initial medical record review. After inclusion and exclusion criteria were applied, 50 patients were assigned to the usual care cohort, and 20 were assigned to the lipid clinic cohort (Figure). The majority of potential study participants (70%) were excluded because they had active medication orders for antilipemic agents other than or in addition to the nonformulary statin at baseline.

There were no differences between the groups with regard to the demographic variables of gender, age, ethnicity, CHD risk factors, or comorbidities (Table 1). While more than two thirds of the patients initiated therapy with rosuvastatin, there were no differences between the groups with regard to the initial nonformulary statin used (P = .482 by chi-square test) (Table 2). There were no differences in baseline lipid panel results between the groups, with the exception of a higher mean baseline LDL-C level in the lipid clinic cohort compared with the usual care cohort (151.7 mg/dL versus 127.1 mg/dL, respectively; P = .023) (Table 3). Among all 70 study participants, 61 (87%) had the most stringent LDL-C goal of less than 100 mg/dL. There was no difference between the groups regarding initial LDL-C goal (P = .378).

Table 2. Nonformulary statins used by study participants

Statin[a]	All participants, no. (%) (N = 70)	Usual care cohort, no. (%) (n = 50)	Lipid clinic cohort, no. (%) (n = 20)
Atorvastatin	19 (27)	15 (30)	4 (20)
Pravastatin	4 (6)	2 (4)	2 (10)
Rosuvastatin	47 (67)	33 (66)	14 (70)

[a]Statin use was not significantly different between the cohorts (P = .482 by chi-square test).

Primary and secondary lipid outcomes

Overall, 31 (62%) of the 50 patients in the usual care cohort achieved their LDL-C goals, compared with 18 (90%) of the 20 patients in the lipid clinic cohort (P = .043 by chi-square test) (Table 4). Although there was no significant difference in the final calculated CHD risk percent between the groups (P = .883), the lipid clinic cohort experienced a greater change in calculated CHD risk percent from baseline compared with that experienced in the usual care cohort (–3% versus –0.05%, respectively; P = .029 by unpaired t test). This difference may be attributed to a greater reduction in total cholesterol and LDL-C levels in the lipid clinic cohort.

There were no differences in lipid panel results between groups at the first follow-up, on the most current panel, at the goal panel, or at the three- or six-month follow-up panels (P > .05 for all comparisons using one-way ANOVA with Bonferroni's t test correction). Furthermore, there were no differences in triglyceride or HDL-C levels within each group

Table 3. Baseline lipid panel results and NCEP[a] LDL-C[b] goals for study participants

Parameter	All participants (N = 70)	Usual care cohort (n = 50)	Lipid clinic cohort (n = 20)	*P* value
Baseline lipid panel results in mg/dL, mean (SD)				
Total cholesterol	211.1 (44.5)	205.6 (41.1)	224.8 (50.8)	.104[c]
Triglycerides	150.6 (63.3)	151.9 (59.8)	147.4 (72.9)	.770[c]
HDL-C[d]	48.0 (13.4)	47.6 (12.3)	49.2 (16.3)	.656[c]
LDL-C	134.1 (41.1)	127.1 (37.8)	151.7 (44.8)	.023[c]
LDL-C goal, no. (%)				.378[e]
< 100 mg/dL	61 (87.1)	43 (86.0)	18 (90.0)	
< 130 mg/dL	5 (7.1)	3 (6.0)	2 (10.0)	
< 160 mg/dL	4 (5.7)	4 (8.0)	0 (0.0)	

[a]NCEP = National Cholesterol Education Program. [b]LDL-C = low-density lipoprotein cholesterol. [c]By unpaired t test. [d]HDL-C = high-density lipoprotein cholesterol. [e]By chi-square test.

Table 4. LDL-C[a] goal achievement and reduction in CHD[b] risk among study participants

Parameter	All participants (N = 70)	Usual care cohort (n = 50)	Lipid clinic cohort (n = 20)	*P* value
Achieved LDL-C goal, no. (%)	49 (70.0)	31 (62.0)	18 (90.0)	.043[c]
Final CHD risk %, mean (SD)	14.6 (7.7)	14.5 (7.7)	14.8 (7.7)	.883[d]
Change in CHD risk % from baseline, mean (SD)	–1.2 (5.1)	–0.5 (5.2)	–3.0 (4.5)	.029[d]

[a]LDL-C = low-density lipoprotein cholesterol. [b]CHD = coronary heart disease. [c]By chi-square test. [d]By unpaired *t* test.

Table 5. Comparison of total cholesterol and LDL-C[a] levels at baseline and various follow-up points within study groups

Lipid panel	Usual care cohort (n = 50)	Lipid clinic cohort (n = 20)
Total cholesterol levels in mg/dL, mean (SD)		
Baseline	205.6 (41.1)	224.8 (50.8)
First follow-up	190.0 (44.7)	193.1 (48.6)[b]
Most current	188.0 (36.0)[b]	172.4 (39.7)[b]
Goal	160.6 (19.5)[b]	161.7 (17.9)[b]
Three-month	195.8 (53.8)	176.7 (35.8)[b]
Six-month	189.0 (43.6)	182.2 (19.5)
LDL-C levels in mg/dL, mean (SD)		
Baseline	127.1 (37.8)	151.7 (44.8)
First follow-up	112.7 (39.5)	116.1 (42.7)[b]
Most current	112.0 (33.4)[b]	98.2 (37.1)[b]
Goal	86.5 (17.7)[b]	87.0 (11.9)[b]
3-month	121.7 (46.9)	100.1 (25.4)[b]
6-month	111.4 (44.3)	107.8 (21.0)[b]

[a]LDL-C = low-density lipoprotein cholesterol. [b]*P* < .05 by paired or unpaired *t* test.

from baseline to any follow-up point (*P* > .05 for all comparisons using unpaired *t* test for usual care cohort and paired *t* test for lipid clinic cohort). When comparing baseline mean total cholesterol and LDL-C levels to those recorded from the various follow-up panels for each cohort, patients in the lipid clinic cohort demonstrated significant reductions in both lipid panel components over a greater number of follow-up panels compared to patients in the usual care cohort (Table 5).

When evaluating the percent reduction in total cholesterol levels from baseline, these reductions were of greater magnitude at all points of follow-up for the lipid clinic cohort as compared to usual care cohort: 14.1% versus 7.59% at the first follow-up panel, 23.31% versus 8.56% at the most current panel, 28.07% versus 21.89% at the goal panel, 21.4% versus 4.77% at the three-month panel, and 18.95% versus 8.07% at the six-month panel. Similarly, the percent reduction in LDL-C levels from baseline were of greater magnitude for all points of follow-up for the lipid clinic cohort as compared to usual care cohort: 23.47% versus 11.33% at the first follow-up panel, 35.27% versus 11.88% at the most current panel, 42.65% versus 31.94% at the goal panel, 34.01% versus 4.25% at the three-month panel, and 28.94% versus 12.35% at the six-month panel.

Absolute reductions in total cholesterol levels calculated from baseline to follow-up panels were greater for the lipid clinic cohort as compared to usual care cohort at all points of follow-up, with the exception of the six-month panel (Table 6). Absolute changes in LDL-C levels calculated from baseline to follow-up panels also were of larger magnitude for the lipid clinic cohort as compared to the usual care cohort at all points of follow-up, with the exception of both the three- and six-month panels (Table 7).

Follow-up and medication adjustment

The number of telephone consultations or follow-up visits completed from the time of nonformulary statin approval until attainment of LDL-C goal (for applicable patients) were as follows: the lipid clinic conducted a

Table 6. Absolute change in total cholesterol levels from baseline

Follow-up lipid panel	Usual care cohort (n = 50)		Lipid clinic cohort (n = 20)		*P* value[a]
	No.	Absolute change (SD) from baseline in mg/dL	No.	Absolute change (SD) from baseline in mg/dL	
First follow-up	49	14.96 (41.3)	20	31.65 (60.2)	.19
Most current	50	17.60 (29.9)	20	52.30 (54.7)	< .001
Goal	32	7.97 (18.3)	18	60.22 (48.0)	< .001
Three-month	12	5.67 (25.5)	11	7.55 (16.7)	.84
Six-month	16	15.44 (36.7)	5	5.40 (40.6)	.61

[a]Calculated for usual care versus lipid clinic cohorts using one-way analysis of variance with Bonferroni's *t* test correction.

mean (SD) of 3.28 (2.11) telephone consultations, and usual care providers conducted a mean (SD) of 2.67 (1.3) follow-up visits. From the time of nonformulary statin approval to goal attainment, patients in the lipid clinic cohort experienced a mean (SD) of 1.28 (1.53) medication adjustments, compared with 0.7 (0.84) in the usual care cohort.

Among all 70 study participants, the mean (SD) follow-up time between nonformulary statin approval and the last lipid-related progress note was 210.39 (122.93) days. A progress note was considered to be lipid-related if it addressed lipid management in any part of the note. When looking specifically at the two cohorts, the mean (SD) total follow-up time was 167.55 (114.41) days for patients in the lipid clinic cohort versus 222.52 (123.11) days for patients in the usual care cohort.

For the 38 study patients who achieved their LDL-C goals sometime after nonformulary statin approval, the total time from nonformulary statin approval to the goal panel was a mean (SD) of 124.5 (65.48) days. Among these 38 patients, the 17 in the lipid clinic cohort had a mean (SD) time of 141.38 (108.03) days between nonformulary statin approval and the goal panel, while the 21 patients in the usual care cohort had a mean (SD) time of 112.23 (65.48) days (P = .31 by one-way ANOVA). The reason there were fewer patients included in this analysis compared to the total number of study patients who achieved their LDL-C goal is that some of these patients were at their LDL-C goal at the time of nonformulary statin approval.

DISCUSSION

The impact of pharmacist-managed clinics on patient outcomes has been widely recognized in medical literature, with many papers highlighting positive results of such patient-pharmacist interactions.[8,9,11,12] The results of the current study complement these findings: In comparison to traditional management of dyslipidemia within primary care, management through a pharmacist-managed lipid clinic significantly enhanced patients' attainment of NCEP LDL-C goals, reduced their calculated CHD risk, and produced significant reductions in both total cholesterol and LDL-C levels at several points of follow-up for patients at the LSCVAMC.

In the Lipid Treatment Assessment Project (L-TAP) study, investigators examined data on 488 patients with dyslipidemia from five regions of the United States in order to assess the percentages achieving LDL-C goals defined by NCEP guidelines.[12] They found that, overall, only 38% of patients were able to achieve their goal levels. When patients were stratified by their respective CHD risk categories, it became apparent that this success rate decreased with increasing CHD risk—and was poorest for those with established CHD.[12] By comparison, in the current study, 90% of patients whose care was managed by the lipid clinic were able to achieve their LDL-C goal, despite the fact that 70% of these patients had established CHD and 90% had the most stringent LDL-C goal of less than 100 mg/dL.

Although there was no significant difference in the time required to reach LDL-C goals between the two cohorts in our study, the percentage of patients who attained their goals was greater in the lipid clinic cohort than in the usual care cohort. A likely contributor to improved lipid goal attainment is more intensive counseling on lifestyle modification and the importance of adherence to medication schedules and regular laboratory follow-up. Several studies have cited improvement in these areas for patients whose care is managed within pharmacist-driven clinics. Bozovich and colleagues documented a 72% increase in adherence to laboratory studies and medication refills after six months of management within a pharmacist-managed lipid clinic.[9]

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Table 7. Absolute change in LDL-C[a] levels from baseline

Follow-up lipid panel	Usual care cohort (n = 50)		Lipid clinic cohort (n = 20)		*P* value[b]
	No.	Absolute change (SD) from baseline in mg/dL	No.	Absolute change (SD) from baseline in mg/dL	
First follow-up	49	14.23 (35.8)	20	35.60 (58.0)	.07
Most current	50	15.12 (27.5)	20	53.50 (50.4)	.001
Goal	32	7.91 (17.1)	18	57.22 (42.6)	< .001
Three-month	12	6.50 (21.7)	11	7.82 (19.2)	.88
Six-month	16	15.44 (35.7)	5	2.6 (37.3)	.50

[a]LDL-C = low-density lipoprotein levels. [b]Calculated for usual care versus lipid clinic cohorts using one-way analysis of variance with Bonferroni's *t* test correction.

Another study examining the effectiveness of a pharmacist-coordinated, multidisciplinary lipid clinic in achievement and maintenance of LDL-C goals found that both diet and exercise were monitored significantly more often when patients were enrolled in the lipid clinic than after they were discharged from this clinic ($P < .0001$).[13]

Within the LSCVAMC's pharmacist-managed lipid clinic, adherence and medication refill history are assessed at baseline and at follow-up consultations. Diet and exercise counseling are documented in the encounter progress note for each patient. In addition, the lipid clinic pharmacist sends the patient written educational materials concerning lifestyle modifications tailored to specific comorbidities, such as diabetes and hypertension. If a patient misses a scheduled laboratory appointment, a letter is sent to the patient reminding him or her to complete the necessary studies. Intensive follow-up of patients with complex issues by the LSCVAMC pharmacist-managed lipid clinic likely contributes to the successful management demonstrated in this evaluation. The benefit of this more intensive follow-up by the pharmacist-managed lipid clinic appears to be associated with a greater improvement in the lipid endpoints and, presumably, patients' CHD risk.

Given the demographics of the veteran population, it is reasonable to expect that many dyslipidemic veterans treated at a large VA medical center, like the LSCVAMC, would be at especially high risk for CVD due to their age and, as a result, would require aggressive management of antilipemic therapy. Furthermore, patients referred to specialty clinics typically have more complex conditions, suffer from more severe dyslipidemia, have multiple comorbidities, and may require not only the more potent nonformulary statin but additional antilipemic agents to achieve lipid goals. Patients who qualified for inclusion into the lipid clinic cohort were prescribed monotherapy with a nonformulary statin; those prescribed multiple antilipemic agents, including the nonformulary statin, were excluded. This exclusion in the current study may explain the small number of patients that qualified for inclusion into the lipid clinic cohort. Moreover, the fact that the majority of the consults reviewed were for nonformulary agent approval only may have limited further the number of patients qualifying for the lipid clinic cohort. Due to the retrospective design, small number of patients included in the study cohorts, and the insufficient power of the current study, additional research is needed to validate these findings and to provide further support for implementation of large practice changes.

IN SUMMARY

A lipid clinic managed by a clinical pharmacist was able to produce more favorable lipid outcomes for patients using high cost nonformulary medications as compared with usual care provided by nonpharmacist practitioners. We speculate that these benefits may be due to more intense follow-up that is made feasible by this model of care. In the long term, aggressive control of dyslipidemia can be expected to reduce morbidity and mortality, as well as the financial burden associated with CVD. Therefore, it may be reasonable for institutions with a closed formulary to consider that such clinics manage the care of those patients who have complicated dyslipidemia and have failed initial therapy with a formulary agent.

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Author disclosures

The authors report no actual or potential conflicts of interest with regard to this article.

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