A Treatment Guideline for the Management of Dyslipidemia at the Hope Clinic

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Executive Summary

Dyslipidemia is a major independent risk factor for the development of coronary heart disease, the current leading cause of death in the United States. Despite wide dissemination of the National Cholesterol Education Program Adult Treatment Panel III guidelines and strong evidence supporting the clinical benefits of medical treatment, there continues to be significant variation in provider approaches to screening and treating dyslipidemia in clinical practice with providers failing to screen, initiate, or intensify treatment when warranted. This recognition of a problem but failure to act, termed clinical inertia, is a major factor in the sub-optimal rate of dyslipidemia treatment.

An effective approach for overcoming clinical inertia in implementing best practice guidelines in the practice setting is to combine flow sheets with reminders and feedback on clinicians' performance. This scholarly project combined the current Adult Treatment Panel III guidelines with current research to create a simplified guideline in the form of a flip chart for use by providers at the Hope Clinic in Midvale, Utah.

The Hope Clinic is a primary care medical facility that provides free medical care to the underserved and uninsured in the Salt Lake Valley region. Providers at the clinic noted that there was wide variation in prescribing practices relating to dyslipidemia treatment, in part spurring this project. There is a range of educational backgrounds among providers, who include physicians, nurse practitioners, physician assistants, medical residents, and students of various disciplines. In order to meet the needs of the Hope Clinic, the guideline created for this project was focused on being a quick and effective resource that could be utilized by providers with different educational backgrounds.

The ultimate objective of this project was to improve patient outcomes. This outcome was indirectly measured through meeting the objectives of this project which were to: (a) create a treatment guideline in the form of a flip chart for use at the Hope clinic, (b) improve provider adherence to current evidence-based recommendations for the management of dyslipidemia, (c) increase consistency in provider treatment of dyslipidemia, and (d) increase the frequency with which providers initiate treatment of dyslipidemia in patients with non-alcoholic fatty liver disease.

Chart audits were conducted comparing treatment of dyslipidemia before and after implementation of the guideline in order to assess if these objectives were met. With use of the guideline, adherence to the ATP III guidelines improved from 40% to 78%. There was a significant decrease in the use of fish oil from 46% to 11% as providers focused on targeting LDL cholesterol over triglycerides. There was an increase in the prescription of statin medications from 35% to 46%. Reviews of provider notes revealed an increase in individualizing care based on risk category, starting at therapeutic statin dosages, and titrating up statin dosages when appropriate. Use of the guideline also improved consistency in treatment of dyslipidemia and increased the rate of dyslipidemia treatment in patients with non-alcoholic fatty liver disease. Given that these parameters are measurable markers of improved dyslipidemia management, continued use of this guideline should improve long-term patient outcomes.

The supervisory committee for this scholarly project included Dr. Dianne Fuller DNP, APRN, FNP-C and Dr. Katie Ward DNP, WHNP, ANP. The chair for this scholarly project was Pamela Phares PhD, APRN-BC, CNM. Committee members serving as content experts were Debbra Whipple, DNP, FNP-BC and David Winmill DNP, ANP-BC, CDE, BC-ADM. Special thanks to these individuals as well as to the providers and staff of the Hope Clinic where this project was implemented.

Problem Statement

Dyslipidemia is a major risk factor for the development of coronary heart disease (CHD), the leading cause of death in the United States. In 2008 it was estimated that 16.3 million Americans had CHD (Roger et al., 2012). Overall 385,000 people die from CHD annually, with an estimated financial cost of 108.9 billion dollars (Heidenreich et al., 2011; Kochanek, Xu, Murphy, Minino, & Kung, 2011).

Based on data from the NHANES report from 2005-2008, 71 million US adults over the age of 20 years had higher than normal low-density lipoprotein (LDL) cholesterol levels. Of these, 34 million (48.1%) were treated, and only 23 million (33.2%) had their LDL cholesterol controlled (CDC, 2011). Strong evidence linking dyslipidemia to CHD and evidence supporting the clinical benefits of medical therapy to treat dyslipidemia prompted the publication of the National Cholesterol Education Program Adult Treatment Panel III (ATP III) guidelines (Eaton et al., 2011).

Most patients treated for dyslipidemia in clinical trials achieve goal levels, but this has not translated into clinical practice despite the wide dissemination of evidence-based clinical guidelines among the medical community. Poor implementation of guidelines in primary care has lead to an estimated 40 million Americans with sub-optimally treated dyslipidemia (Bertoni et al., 2006; Grant et al., 2004; Keeval, Cullen, Gangnon, McBride, & Stein, 2007).

Significance

Health care providers often do not screen, initiate, or intensify treatment of dyslipidemia when warranted. This recognition of a problem but failure to act, termed clinical inertia, can be attributed to a number of barriers. Some of these include: (a) a lack of provider knowledge about the dyslipidemia treatment guidelines and effective treatment regimens, (b) inconsistent

application of guidelines among providers in the same practice, (c) poor patient adherence, (d) lack of time, (e) concerns regarding side effects of treatment, (f) the complexity of the guidelines, (g) lack of access to guidelines, (h) competing demands, and (i) a lack of reminder systems for follow up (Eaton, Galliher, McBride, Bonham, Kappus, & Hickner, 2006; Parker et al., 2008; Phillips et al., 2001).

Given that CHD is the leading cause of death in the US and the efficacy of lipid-lowering therapy for primary and secondary prevention of CHD has been demonstrated in several major randomized controlled trials, efforts to improve the consistent use of evidence-based guidelines in the treatment of dyslipidemia in primary care practice is a major priority (Bertoni et al., 2006).

Purpose

The Hope Clinic is a primary care medical facility that provides free medical care to the underserved and uninsured in the Salt Lake Valley region (utahhopeclinic.org, 2012). The number of patients seeking treatment often exceeds the capacity of the clinic. Reducing treatment times increases the number of patients that can be seen, making time management critical. There is a wide range of provider educational backgrounds including physicians, nurse practitioners, physician assistants, medical residents, and various students in training. Providers at the Hope Clinic have demonstrated significant variation in prescribing practices regarding dyslipidemia management, spurring the development of this project.

An effective approach for overcoming clinical inertia in implementing best practice guidelines in the clinical practice setting is to combine flow sheets with reminders and feedback on clinicians' performance (Phillips et al., 2001). In this scholarly project, the ATP III guidelines were combined with current research to create an evidence-based guideline that could be quickly utilized by providers with a wide range of educational backgrounds.

Objectives

The primary aim of this guideline was to improve long-term patient outcomes by promoting compliance with existing research and dyslipidemia guidelines and improve consistency of dyslipidemia treatment among clinicians at the Hope Clinic. Because evaluation of this objective cannot be accomplished during the time frame of this project, it has been broken down into the following objectives that can be measured within the timeframe of this project.

1. A dyslipidemia treatment guideline will be developed and implemented at the Hope clinic.
2. Providers will adhere to current evidence-based recommendations for the management of dyslipidemia.
3. There will be increased consistency in provider treatment of dyslipidemia.
4. Providers will initiate treatment of dyslipidemia with greater frequency in patients with non-alcoholic fatty liver disease.

Review of Literature

The presence of hypercholesterolemia is a prerequisite for atherogenesis. Researchers have shown that elevated LDL cholesterol is the leading risk factor for atherogenesis. Based on this evidence, the National Cholesterol Education Program (NCEP) panel of experts prioritized treatment of LDL cholesterol during their development of the ATP III guidelines. Cardiovascular disease risk demonstrates a log-linear relationship, continuing to increase as LDL levels increase (Grundy et al., 2004; Law, Wald, & Rudnicka, 2003; Law, Wald, & Thompson, 1994; NCEP, 2001). Data suggests that for every 30 mg/dl increase in LDL cholesterol, the relative risk for CHD increases by 30% (Grundy, 2004). Although elevated triglycerides (TG) and non-HDL cholesterol are also atherogenic, increased levels of these lipoproteins are less strongly linked to

CHD and, therefore, treatment of these cholesterol components have a secondary focus in the ATP III guidelines (NCEP, 2002).

Screening

Data from randomized controlled trials have demonstrated that earlier treatment of dyslipidemia significantly improves CHD outcomes (Law, 1999). According to the ATP III guidelines, the intensity of prescribed therapy is dictated by absolute CHD risk. The first step in assessing risk is to screen all individuals over the age of 20 by obtaining a fasting serum lipid profile consisting of total cholesterol, LDL cholesterol, HDL cholesterol, and TG. Screening fasting lipid profiles should be performed every five years, or sooner if dictated by therapy. Prior to initiation of pharmaceutical intervention, an elevated LDL or TG level should prompt further clinical or laboratory investigation to rule out causes of secondary dyslipidemia such as diabetes, hypothyroidism, obstructive liver disease, chronic renal failure, or use of progestins and steroids (NCEP, 2001).

Risk Assessment

Individual patient risk for developing CHD is determined by reviewing a patient's relevant risk factors and calculating a 10-year risk profile using risk tables developed from the Framingham Heart Study (NCEP, 2001; Wilson et al., 1998). Major risk factors for CHD include cigarette smoking, hypertension (either treated or untreated), HDL cholesterol < 40mg/dl, family history of premature CHD (first degree male relative < 55 years, female < 65 years), and age > 45 years for men and > 55 years for women. Having an HDL cholesterol level > 60mg/dl is cardio-protective and removes one patient risk factor from the risk assessment. Based on the number of major risk factors and Framingham risk score, patients are classified into one of four CHD categories: high, moderately high, moderate, and low risk (NCEP, 2001).

Patients in the high-risk category consist of individuals with CHD or CHD equivalents. Coronary heart disease equivalents include diabetes, other clinical atherosclerosis-accelerating diseases such as peripheral artery disease, abdominal aortic aneurysm, carotid artery disease, and two or more risk factors in combination with a Framingham 10-year risk of CHD > 20% (NCEP, 2001).

The moderately high-risk category consists of having two or more risk factors and a Framingham 10-year risk score of 10- 20%. People with two or more risk factors and a Framingham risk score less than 10% are considered at moderate risk. People with one or zero major risk factors are considered to be at low risk for CHD. The use of Framingham tables is only necessary when a patient presents without a known CHD equivalent, but has two or more major risk factors. At this point the tables are used to distinguish between those at highest risk (>20%), moderately high risk (10-20%), and moderate risk (0-10%). Risk categories are then used in conjunction with LDL cholesterol levels in guiding treatment (NCEP, 2004; NCEP, 2001).

Some researchers are critical of using the Framingham risk score assessment in the ATP III guidelines. They feel that the focus is too narrow and that by not taking lifetime risk into consideration, the opportunity to prevent arthrosclerosis in high lifetime risk patients is missed Martin et al., 2012). However, in a guideline criticized for being too complicated, calculating lifetime risk in addition to 10-year risk adds an additional step, and would pose an additional barrier to implementation. Experts did not intend for this guideline to replace clinical judgment; but rather to provide prioritization for pharmaceutical treatment in situations where benefit grossly outweighs costs (NCEP, 2001).

Treatment

The primary focus of treatment in the ATP III guideline is LDL cholesterol reduction (NCEP, 2002). TG and non-HDL cholesterol are secondary foci of treatment once LDL cholesterol goals are achieved. An exception to this is the presence of a TG level ≥ 500mg/dl, which requires immediate treatment to prevent acute pancreatitis. Once TG levels are below this threshold, primary focus returns to lowering LDL cholesterol (NCEP, 2001). Current ATP III recommendations for high-risk individuals are to begin treatment with therapeutic lifestyle changes (TLC) and drug therapy when LDL cholesterol level is ≥ 100mg/dl, with the goal LDL level of < 100mg/dl. Lower LDL cholesterol levels directly correlate to a lower relative risk of CHD. Data from studies subsequent to the original NCEP 2001 guidelines showed continued risk reduction when LDL levels fell well below 100mg/dl, which prompted an updated recommendation by the NCEP in 2004 that LDL cholesterol in very high risk individuals have the optional goal of ≤ 70mg/dl (Cannon et al., 2004; Heart Protection Study Collaborative Group, 2002; Grundy et al., 2004).

Individuals classified as moderately high risk for CHD have a recommended LDL cholesterol goal of < 130mg/dl. The recommendation is to begin TLC and statin drug therapy concomitantly when a patient's LDL value is above this goal level. There is an optional recommendation to initiate antihyperlipidemic therapy when the LDL cholesterol is > 100mg/dl (Grundy et al, 2004).

Individuals classified as moderate risk for CHD have a recommended LDL goal of < 130mg/dl. TLC should be initiated when LDL is at or above this goal, and antihyperlipidemic drug therapy should be started when LDL cholesterol exceeds 160mg/dl (Grundy et al, 2004).

Individuals classified as low risk for CHD have a recommended LDL goal of < 160mg/dl. TLC should be initiated when LDL cholesterol is above goal and drug therapy initiated when LDL cholesterol exceeds 190mg/dl. There is the optional recommendation of initiating antihyperlipidemic drug therapy when LDL cholesterol exceeds 160mg/dl (Grundy et al., 2004).

Therapeutic lifestyle changes. The first step of treatment with the ATP III guidelines is TLC. Therapeutic lifestyle changes are highly recommended at stipulated threshold levels according to risk, but can be employed at lower thresholds and for any patient with lifestyle related risk factors such as obesity, physical inactivity, elevated TG, low HDL cholesterol, or the metabolic syndrome, regardless of LDL cholesterol level (Grundy et al, 2004). Therapeutic lifestyle changes include the TLC diet, weight reduction, and regular physical activity of 30 minutes a day. The combination of all three TLC interventions can lead to LDL cholesterol reductions of up to 25-30% (Jenkins et al., 2003; NCEP, 2002).

The NCEP (2002) recommends prioritizing dietary changes above weight loss and exercise in TLC. The TLC diet focuses on reducing intake of saturated fats and cholesterol, increasing intake of plant stanol and sterols, and increased viscous fiber. Intake of saturated fats and cholesterol is kept to a minimum while total fat intake should be limited to 25-35% of total daily calories, allowing for intake of omega-3 fatty acids to increase HDL and reduce TG. Plant sterols and stanols are naturally occurring in many fruits, vegetables, vegetable oils, nuts, seeds, cereals and legumes. Their therapeutic effect involves blocking the absorption of cholesterol from the small intestine (Cleveland Clinic, 2009). Margarine enriched with plant stanols and sterols is the major commercial food source. The recommended daily intake of 2-3 grams per day to reduced LDL cholesterol by 6-15% correlates to two to four tablespoons (Cleveland Clinic,

2009; NCEP, 2002). Viscous fiber intake should be at least five to ten grams with a goal of around 10-25 grams per day. Most fruits and vegetables are high in viscous fiber with oats, barley, psyllium, pectin-rich fruit, and beans having the highest levels (NCEP, 2002).

Medications and laboratory values. HMG CoA reductase inhibitors (statins) are first-line drugs for the treatment of dyslipidemia. This is due to their superior performance in lowering LDL cholesterol as well as their low side effect profiles at standard doses in trials (NCEP, 2001; Grundy et al., 2004). Statins continue to be the only medications indicated for both primary and secondary prevention of cardiovascular events (Studer et al., 2005). Improvement outcomes are realized in patients with elevated LDL cholesterol and patients without dyslipidemia but with elevated high-sensitivity C-reactive protein levels (Grundy et al., 2004; Ridker et al., 2008). The effect of statins on patients' cardiovascular outcomes is not fully explained by LDL reduction, but may be related to the ability of statins to reduce the accumulation of inflammatory cells in atherosclerotic plaques, inhibition of vascular smooth muscle proliferation, improved vascular endothelial function, and inhibition of platelet function (Wierzbicki, Poston, & Ferro, 2003).

Experts recommend that an appropriate dose of statin be employed to reduce LDL cholesterol by at least 30-40% and below the target goal indicated by risk category. Doubling of a dose will generally decrease LDL cholesterol an additional 6-7% (Grundy et al., 2004). Therefore, the initial statin dose will depend on the patient's starting LDL level as well as the percentage reduction necessary to achieve the targeted goal (NCEP, 2002). Statin medications are most effective when taken at bedtime since this targets when the body produces the majority of cholesterol. Liver transaminases (or enzymes), specifically alanine aminotransferase (ALT)

should be assessed prior to the initiation of statin therapy to establish the patient's baseline values (Calderon, Cubeddu, Goldberg, & Schief, 2010).

The NCEP guidelines and some researchers recommend continued periodic evaluation of liver function tests (LFTs). Liver transaminase values exceeding 3 times the upper limit or conjugated bilirubin more than 2 times the upper limit should be re-checked and if persistent, statin therapy should be discontinued (Calderon et al., 2010; NCEP, 2002). Some researchers have observed no difference in liver enzyme fluctuations between patients receiving statin medications and placebo, and therefore recommend checking LFTs only during routine visits or if patients become jaundiced, have abdominal pain, or show other signs of liver injury. Liver enzymes often elevate following the initiation of statin therapy, changes in dosage, and in changing from one statin to another. This enzyme elevation resolves spontaneously and should not result in statin discontinuation in the absence of other clinical signs or symptoms (Bays, 2006; Cohen, Anania, & Chalasani, 2006; Law & Rudnicka, 2006; McKenney, Davidson, Jacobsen, & Guyton, 2006; Tzefos & Olin, 2011). Based on these studies and post-marketing data, the FDA updated the statin prescribing information by removing the recommendation to perform routine liver enzyme monitoring. Currently, the recommendation is to perform liver enzymes before initiating treatment and then only if clinically indicated (FDA, 2012).

It is estimated that half of patients presenting with dyslipidemia have non-alcoholic fatty liver disease (NAFLD), a co-morbidity frequently seen in type-2 diabetes or the metabolic syndrome (Athyros et al., 2010; Canon, 2004). Providers are often reluctant to initiate statin therapy in these patients due to fear of causing liver failure. Data from multiple studies have shown that statin use in patients with NAFLD is safe, does not increase the risk of liver injury, and improves LFTs as well as liver function (Athyros et al., 2010; Chalasani et al., 2012; Lewis

et al., 2007). Although all statins are effective in improving cholesterol in patients with NAFLD, more studies have been conducted with atorvastatin. Atorvastatin is currently the only statin that has been shown to reduce cardiovascular morbidity in patients with NAFLD (Chatrath, Vuppalanchi, & Chalasani, 2012).

Statin-induced myopathy is exceedingly rare, usually transient, and rarely has long-term sequelae. The increase in creatinine kinase (CK) associated with myopathy occurs very rapidly, making it impossible to detect with routine screening. Instead, CK levels should be checked when there is a patient complaint of muscle pain, weakness, numbness, tingling, or neurologic changes (Daugird & Crowell, 2003). Myopathy with CK elevations greater than 10 times the normal value should prompt discontinuation of the statin. Statin re-challenge may be considered when CK levels return to normal and symptoms resolve (Pasternak et al., 2002).

For patients with elevated LDL cholesterol who cannot tolerate statin medications or who are unable to reach goal despite maximal dosing of statin medications, treatment with bile acid sequestrants such as cholestyramine, colestipol, and colesevelam, may be used. Nicotinic acid and fibric acids such as gemfibrozil, fenofibrate, and clofibrate, minimally reduce LDL cholesterol and are therefore most useful for treating TG and non-HDL cholesterol once LDL cholesterol is within target range (NCEP, 2001).

Structured Visits: A Stepwise Approach

During the first office visit, the patient's CHD risk, dietary knowledge and practices, and level of physical activity are assessed (NCEP, 2002). Ideally, the lab results of the fasting lipid profile and LFTs are available to the clinician to help guide treatment. Medications and TLC are started if LDL cholesterol is above threshold based on individual risk factors, or if the TG level

is above 500mg/dl (Grundy et al., 2004). If indicated, the patient should be referred for nutritional counseling (NCEP, 2002).

The second visit should occur approximately six weeks after the first visit. A fasting lipid profile is obtained to determine therapeutic response. If the patient is within target LDL, then the TLC diet and/or medications should be continued and attention focused on non-HDL cholesterol and TG goals. Weight reduction and exercise should be encouraged as part of an overall healthy lifestyle and treatment of the metabolic syndrome should be pursued if present. If the targeted LDL goal has not been achieved, then the TLC diet should be reinforced and intensified with increases in viscous fiber and plant sterols and stanols, and medications titrated.

The third visit occurs six weeks after the second. A fasting lipid profile is again reviewed. If the LDL cholesterol is at or below goal, attention can be turned to treating non-HDL cholesterol and TG elevations. If LDL cholesterol goals are not achieved and medication has not been implemented prior to this visit, it should be considered at this time. A second purpose of this visit is to initiate the lifestyle therapies of weight loss and exercise for the metabolic syndrome if present. Referrals to a nutritionist and or exercise specialist should be considered. Follow up appointments should occur six weeks after initiation or titration of drug therapy until LDL goal is achieved, and then every 4-6 months thereafter if lipid profiles remain stable and below threshold range (NCEP, 2001).

Theoretical Framework

The diffusion of innovations (DOI) theory is the theoretical backbone utilized in this project. The DOI theory, created by Everett M. Rogers, has its roots in rural sociology. It attempts to explain the process by which innovations are adopted or not adopted (Edberg, 2007). Because this project took current evidence, created a new product, and had this new product

utilized in the clinical setting, DOI was used as a road map for increasing adoption and to identify potential roadblocks along the way.

As outlined by this theory, the guideline should possess several attributes in order for it to be utilized. It should have a relative advantage over the existing guideline, be compatible with existing values and practices, and be simple to use (Edberg, 2007). The guideline created for this project is in the form of a flip chart, a medium familiar to providers at the Hope Clinic. The guideline follows a stepwise approach with emphasis placed on balancing efficacy and speed of use in the clinical setting. The guideline was explained to providers in both group and individual meetings, had an initial trial period, and was continually improved, increasing adoption of this new innovation.

Implementation and Evaluation

Objective 1: A Dyslipidemia Treatment Guideline Will Be Developed and Implemented at the Hope Clinic

Provider input was elicited during construction of the guideline. A flip chart format was utilized, as this was a format familiar to providers at the clinic and facilitated adoption and use. This format also allowed for the grouping of related charts with step-by-step instructions, a format promoting quick and consistent utility among providers of different educational backgrounds. Once formalized, the guideline was presented to providers at the Hope Clinic in both group and individual sessions. Ongoing provider feedback regarding the guideline was collected in person during clinic hours over a six-week period. A questionnaire was also available as a means for eliciting anonymous provider feedback in the areas of utility of the guideline, provider satisfaction, and recommendations for improvement.

A preliminary version of the guideline was given to providers and support staff to use during the early implementation phase of the project, allowing them the ability to suggest changes to the guideline. The guideline was updated four times during the implementation phase based on provider feedback. The final guideline was then laminated, which allowed providers to circle point values on the Framingham table and then later wipe it clean.

Objective 2: Providers Will Adhere to Current Evidence-Based Recommendations for the Management of Dyslipidemia

Since the guideline created was based on current evidence-based information, proper use of the guideline should improve provider adherence to the current dyslipidemia management recommendations. To determine if this translated into clinical practice, chart reviews were conducted on patients with dyslipidemia treated before and after guideline implementation. There were 52 randomly selected charts from patients treated in the last two years for dyslipidemia before guideline implementation, and 37 charts of patients treated with the guideline. The actions of the providers were evaluated on the following outcome criteria: (a) accuracy of patient risk assessment, (b) appropriately targeting LDL cholesterol first, unless TG were over 500, (c) appropriateness of pharmacologic choices, (d) initiating appropriate beginning dosages of medication, (e) appropriate titration of dosage, and (f) adherence to appropriate follow up. Although six-week intervals are the preferred follow-up time frame, the unique circumstances of the clinic precluded strict adherence to that interval, and three months was used instead.

Chart audits revealed that prior to implementation of the guideline, 40% of patients were treated according to ATP III guidelines compared to 78% following guideline implementation. There was a significant reduction in the promotion of fish oil following guideline

implementation (46% vs. 11%) and there was an increase in the prescription of statin medications (35% vs. 46%). With use of the guideline, providers focused more frequently on targeting LDL cholesterol first unless TG were over 500, individualizing care based on risk category, starting at therapeutic statin dosages, and increasing statin dosages when appropriate. This was evident in the physician notes and the use of Framingham tables. Provider notes prior to guideline implementation were characterized by circled abnormal lab values and pharmacologic interventions targeting the largest abnormal value whether it was LDL, HDL, or TG. Statins, when used, were often started at sub-therapeutic dosages and were not titrated up.

Following implementation of the guideline, provider notes included risk categorization, corresponding LDL action level, Framingham table score if appropriate, and adherence to recommended starting dosages of statins. Overall, adherence to the current evidence-based recommendations for dyslipidemia improved after implementation of the guideline.

Objective 3: There Will be Increased Consistency in Provider Treatment of Dyslipidemia.

Formal introduction and dissemination of the guideline to users was the initial step. Nurses and support staff were recruited and taught how to use the guideline, as they are often responsible for reviewing the incoming lab work, circling abnormal values, and making recommendations to the providers. They are also involved in calling in prescriptions that are approved by providers and explaining to patients why and how to take new medications.

Chart audits were used to evaluate prescribing patterns among the providers and consistency with the guideline. Implementation of the guideline reduced the rate of not starting a clinically indicated statin medication (29% vs. 8%), and reduced the rate of missed statin dose titration (23% vs. 8%). Use of the guideline did not substantially reduce the rate at which

providers decided not to take any action in treating dyslipidemia, although intervention was clinically indicated (4% vs. 3%).

Providers were asked to complete a questionnaire to elicit the frequency of guideline use, perceived barriers to guideline use, and to query if using of the guideline led to a change in how they managed dyslipidemia. Due to poor response rates, this information was gathered by personal interviews with providers. All providers questioned said that they were using the guideline when treating dyslipidemia and that the guideline had helped them in assessing risk, targeting LDL cholesterol, and in making informed pharmacology choices.

Overall, providers' consistency of dyslipidemia treatment improved following implementation of the guideline. Much of this change was attributed to post-implementation changes in support staff behaviors when evaluating the lab values and making recommendations to the providers.

Objective 4: Providers Will Initiate Treatment of Dyslipidemia with Greater Frequency in Patients with Non-Alcoholic Fatty Liver Disease (NAFLD).

Information regarding the safety and indication of statin use in NAFLD was included in the treatment section of the flip chart and providers were educated about this recent change during the educational sessions. Chart audits were performed to determine whether more patients with NAFLD were appropriately treated for dyslipidemia after guideline implementation versus before. Six patients were identified as having NAFLD, three treated before and three treated after guideline implementation. According to the ATP III recommendations, the three patients treated before guideline implementation should have been started on statin therapy, but were not. Following implementation of the guideline, two were appropriately treated with statins and the third was not treated because that individual's LDL cholesterol was below the risk-associated

action level. This patient was instead appropriately treated with fenofibrate for elevated TG. Although the patient sample size was small, the frequency with which dyslipidemia was treated in patients with NAFLD increased after guideline implementation.

Recommendations

Although created with the Hope Clinic in mind, the flip chart has direct application to treatment of dyslipidemia in other clinics as well. This or similar guidelines may be an effective means of improving treatment of dyslipidemia generally, but especially in multi-provider settings like the Hope Clinic. This guideline will continue to be used by providers at the Hope Clinic and will be updated by the author when the ATP IV guideline and new research becomes available. The process of taking existing evidence and creating a flip chart for clinical use can be utilized to overcome clinical inertia in the treatment of many other diseases such as hypertension, diabetes, and asthma, and remains a promising area for future scholars.

Use of the guideline improved adherence to the current evidence-based treatment of dyslipidemia at the Hope Clinic. Given that studies have shown a direct correlation with improved dyslipidemia treatment and improved long-term patient outcomes, continued use of the guideline should lead to improved long-term outcomes for patients at the Hope Clinic (Bertoni et al., 2006; Eaton, 2011; Grundy et al., 2004). Use of chart audits to document long-term patient outcomes of those treated with this guideline remains an area for future researchers.

Conclusion

This project combined current research with the most recent evidence-based guidelines to create a user-friendly, quick reference to improve the clinical practice of treating dyslipidemia by providers at the Hope Clinic. Chart audits revealed that with use of the guideline, adherence to the ATP III guidelines improved from 40% to 78%. There was a significant decrease in the use

of fish oil from 46% to 11% as providers focused on targeting LDL cholesterol over TG, and there was an increase in the prescription rate of statin medications from 35% to 46%. Reviews of provider notes revealed an increase in individualizing care based on risk category, starting at therapeutic statin dosages, and titrating up statin dosages when appropriate. Use of the guideline also improved consistency in the treatment of dyslipidemia and increased the rate of dyslipidemia treatment in patients with NAFLD. Given that these parameters are measurable markers of improved dyslipidemia management, and that there is a direct correlation between appropriate dyslipidemia management and improved long-term patient outcomes, continued use of this guideline should lead to improved long-term patient outcomes (Bertoni et al., 2006; Eaton, 2011; Gundy et al., 2004).

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Appendix A: Guideline

Risk Assessment

Total Cholesterol	
• <200	Desirable
• 200-239	Borderline High
• ≥ 240	High
LDL Cholesterol	
• <70	Optimal
• 70-100	Near optimal
• 100-129	Above optimal
• 130-159	Borderline high
• 160-189	High
• ≥ 190	Very High
HDL Cholesterol	
• <40	Low (bad)
• ≥60	High (good)
Triglycerides	
• <150	Normal
• 150-199	Borderline High
• 200-499	High
• ≥500	Very High

Major Risk Factors
Cigarette smoking
Hypertension (BP >140/90) or on antihypertensive therapy
Low HDL Cholesterol (<40mg/dl)
Family history of premature CHD in first degree relative: Male <55, female <65
Age: Men>45; Women >55
HDL cholesterol>60 removes one risk factor

Step 1: Obtain fasting lipid panel at age 20
- If normal, repeat again in 5 years

Step 2: If lipids are elevated, consider other causes, repeat labs when treated:
- Hypothyroidism
- Uncontrolled diabetes
- Liver or renal impairment.
- Use of progestins, corticosteroids, anabolic steroids.

Step 3: Treat elevated triglycerides if ≥ 500
- See information box last page.

Step 4: Identify presence of CHD risk equivalents. If present, patient is in the **High Risk category**:
- Clinical CHD
- Symptomatic carotid artery disease
- Peripheral artery disease
- Abdominal aortic aneurysm
- Diabetes

Step 5: Determine number of risk factors:
- If 0-1: patient is in low risk category
- If 2+ without CHD or CHD risk equivalent, assess 10-year risk with Framingham tables.

Step 6: Evaluate lipoprotein profile in relation to risk category in table below. LDL cholesterol is targeted first.

Risk Category	LDL-C Goal	Initiate TLC	Initiate Drug Therapy
High Risk: CHD, CHD Risk Equivalent 10-year risk> 20%	Less than 100 (consider <70)	LDL ≥ 100	LDL≥ 100 Consider if 70-99
Moderately High Risk: 2+ risk factors 10-year risk 10-20%	Less than 130	LDL ≥130	LDL ≥ 130 Consider if 100-129
Moderate Risk: 2+ risk factors 10-year risk <10%	Less than 130	LDL ≥ 130	LDL ≥160
Lower Risk: 0-1 risk factors	Less than 160	LDL ≥ 160	LDL ≥ 190 Consider if 160-190

Estimate of 10-Year Risk for Men

(Framingham Point Scores)

Age	Points
20-34	-9
35-39	-4
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	11
70-74	12
75-79	13

Total Cholesterol	Points				
	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
<160	0	0	0	0	0
160-199	4	3	2	1	0
200-239	7	5	3	1	0
240-279	9	6	4	2	1
≥280	11	8	5	3	1

	Points				
	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
Nonsmoker	0	0	0	0	0
Smoker	8	5	3	1	1

HDL (mg/dL)	Points
≥60	-1
50-59	0
40-49	1
<40	2

Systolic BP (mmHg)	If Untreated	If Treated
<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥160	2	3

Point Total	10-Year Risk %
<0	< 1
0	1
1	1
2	1
3	1
4	1
5	2
6	2
7	3
8	4
9	5
10	6
11	8
12	10
13	12
14	16
15	20
16	25
≥17	≥ 30

10-Year risk _____%

Estimate of 10-Year Risk for Women

(Framingham Point Scores)

Age	Points
20-34	-7
35-39	-3
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	12
70-74	14
75-79	16

Total Cholesterol	Points				
	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
<160	0	0	0	0	0
160-199	4	3	2	1	1
200-239	8	6	4	2	1
240-279	11	8	5	3	2
≥280	13	10	7	4	2

	Points				
	Age 20-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
Nonsmoker	0	0	0	0	0
Smoker	9	7	4	2	1

HDL (mg/dL)	Points
≥60	-1
50-59	0
40-49	1
<40	2

Systolic BP (mmHg)	If Untreated	If Treated
<120	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥160	4	6

Point Total	10-Year Risk %
< 9	< 1
9	1
10	1
11	1
12	1
13	2
14	2
15	3
16	4
17	5
18	6
19	8
20	11
21	14
22	17
23	22
24	27
≥25	≥ 30

10-Year risk _____%

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
National Institutes of Health
National Heart, Lung, and Blood Institute

NIH Publication No. 01-3305
May 2001

Treatment

Drug	Starting dose	Max Dose	Expected Reduction	Tab Strengths	Cost for 30 tabs At Costco
Simvastatin "Zocor"	20- 40mg	40mg	35-41%	20, 40	$6
Pravastatin "Pravachol"	40mg	80mg	34%	10,20,40, 80	$6 $25
Lovastatin "Mevacor"	40mg	80mg	31%	10, 20 40	$6 $10
Atorvastatin "Lipitor"	10 mg	80mg	39%	10, 20 40, 80	$15, $16 $20, $22
Rosuvastatin "Crestor"	5-10mg	40mg	39-45%	5,10 20,40	$175

Note: Each doubling of the Statin dose increases LDL reduction by 6%

Statin Dosing Considerations:

- CPK and liver enzymes should be evaluated prior to starting medications. Do not start if CPK is elevated or AST, ALT > 3 times upper limit of normal.
- Statins are contraindicated with chronic or active liver disease, with the exception of non-alcoholic fatty liver disease (NAFLD) in which statins will actually improve liver function, and are now considered safe.
- Atorvastatin & Rosuvastatin have highest LDL reduction potential, higher financial cost.

Step 7: Start TLC interventions and medications at indicated LDL level. **Statins are the first-line medications** (see dosing above). Statins are most effective when taken in the evening.

TLC Interventions: all together can reduce LDL by up to 30%

- TLC diet:
  - Saturated fat <7%, cholesterol <200mg/day, total fat 25-35 % of total calories
  - Viscous (soluble) fiber 20-30g/day. Increasing amount slowly decreases bloating.
  - Carbohydrate 50-60% of calories, Protein 15%
  - Add plant stanols and sterols 2g/day (present in special margarine preparations
- Weight management
- Daily cardiovascular exercise of 30 minutes a day.

Step 8: Recheck labs 6 weeks after initiating TLC, Statin medications, or dose changes.

- If LDL still above goal, titrate up statin dose.
- If statin at maximum dose and LDL above goal, consider additional medications.
- If LDL is at goal level, target non-HDL cholesterol and triglycerides >200(see next page).

Step 9: Continued monitoring of treatment:

- Once lipids are stable and therapeutic, continue to check lipids once a year.
- There is no benefit to routinely monitoring liver enzymes or CPK values.
  - Stop medication and check CPK if muscle weakness, pain, neuralgias, neuropathy
  - Stop mediation and check AST, ALT if S/S of liver problems.
  - May restart statin once labs back to baseline and no symptoms.
    - Consider starting at lower dose or different statin (namely pravastatin)

Drug Class	Agents and daily dose	Lipoprotein Effects	Side Effects	Contraindications
Statins	Atorvastatin (10-80) Simvastatin (20-40) Pravastatin (40-80) Lovastatin (40-80) Rosuvastatin (5-40)	LDL ↓ 18-55% HDL ↑ 5-15% TG ↓ 7-30%	• Myopathy • Increased liver enzymes	Absolute: • Active or chronic liver disease other than NAFLD Relative: • Drug-drug interactions
Bile Acid Sequestrants	Cholestyramine (4-16g) Colestipol (2-20g) Colesevelam (3750mg)	LDL ↓ 15-30% HDL ↑ 3-5% TG No change	• GI distress • Constipation • Decreased absorption of other drugs	Absolute: • Dysbeta-lipoproteinemia • TG>400 Relative: • TG>200
Fibric Acids	Gemfibrozil (600mg bid) Fenofibrate (48-160) Clofibrate (1000mg bid)	LDL ↓ 5-20% (May increase with↑ TG) HDL ↑ 10-20% TG ↓ 20-50%	• Dyspepsia • Gallstones • Myopathy	Absolute: • Severe renal disease • Severe hepatic disease
Nicotinic Acid	• Immediate Release (crystalline) (1.5-3g) • Extended Release (Niaspan) (1-2g) • Sustained Release (1-2g)	LDL ↓ 5-25% HDL ↑ 15-35% TG ↓ 20-50%	• Flushing • Hyperglycemia • Gout • Upper GI distress • Hepatotoxicity	Absolute: • Chronic liver disease • Severe gout Relative: • Diabetes • Gout • Peptic ulcer disease
Omega 3 fatty acids (fish oil)	OTC: 2-4 grams a day	LDL ↑ 44% (Note increase) HDL ↑ 9% TG ↓ 45%	• Belching • Dyspepsia • Taste aversion	Absolute: • Fish or shellfish allergy • Liver disease • Pregnancy/nursing
Ezetemibe	10mg po daily	LDL ↓ 18% HDL ↑ 1% TG ↓ 8%	• Headache • Myalgia • diarrhea	Absolute: • Liver disease • Pregnancy/nursing

Risk Category	LDL Goal	Non-HDL
CHD and CHD equivalent 10-year risk >20%	< 100	< 130
Multiple (2+) risk factors 10-year risk ≤ 20%	< 130	< 160
0-1 risk factor	< 160	< 190

Treatment of elevated triglycerides

- If over 500, start very low fat diet <15% of calories, weight reduction, physical activity, nicotinic acid or fibric acid.
- If 200-500 consider TLC interventions, increasing statin dose, or starting fibrate or nicotinic acid.
- Omega-3 fatty acids dramatically decrease triglycerides, but increase LDL. Consider combo with statin if used.

Guideline Source: National Heart, Lung, and Blood Institute; National Instutites of Health; U.S. Department of Health and Human Services.

Considerations:

- Atorvastatin, lovastatin, simvastatin use CYP 3A4 pathway and are prone to more drug interactions.
- Pravastatin is not metabolized via a CYP pathway, fewer interactions.
- Rosuvastatin (10%) by CYP 2C9
- Pravastatin exhibits less distribution into non-hepatic cells, thereby less risk of myopathies.
- Patients should avoid alcohol in excess and grapefruit juice while taking statin medications.
- Gemfibrozil increases statin blood levels, consider using fenofibrate if already on a statin medication.

References

Unless otherwise specified, this guideline is based on:

Grundy, S.M., Cleeman, J.I., Merz, C.N., Brewer, H.B. Jr., Clark, L.T., Hunninghake, D.B.,…Stone, N.J. (2004). Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. *Circulation* 110, 227-239.

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Information regarding TLC interventions (Step 7) based on above 3 articles as well as:

Jenkins, D.J., Kendall, C.W., Marchie, A., Faulkner, D.A., Wong, J.M. de Souza, R.,…Connelly, P.W. (2003). Effects of a dietary portfolio of cholesterol-lowering foods vs lovastatin on serum lipids and C-reactive protein. *Journal of the American Medical Association* 290, 502-510.

Information regarding NAFLD and Step 9: Continued monitoring of treatment:

Chalasani, N., Younossi, A., Lavine, J.E., Diehl, A.M., Brunt, E.M., Cusi, K.,… Sanyal, A.J. (2012). The diagnosis and management of non-alcoholic fatty liver disease: Practice guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. *American Journal of Gastroenterology* 107, 811-826.

Chatrath, H., Vuppalanchi, R., & Chalasani, N. (2012). Dyslipidemia in patients with nonalcoholic fatty liver disease. *Seminal Liver Disease* 32, 22-29.

Cohen, D.E., Anania, F.A., & Chalasani, N. (2006). An assessment of statin safety by hepatologists. *American Journal of Cardiology* 87, 77C-81C.

McKenney, J.M., Davidson, M.H., Jacobson, T.A., & Guyton, J.R. (2006). Final conclusions and recommendations of the National Lipid Association Statin Safety Assessment Task Force. *American Journal of Cardiology* 97, supplement 89C-94C.

Considerations box based on information from:

Jacobsen, T.A. (2009). Myopathy with statin-fibrate combination therapy: clinical considerations. *Nature Reviews Endocrinology* 5, 507-518.

Neuvonen, P.J., Niemi, M., & Backman, J.T. (2006). Drug interactions with lipid-lowering drugs: mechanisms and clinical relevance. *Clinical Pharmacology & Therapeutics 80*(6), 565-81.

Appendix B: Questionnaire

Dyslipidemia Project Provider Survey

1. Are you familiar with the dyslipidemia algorithm? **Y** **N**

2. Have you used the algorithm in providing patient care? **Y** **N**

3. In patients with dyslipidemia, what percentage of patients are you using the algorithm with to aid in clinical decision-making?

None **0-25%** **25-50%** **50-75%** **75-100%** **ALL**

What are the major barriers to using the algorithm?

Has implementation of the algorithm changed your clinical management of dyslipidemia? **Y** **N**

If you answered yes, please elaborate on how your practice has changed.
If you answered no, please elaborate on why your practice has not changed.

Is there information that is included in the algorithm that is not useful?

Is there information that is missing that should be included?

Please provide any additional suggestions for improving the algorithm.

Appendix C: NCEP Approval to Use Information

Dear Mr. Heckel:

Thank you for your inquiry to the National Heart, Lung, and Blood Institute (NHLBI) Health Information Center about NHLBI's copyright policy.

Unless specified otherwise, the text of and information contained in materials published by the NHLBI are in the public domain. No further permission is required to reproduce or reprint the text in whole or in part. This applies to print publications, graphics, and animations in the NHLBI's Health Topics index as well as documents and content from the NHLBI website. The NHLBI asks only that no changes be made to the content of the materials, and that the material as well any NHLBI Internet links not be used in any direct or indirect product endorsement or advertising. Organizations may add their own logo or name.

Please use the following language to cite the source of the materials: Source: National Heart, Lung, and Blood Institute; National Institutes of Health; U.S. Department of Health and Human Services.

Your assistance in making our research and health-related information available to the largest number of people possible is greatly appreciated.

Feel free to contact us again if you have more questions.

Sincerely,

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Appendix D: IRB Waiver

IRB: IRB_00061544

PI: Geoff Heckel

Title: An Algorithm For Improving Dyslipidemia Treatment at the Hope Clinic

Thank you for submitting your request for approval of this project. The IRB has administratively reviewed your application and has determined on 2/2/2013 that your project does NOT meet the definitions of Human Subjects Research according to Federal regulations. Therefore, IRB oversight is not required or necessary for your project.

This project does not meet the DHHS definition of Human Subjects Research because it is not a systematic investigation designed to develop or contribute to generalizable knowledge. The project is designed as a Quality Improvement project to measure the effectiveness of an algorithm designed to aid providers in following published guidelines when treating elevated cholesterol and lipids at the Hope Clinic.

The project does not meet the FDA definition of Human Subjects Research because it does not involve a drug, device, or any other article regulated by the FDA.

This determination of non-human subjects research only applies to the project as submitted to the IRB. Since this determination is not an approval, it does not expire or need renewal. Remember that all research involving human subjects must be approved or exempted by the IRB before the research is conducted.

If you have questions about this, please contact our office at 581-3655 and we will be happy to assist you. Thank you again for submitting your proposal.

Appendix E: Poster

A Treatment Guideline for the Management of Dyslipidemia At the Hope Clinic

UNIVERSITY OF UTAH COLLEGE OF NURSING

Geoff Heckel DNP Candidate
Family Nurse Practitioner Program

PURPOSE

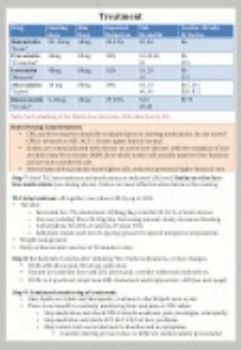
The aim of this scholarly project was to improve clinician treatment of dyslipidemia at the Hope clinic in Midvale, Utah. A flip chart guideline was created based on the Adult Treatment Panel III guidelines and updated with current research. The flip chart was created specifically for the Hope clinic, focusing treatment on the demographic they serve. Because measurement of patient outcomes was beyond the timeframe available, it was broken down into the following measurable objectives:

1. A dyslipidemia treatment guideline will be developed and implemented at the Hope clinic.
2. Providers will adhere to current evidence-based recommendations for the management of dyslipidemia.
3. There will be increased consistency in provider treatment of dyslipidemia.
4. Providers will initiate treatment of dyslipidemia with greater frequency in patients with non-alcoholic fatty liver disease.

BACKGROUND

Dyslipidemia is a major independent risk factor for the development of coronary heart disease, the current leading cause of death in the United States. Despite wide dissemination of the National Cholesterol Education Program Adult Treatment Panel III guidelines and strong evidence supporting the clinical benefits of medical treatment, there continues to be significant variation in provider approaches to screening and treating dyslipidemia in clinical practice with providers failing to screen, initiate, or intensify treatment when warranted. This recognition of a problem but failure to act, termed clinical inertia, is a major factor in the sub-optimal rate of dyslipidemia treatment. An effective approach for overcoming clinical inertia used in this project is to combine flow sheets with cues for action and provide feedback on clinicians' performance.

The Hope Clinic is a primary care medical facility that provides free medical care to the underserved and uninsured in the Salt Lake valley. The clinic is run by volunteer providers and is focused on providing outstanding cost-effective care to as many patients as time and resources allow. Provider backgrounds are varied and include physicians, nurse practitioners, physician assistants, medical residents, and students of multiple disciplines. In order to meet the needs of the Hope Clinic, the guideline created for this project was focused on being a quick and effective resource that could be utilized by providers with different educational backgrounds.



METHODS

- The guideline was developed based on the Adult Treatment Panel III guidelines and updated with information from current research.
- The guideline was presented to providers in both group and individual settings.
- Feedback was elicited from providers weekly and a feedback questionnaire provided as an anonymous means of communicating feedback.
- Changes were made to the guideline based on provider feedback.
- Chart audits were performed on randomly selected charts on patients with dyslipidemia treated before (N=52) and after (N=37) flip-chart start date to evaluate if changes in practice occurred.

CONCLUSIONS

Use of the guideline led to increased adherence to current evidence-based recommendations for the management of dyslipidemia, increased consistency in provider treatment of dyslipidemia, and increased frequency of dyslipidemia treatment in patients with non-alcoholic fatty liver disease. There was also an increased focus on assessing risk categorization, treating LDL cholesterol before other lipid elevations, and using statins over other pharmaceutical interventions.