Dyslipidemia Care Guide

May 2025

CALIFORNIA CORRECTIONAL
HEALTH CARE SERVICES

Information contained in the Care Guide is not a substitute for a health care professional's clinical judgment. Evaluation and treatment should be tailored to the individual patient and the clinical circumstances. Furthermore, using this information will not guarantee a specific outcome for each patient. Refer to "Disclaimer Regarding Care Guides" for further clarification.

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Table of Contents

GOALS

- ✓ **Identify and treat patients on the basis of primary vs. secondary prevention for atherosclerotic cardiovascular disease (ASCVD)**
- ✓ **Counsel all patients on healthy lifestyle choices**
- ✓ **Prescribe high-intensity statin therapy for ALL ASCVD patients**
- ✓ **Decrease morbidity and mortality related to ASCVD**

ALERTS

- **Statin related adverse effects and potential drug-drug interactions (DDI)**
- **Evaluate patient for familial hypercholesterolemia (FH) if LDL-C ≥ 190 mg/dL**
- **Do not start dialysis if patient is on statin**

EVALUATION OVERVIEW

Atherosclerotic cardiovascular disease (ASCVD) remains the leading cause of morbidity and mortality globally and in the United States. This is attributable to suboptimal implementation of prevention strategies and uncontrolled ASCVD risk factors. It is important to distinguish primary prevention from secondary prevention when considering therapy for cholesterol.

Clinical ASCVD is defined as the following conditions that result from atherosclerosis:

- Ischemic heart disease
  - Acute coronary syndrome (ACS)
  - History of myocardial infarction
  - Stable or unstable angina
  - Significant coronary artery stenosis
  - Prior coronary or other arterial revascularization
- Cerebrovascular disease
  - Stroke/transient ischemic attack (TIA)
  - Significant intracranial or extracranial artery stenosis
  - Symptomatic vertebral artery stenosis
- Peripheral artery disease (PAD)
  - Symptomatic, such as claudication
  - Asymptomatic with abnormal resting Ankle-Brachial Index (ABI) ≤ 0.90
- Aortic aneurysm
  - Thoracic aortic aneurysm
  - Abdominal aortic aneurysm
  - Atheroma on imaging

Primary prevention refers to the effort to prevent or delay the onset of clinical ASCVD (see page 8). One of the facets of prevention is identifying dyslipidemia and controlling cholesterol. **Secondary prevention** refers to the effort to treat clinical ASCVD and to reduce the risk of future clinical events and death (see page 9).

The diagnosis of dyslipidemia is made by measuring levels of total cholesterol, low-density lipoprotein cholesterol

(LDL-C), high density lipoprotein cholesterol (HDL-C), and triglycerides (TG). Causes of dyslipidemia may be genetic (See Appendix A), lifestyle factors, or medical conditions that interfere with blood lipid levels. Evaluation includes:

- **History**: Assess each patient for personal ASCVD risk factors and family history of ASCVD. Assess for clinical ASCVD, such as prior acute coronary syndrome (ACS), stable angina from coronary artery disease, history of coronary revascularization, stroke, transient ischemic attack (TIA), or symptomatic peripheral artery disease (PAD) of atherosclerotic origin. See page 11 for details.
- **Physical exam**: Height, weight, body mass index (BMI), waist circumference, blood pressure, cardiac evaluation, peripheral and carotid pulses, vascular bruits, tendon xanthomas, and xanthelasmas.
- **Labs**: Non-fasting lipid panel (LP) is acceptable for initial screening, comprehensive metabolic panel (CMP) including uric acid, thyroid-stimulating hormone (TSH), and hemoglobin A1c (A1c), if DM status is unknown. See page 12 for additional labs.
- **Diagnostic tests:** For patients in whom the decision to start a statin for primary prevention is uncertain, Coronary Artery Calcium (CAC) Score can be determined by placing the following order in EHRS:
  - Request for Radiology CT, Routine Priority, CT CHEST W/O CTRST, Reason For Exam CAC score.

Evaluation Overview Cont'd

- **Documentation**: Patients with clinical ASCVD and patients with severe hypercholesterolemia are already at very high-risk or high-risk for future ASCVD events, so calculating 10-year risk of ASCVD by using the pooled cohort equations (PCE) is not needed.

  For primary prevention, assess and document traditional cardiovascular risk factors and calculate 10-year risk of ASCVD by using the race- and sex-specific PCE, which is based on sex, age, race, total cholesterol, HDL-C, blood pressure, and history of diabetes mellitus (DM), and smoking history using EBM Calc ASCVD Tool, modeled after the American College of Cardiology (ACC) ASCVD risk calculator (Risk Estimator Plus) and determine appropriate statin benefit group. Additionally, document any risk-enhancing factors as a supplement to PCE for ASCVD risk for further risk stratification and guidance of statin therapy use. Keep in mind that the PCE are best validated among non-Hispanic Whites and non-Hispanic Blacks living in the US. In other racial and ethnic groups or in some non-US populations, the PCE may overestimate or underestimate risk. Therefore, providers may consider the use of another risk prediction tool as an alternative to the PCE if the tool was validated in a population with characteristics similar to those of the evaluated patient. Examples include the general Framingham CVD risk score, the Reynolds risk scores, SCORE (Systemic Coronary Risk Evaluation), and the QRISK/JBS3 tools.

  After age 20, it is reasonable to measure traditional risk factors at least every 4-6 years. For adults aged 20-39, limited data exist on the performance and utility of 10-year risk estimation tools.

- **Patient education**: Explain the relationship of dyslipidemia to ASCVD and the importance of addressing ASCVD risk factors. Use patient education pages PE1-PE3 for guidance.

TREATMENT OVERVIEW

For all patients with clinical ASCVD or increased risk of ASCVD, therapeutic lifestyle changes is recommended as the foundation of treatment. A comprehensive patient-centered approach that addresses all aspects of a patient's lifestyle habits and risk of future ASCVD events includes following a low-fat diet, increased exercise, weight loss, adequate sleep, smoking cessation, optimizing diabetes management, and optimizing hypertension management. Lifestyle goals should be emphasized on a regular basis.

Pharmaceutical therapy is managed according to a secondary prevention strategy for patients with clinical ASCVD or a primary prevention strategy based on the patient's calculated 10-year risk of ASCVD per table. For patients with clinical ASCVD and patients with severe hypercholesterolemia, specialty referral is advised. Specialty referral can be to cardiology, endocrinology, other lipid specialist, and/or registered dietician nutritionist.

Treatment Overview Cont'd

STATIN BENEFIT GROUPS AND TREATMENT RECOMMENDATIONS			
PREVENTION STRATEGY	**GROUP**	**RISK LEVEL**	**RECOMMENDATIONS**
SECONDARY PREVENTION AGE 18+	1	**Very high risk ASCVD** See algorithm page 9	Maximally tolerated statin is recommended: • If LDL-C ≥ 70 mg/dL, consider adding ezetimibe • If persistent LDL-C ≥ 70 mg/dL or non-HDL–C ≥ 100, consider adding PCSK9 monoclonal antibody (PCSK9 mAb). Specialty referral advised. *If LDL–C ≥ 55 mg/dL and/or LDL-C ≥ 50% reduction not achieved, consider adding ezetimibe or PCSK9 mAb.**
		Stable ASCVD See algorithm page 9	High- or moderate-intensity statin is recommended: • If high-intensity statin, goal LDL-C lowering ≥ 50% • If moderate-intensity statin, goal LDL-C lowering 30-49% • Increase statin intensity and/or add ezetimibe with inadequate LDL-C reduction. Specialty referral advised.
		Diabetes mellitus with ASCVD	Maximally tolerated statin is recommended: *If LDL–C ≥ 55 mg/dL and/or LDL-C ≥ 50% reduction not achieved, consider adding ezetimibe or PCSK9 mAb. Specialty referral advised.*†
PRIMARY PREVENTION ***Assess ASCVD risk in each age group. Emphasize adherence to heart healthy lifestyle.***	2	**Severe hypercholesterolemia** Requires no ASCVD risk assessment	LDL-C ≥ 190 mg/dL, maximally tolerated statin. Specialty referral advised. • If LDL-C ≥ 50% reduction not achieved, consider adding ezetimibe • If LDL-C ≥ 50% reduction still not achieved, consider adding PCSK9 mAb If LDL-C ≥ 50% reduction still not achieved and fasting TG's ≥ 300 mg/dL, consider bile acid sequestrant Heterozygous FH aged 30-75 years, maximally tolerated statin. Specialty referral advised. • If LDL-C ≥ 100 mg/dL, consider adding ezetimibe • If persistent LDL–C ≥ 100 mg/dL, consider adding PCSK9 mAb
	3	**Diabetes mellitus without clinical ASCVD** Age 20-39	• For patients with diabetes aged 20–39 years with additional ASCVD risk factors, it may be reasonable to initiate statin therapy in addition to lifestyle therapy
		Diabetes mellitus without clinical ASCVD Age 40-75	• Irrespective of estimated 10-year ASCVD risk percentage, use at least moderate-intensity statin therapy • For patients with higher cardiovascular risk, including those with one or more ASCVD risk factors, use high-intensity statin therapy to reduce LDL cholesterol by ≥ 50% of baseline and to target LDL goal of < 70 mg/dL • For patients with higher cardiovascular risk, especially those with multiple ASCVD risk factors and an LDL cholesterol > 70 mg/dL, add ezetimibe or a PCSK9 mAb to maximum tolerated statin therapy
		Diabetes mellitus without clinical ASCVD Age > 75	• Reasonable to continue statin treatment if already on statin • Reasonable to initiate moderate-intensity statin therapy with shared decision making

Treatment Overview Cont'd

STATIN BENEFIT GROUPS AND TREATMENT RECOMMENDATIONS			
PREVENTION STRATEGY	**GROUP**	**RISK LEVEL**	**RECOMMENDATIONS**
Primary Prevention, cont'd	4	**Primary prevention in nondiabetics** Age 20-39 See algorithm page 8	• Patient with family history of premature ASCVD and LDL-C ≥ 160 mg/dL, consider adding statin
		Primary prevention in nondiabetics Age 40-75 See algorithm page 8	LDL-C between 70 and 190 mg/dL, calculate estimated 10-year ASCVD risk percentage and engage in risk discussion • Low risk (<5%): emphasize therapeutic lifestyle changes • Borderline risk (5% to < 7.5%): consider starting moderate-intensity statin. Consider measuring CAC if risk decision uncertain. • Intermediate risk (≥ 7.5% to < 20%): recommend starting moderate-intensity statin with goal LDL-C lowering 30-49%. Consider measuring CAC if risk decision uncertain. • High risk (≥ 20%): recommend starting high-intensity statin with goal LDL-C lowering ≥ 50%. Also reasonable is maximally tolerated statin with ezetimibe or bile acid sequestrant with goal LDL-C lowering ≥ 50%.

* Since the 2018 publication of American Heart Association (AHA)/American College of Cardiology (ACC) Guideline on the Management of Blood Cholesterol, the US Food and Drug Adnimistration (FDA) approved several new LDL lowering therapies that were not included in the 2018 national guideline so the ACC published the 2022 Expert Consensus Decision Pathway (ECDP) on the Role of Non-statin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk. Please note that the ECDP process did not involve formal systematic reviews, grading of evidence, or synthesis of evidence. The goal of the 2022 publication was to provide practical guidance in clinical situations not covered by the 2018 AHA/ACC cholesterol guideline.

† Based on the 2022 ECDP on the Role of Non-statin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk and the 2023 American Diabetes Association (ADA) Standards of Care in Diabetes recommendations.

Very High-Risk* ASCVD Group	
MAJOR ASCVD EVENTS	**HIGH-RISK CONDITIONS**
Recent ACS within the past 12 months	Age ≥ 65 y
History of MI other than recent ACS	Heterozygous FH
History of ischemic stroke	History of prior CABG or PCI outside of major ASCVD events
Symptomatic PAD (history of claudication with ABI < 0.85 or previous revascularization or amputation)	DM
	HTN
	CKD (eGFR 15-59 mL/min)
	Current smoking
	Persistently elevated LDL-C ≥ 100 mg/dL despite maximally tolerated statin therapy and ezetimibe
	History of CHF

* Very high-risk: a history of multiple major ASCVD events or 1 major ASCVD event with multiple high-risk conditions.

CHF = congestive heart failure
CKD = chronic kidney disease
eGFR = estimated glomerular filtration rate
MI = myocardial infarction

Treatment Overview Cont'd

RISK-ENHANCING FACTORS FOR PROVIDER-PATIENT RISK DISCUSSION
Family history of premature ASCVD (males, age <55 y; females, age <65 y)
Primary hypercholesterolemia (LDL-C 160–189 mg/dL [4.1–4.8 mmol/L); non–HDL-C 190–219 mg/dL [4.9–5.6 mmol/L])
Metabolic syndrome (increased waist circumference, elevated triglycerides [≥150 mg/dL], elevated blood pressure, elevated glucose, and low HDL-C [<40 mg/dL in men; <50 mg/dL in women] are factors; tally of 3 makes the diagnosis)
Chronic kidney disease (eGFR 15–59 mL/min/1.73 m2 with or without albuminuria; not treated with dialysis or kidney transplantation)
Chronic inflammatory conditions such as rheumatoid arthritis, psoriasis, ankylosis spondylitis, vasculitis, systemic erythematous lupus, HIV, hepatitis C, etc.
History of premature menopause (before age 40 y) and **history of pregnancy-associated conditions** that increase later ASCVD risk such as preeclampsia
High-risk race/ethnicities (e.g., South Asian ancestry)

Lipid/biomarkers associated with increased ASCVD risk
• Persistently elevated, primary **hypertriglyceridemia** ≥175 mg/dL
• If measured
  ○ Elevated **high-sensitivity C-reactive protein** ≥ 2.0 mg/L
  ○ Elevated **lipoprotein (a) [Lp(a)])** is a relative indication for its measurement if family history of premature ASCVD. An Lp(a) ≥ 50 mg/dL or ≥ 125 nmol/L constitutes a risk-enhancing factor especially at higher levels of Lp(a).
  ○ Elevated **apoB** ≥ 130 mg/dL is a relative indication for its measurement would be TG ≥ 200 mg/dL. A level of ≥ 130 mg/dL corresponds to an LDL-C ≥ 160 mg/dL and constitutes a risk factor.
• **ABI** < 0.9

MONITORING OVERVIEW

Starting at 20 years of age, it is reasonable to assess traditional ASCVD risk factors every 4 to 6 years with either a fasting or a nonfasting lipid panel to document baseline LDL-C for patients who have low ASCVD risk. For patients with elevated risk who decide not to start lipid-lowering therapy, it is reasonable to reassess ASCVD risk factors and recheck a nonfasting lipid panel annually. If the nonfasting lipid panel reveals triglycerides of 400 mg/dL or higher, do a fasting lipid panel for assessment of fasting triglyceride levels and baseline LDL-C. For adults with LDL-C < 70 mg/dL, measurement of direct LDL-C or modified LDL-C estimate may be ordered for improved accuracy. Fasting and nonfasting TC and HDL-C levels appear to have fairly similar prognostic value and associations with CVD outcomes.

Two lipoprotein entities related to LDL-C are apoB and Lp(a). Under certain circumstances, particularly in patients with hypertriglyceridemia (persistent fasting TG ≥ 200 mg/dL), measurement of apoB may have advantages. ApoB level > 130 mg/dL corresponds to LDL-C ≥ 160 mg/dL and constitutes a risk-enhancing factor.

Lp(a) is a modified form of LDL. Relative indications to measure Lp(a) are family history of premature ASCVD or personal history of ASCVD not explained by major risk factors. Lp(a) level > 50 mg/dL constitutes a risk-enhancing factor.

Once lipid-lowering drug therapy has started, check patient's fasting lipids:

- 1-3 months after starting treatment
- 1-3 months after dose adjustment until within the therapeutic range. See page 32 for information on monitoring side effects.

Once patient has reached the appropriate/optimal lipid levels:

- Check fasting lipids every 12 months, unless there are adherence problems or other reasons for more frequent testing, such as changes in therapy

Measure total creatinine kinase levels (total CK) in patients with statin-associated muscle symptoms and/or objective muscle weakness. Measure transaminases (AST, ALT), total bilirubin, and alkaline phosphatase if there are symptoms suggesting hepatotoxicity. In patients with chronic, stable liver disease, including non-alcoholic fatty liver disease (NAFLD), check a baseline measurement of transaminases (AST, ALT), total bilirubin, and alkaline phosphatase, then schedule monitoring labs. An asymptomatic increase in transaminases more than 3 times the upper limit of normal is an infrequent statin-associated side effect that often resolved with dose reduction or rechallenge with an alternative statin. Severe statin-associated hepatotoxicity is rare and not impacted by routine monitoring of transaminases. Total CK and transaminase levels should NOT be routinely measured given the unlikely impact on clinical outcomes and the lack of established cost effectiveness. Total CK and transaminase levels should NOT be routinely measured given the unlikely impact on clinical outcomes and the lack of established cost effectiveness.

DECISION SUPPORT ALGORITHM

PRIMARY PREVENTION

Patient presents with suspected dyslipidemia. Perform comprehensive evaluation including:

- Personal and family history, including ASCVD risk factors and physical exam (see pages 4-6)
- Baseline labs: lipid panel typically required prior to prescribing statin therapy (may be non-fasting if initial screen), CMP, HbA1c, and TSH (see page 6 for details)
- Diagnostic studies as indicated such as ECG (resting), stress tests (treadmill, chemical, nuclear)

→ **Patients with Clinical ASCVD** See Secondary Prevention algorithm next page

Primary Prevention: Assess ASCVD Risk

- Emphasize Adherence to Healthy Lifestyle

If LDL-C ≥ 190 mg/dL

No risk assessment; High intensity statin (Class I)

Diabetes* and age 40-75

Moderate-intensity statin (Class I)

Diabetes* and age 40-75

Risk Assessment to consider High-intensity statin (Class IIa)

Age > 75: Clinical assessment, risk discussion

Age 18-39
Estimate Lifetime Risk

- Encourage lifestyle changesto prevent or reduce ASCVD risk
- Consider statin if family history of premature ASCVD, and LDL-C ≥ 160 mg/dL

Age 40-75 and LDL-C ≥ 70 to < 190 mg/dL without Diabetes

- Estimate 10-year ASCVD Risk % to begin risk discussion

< 5% **Low Risk**	**5 – < 7.5%** **Borderline Risk**	**7.5% - < 20%** **Intermediate Risk**	**≥ 20%** **High Risk**
Risk Discussion Emphasize lifestyle to reduce risk factors (Class I)	If Risk Enhancers** present then risk discussion regarding moderate-intensity statin therapy (Class IIb)	**Risk Discussion** If risk estimate + risk enhancers favor statin, initiate moderate-intensity stat in to reduce LDL-C by 30-49% (Class I)	**Risk Discussion** Initiate statin to reduce LDL-C ≥ 50% (Class I)

If risk is uncertain, discuss with patient the consideration of additional risk factors including measuring Coronary Artery Calcium (CAC) in selected adults. For additional information on CAC including scoring, see page 6.

****ASCVD Risk Enhancers**

- Family history of premature ASCVD
- Persistently elevated LDL-C ≥ 160 mg/dL
- Chronic Kidney Disease (CKD)
- Metabolic Syndrome
- Conditions specific to women (e.g., preeclampsia, premature menopause)
- Inflammatory diseases (esp. rheumatoid arthritis, psoriasis, HIV)
- Ethnicity factors: (e.g., South Asian)

Lipid Biomarkers:

- Persistently elevated triglycerides(≥ 175 mg/dL)

In selected individuals if measured:

- Hs-CRP 2.0 mg/dL
- Lp(a) levels > 50 mg/dL or > 125 nmol/L
- apoB 130 mg/dL
- Ankle-brachial index < 0.9

ACC/AHA Class of Recommendations

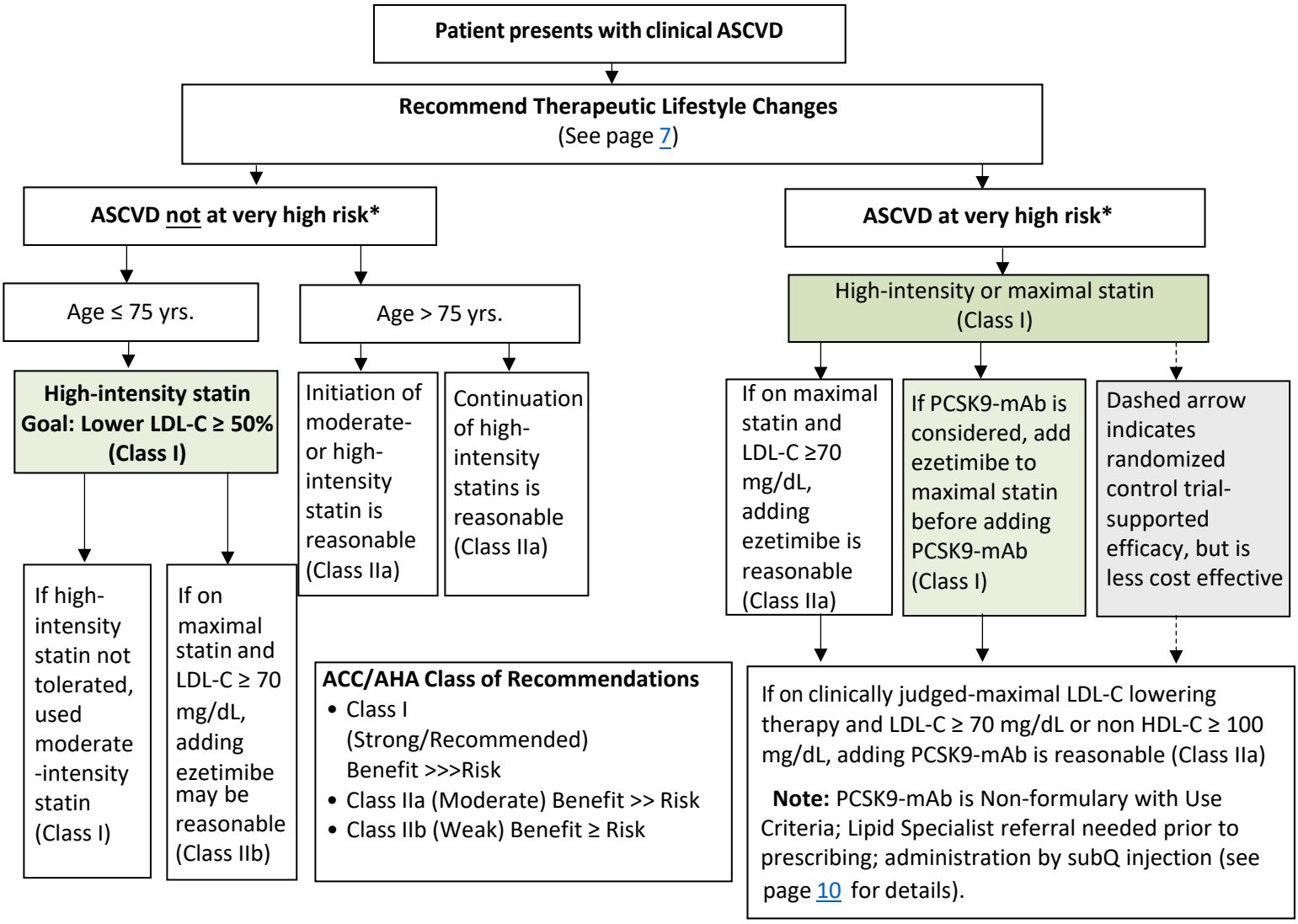
- Class I (Strong/Recommended) Benefit >>> Risk
- Class IIa (Moderate) Benefit >> Risk
- Class IIb (Weak) Benefit ≥ Risk

***Diabetes Specific Risk Factors independent of other Risk Factors in Diabetes**

- Long duration (10 years for type 2 DM or 20 years for type 1 DM)
- Albuminuria ≥ 30 mcg albumin/mg creatinine
- eGFR < 60 ml/min/1.73 m2
- Retinopathy
- Neuropathy
- ABI < 0.9

DECISION SUPPORT ALGORITHM Cont'd

SECONDARY PREVENTION



***Very High-Risk for Future ASCVD Events**

Major ASCVD Events

- Recent acute coronary syndrome (ACS) (within the past 12 months
- History of myocardial infarction (MI) (other than recent ACS event listed above)
- History of ischemic stroke
- Symptomatic peripheral arterial disease (PAD) (history of claudication with ankle-brachial index [ABI] <0.85 or previous revascularization or amputation

High-Risk Conditions

- Age ≥ 65 years
- Current smoking
- History of prior coronary artery bypass surgery or PCI outside of the major ASCVD event(s)
- Chronic Kidney Disease (eGFR 15-59 mL/1.73m^2)

Note: In adults with dialysis-dependent CKD, KDIGO recommends avoiding initiation of statins or statin/ezetimibe combinations. However, there is no recommendation to stop therapy in dialysis patients who are already receiving statins or statin/ezetimibe combinations.[13]

- Diabetes
- Hypertension
- Heterozygous familial hypercholesterolemia
- Persistently elevated LDL-C ≥ 100 mg/dL despite normally tolerated statin therapy and ezetimibe
- History of congestive heart failure

DECISION SUPPORT OVERVIEW

Circulating lipids, such as cholesterol and TG, are insoluble in plasma. They are made soluble by inclusion into circulating lipoproteins. The lipoprotein consists of esterified and unesterified cholesterol, TGs, phospholipids, and protein. There are five major lipoproteins in blood: chylomicrons, very low-density lipoprotein (VLDL), intermediate-density lipoprotein, LDL-C, and HDL-C.

Dyslipidemias are disorders of lipoprotein metabolism that can lead to atherosclerosis. Dyslipidemias were traditionally classified by patterns of elevation in lipids and lipoproteins (Fredrickson phenotype). A more practical system categorizes dyslipidemias as primary or secondary and characterizes them by:

- Increases in cholesterol only (pure or isolated hypercholesterolemia)
- Increases in TGs only (pure or isolated hypertriglyceridemia) - covered in Attachment C
- Increases in both cholesterol and TGs (mixed or combined hyperlipidemias) - most common

This system does not consider specific lipoprotein abnormalities (e.g., low HDL-C or high LDL-C) that may contribute to disease despite normal cholesterol and TG levels.

Primary and secondary dyslipidemia is discussed below:

Primary	Genetic	Genetic dyslipidemia caused by a single or multiple gene mutations that results in either overproduction or defective clearance of LDL-C or TGs, or underproduction or excessive clearance of HDL-C. Familial combined hyperlipidemia, familial hypertriglyceridemia, and homozygous familial are probably genetic hypercholesterolemias. Genetic dyslipidemias are briefly mentioned in this Care Guide given the uncommon occurrence and scarce clinical trial data. See Appendix A.
Secondary	Lifestyle factors	Obesity (especially excess weight around the waist) smoking, excess consumption of fat (especially saturated and trans fats) and diet high in refined carbohydrates.
	Medical conditions	Diabetes, hypothyroidism, alcohol use disorder, metabolic syndrome, severe infections such as HIV, cholestatic liver disease, nephrotic syndrome, and chronic kidney disease. Levels of cholesterol and TGs naturally increased during pregnancy. Statins are contraindicated during pregnancy and breastfeeding.
	Medications	Thiazides, beta-blockers, oral estrogens, atypical antipsychotics, tamoxifen, glucocorticoids, HIV antiretroviral therapy, retinoids, cyclosporine.

Additional factors affecting severity of dyslipidemia and cardiovascular risk:

- **ASCVD risk factors**: Severity of ASCVD 10-year risk estimate is based on age, sex, race, total cholesterol, HDL-C, blood pressure, history of diabetes mellitus, and smoking history.
- **ASCVD risk enhancers**: In intermediate risk adults, risk enhancers are factors, if present, favor statin initiation or intensification. See page 4 for additional details. Some examples include:
  - Chronic conditions, such as metabolic syndrome, chronic kidney disease, premature menopause
  - Laboratory abnormalities, such as hypertriglyceridemia or elevated Lp(a) or apoB
  - High risk race/ethnicity, which can influence ASCVD risk when making treatment decisions. Different cardiovascular outcomes persist based on sociodemographic characteristics that include, but are not limited to, age, sex, and race/ethnicity. There are specific race/ethnic factors to consider for South/Southeast Asian, Pacific Islander, Hispanic/Latinx, Black Americans, and Native American/Alaskan Native in the evaluation, risk decisions, and treatment, due to potential underestimation of risk when using Cohort Equations derived from White populations. There is also potential for over-estimation of risk for East Asian Americans. Important characteristics to consider include:
    - Higher ASCVD risk in South Asians
    - ASCVD risk is higher among individuals from Puerto Rico and Mexico
    - Black women show increased ASCVD risk compared to non-Hispanic White women
    - Native American/Alaskan Natives have higher rates of risk factors for ASCVD versus non-Hispanic Whites

EVALUATION

HISTORY

Conduct a history including both personal and family, paying particular attention to the following:

- **Personal**: Smoking, diet, physical activity, impaired glucose tolerance, metabolic syndrome, DM, obesity, HTN, dyslipidemia, cardiovascular or cerebrovascular events, CKD, Non-Alcoholic Fatty Liver Disease (NAFLD)/Non-Alcoholic Steatohepatitis (NASH), autoimmune/inflammatory disease (e.g., lupus, rheumatoid arthritis, psoriasis), hepatitis C, history of pancreatitis, medications that alter lipids (e.g., steroids, retinoids, HIV antiretroviral therapy (ART), post transplantation antirejection medications)
  - Evidence supports the association between ASCVD and mental health issues. Depression is an independent risk factor as well as more prevalent in patients with ASCVD. Also, schizophrenia, bipolar disorder, anxiety, and post-traumatic stress disorder have been found to increase the risk for ASCVD. Care coordination and collaboration with mental health is very important.
- **Family**: Cardiovascular disease (CVD), HTN, dyslipidemia
- Identify **medical conditions** that increase the patient's risk of dyslipidemia and or ASCVD:
  - Prediabetes/impaired glucose tolerance
  - Metabolic syndrome
  - Overweight/obesity
  - Medications (e.g., steroids, retinoids, ART, antirejection medications, etc.)
  - Diabetes mellitus
  - Hypertension
  - NAFLD/NASH
  - Prior cardiovascular/ cerebrovascular events
  - Autoimmune diseases
  - Hepatitis C HIV
  - HIV

DETERMINE ASCVD RISK

Assessment of ASCVD risk is the foundation of primary prevention with the aim of treating dyslipidemia to delay or prevent new-onset ASCVD. **Note**: In patients without known ASCVD (Primary Prevention) no risk assessment is needed for those with LDL ≥ 190 mg/dL – these patients have a high lifetime risk of ASCVD, and high intensity statins should be started.

ASCVD Risk Factors: For most patients estimating the individual's 10-year absolute ASCVD risk is fundamental in establishing appropriate medical management and enables matching the intensity of preventive interventions to the patient's absolute risk. This helps to maximize anticipated benefit and minimize potential harm from overtreatment. The estimate is based on sex, age, race, total cholesterol, HDL, blood pressure, history of DM, and smoking history.

Use ACC's new ASCVD Risk Estimator Plus Equation to determine patient's 10-year ASCVD risk.
This tool gives an estimate of the patient's risk of a cardiovascular event within the next 10 years, categorized as follows:

- Low risk: < 5%
- Borderline risk: 5% - < 7.5%
- Intermediate risk: 7.5% - < 20%
- High risk: ≥ 20%

After evaluating 10-year risk, clinicians should discuss it with the patient before initiating statin therapy. Risk discussions are the cornerstone of the shared decision-making process.

HEALTH EQUITY ALERT

Race and ethnicity factors can influence estimations of ASCVD risk. The PCE may underestimate risk in individuals of South Asian ancestry and other high-risk groups and may overestimate risk in selected lower-risk groups. Therefore, for clinical decision-making in adults of different races and ethnicities, it is reasonable for clinicians to review race and ethnic features that can influence ASCVD risk to adjust the choice of statin or intensity of treatment.

The CAC score predicts ASCVD events in a graded fashion and is independent of other risk factors, such as age, sec, and race/ethnicity.

Additional Risk "Enhancers": In intermediate-risk adults, risk-enhancing factors that favor statin initiation or intensification include:

- Metabolic syndrome
- CKD
- Chronic inflammatory conditions
- Premature menopause and preeclampsia
- High risk race/ethnicity
- Persistent TG ≥ 175 mg/dL
- Elevated Lp(a)
- Elevated apoB

Evaluation Cont'd

PHYSICAL EXAM

Complete comprehensive physical exam, paying particular attention to the following:

- Height, weight, BMI, waist circumference, blood pressure, cardiac evaluation, peripheral and carotid pulses, vascular bruits

High LDL-C can cause tendinous xanthomas and xanthelasmas (lipid rich yellow plaques on the medial eyelids)

- Severe hypertriglyceridemia (> 2000 mg/dL [> 22.6 mmol/L]) may present with gastrointestinal pain (TGs > 150 mg/dL can cause acute pancreatitis), paresthesia, dyspnea, confusion, and dementia

LABS

Order the following initial labs:

- Lipid Panel includes: Total Cholesterol, HDL-C, TGs, LDL-C, and calculated non-HDL-C (see Appendix B)
  ACC/American Heart Association (AHA) note that the maximal difference between random non-fasting lipids (1-6 hours after meal) and fasting lipids is **not clinically significant** (26 mg/dL for TG and 8 mg/dL for total cholesterol, LDL-C, and non-HDL-C) and can effectively guide ASCVD prevention. LDL-C can be estimated from TC, HDL-C, and TG measurements. They recommend:
  - General screening LP can be done non-fasting
  - Do fasting LP prior to starting statins, when TG > 500 mg/dL and while on statin therapy
  - Some guidelines recommend checking LP twice before beginning lipid-lowering drug treatment
- CMP including uric acid
- HbA1c
- TSH

Tests for secondary causes of dyslipidemia should be done in most patients with newly diagnosed dyslipidemia and when a component of the lipid profile has inexplicably changed for the worse. Such tests include:

- Fasting glucose
- Liver enzymes

Additional labs only to be considered based on individual patient circumstances:

- **ApoB**: May be measured when trying to evaluate a patient's risk of developing ASCVD and when there is a personal or family history of heart disease and/or abnormal lipid levels, especially in patients with significantly elevated TGs
- **Lp(a)**: May be considered for patients with a family history of early ASCVD or to refine the evaluation of patients at moderate risk; high concentrations may support more aggressive control of other lipoprotein factors
- **High-sensitivity C reactive protein (hsCRP)**: May help stratify risk in individuals with a borderline risk assessment or with intermediate or high risk and an LDL-C measurement of < 130 mg/dL. A hsCRP test should not be performed in patients with current acute illness associated with the presence of heart disease.

DIAGNOSTIC STUDIES

- EKG resting, stress tests (treadmill, chemical, nuclear) as appropriate
- Imaging: CAC scoring if risk decision is uncertain in selected adults, consider carotid and/or femoral ultrasound and/or carotid intima-media thickness test (CIMT)
- CAC testing: If uncertain about the need for statin this test can be done to add to the information on future ASCVD risk. AHA considers it an option in patients in intermediate-risk (7.5% - < 20%) or selected borderline-risk adults with uncertainty about statins)

CAC testing strongly informs ASCVD risk discrimination and reclassification regardless of race, ethnicity, or gender and informs shared decision making about allocating statin therapy based on ASCVD risk. CAC testing also aids in shared decision making about aspirin and antihypertensive therapy.

- CAC 0 AU, indicates low ASCVD risk for 10 years, reasonable to withhold statin and reassess in 5-10 years
- CAC 1-99 AU and age ≥ 55 years, reasonable to initiate statin
- CAC ≥ 100 AU or ≥ 75th percentile for age/sex/race group, reasonable to initiate statin

Evaluation Cont'd

PATIENT EDUCATION

- Review therapeutic lifestyle recommendations (see page 7)
- Explain relationship of dyslipidemia to ASCVD and importance of overall attention to ASCVD risk factors to empower patients to participate in their care
- If the patient is prescribed medication, discuss importance of taking as prescribed, and encourage the patient to be open with their primary care team if they have concerns or side effects that may cause them to non-adhere
- Discuss potential side effects of medications
- See PE-1 through PE-3 for details

TREATMENT

THERAPEUTIC LIFESTYLE

The main indication for dyslipidemia treatment is prevention of ASCVD, including acute coronary syndromes, stroke, transient ischemic attack (TIA), or peripheral arterial disease, which are presumed to be caused by atherosclerosis.

Lifestyle management is the foundation of all lipid-reduction treatment regimens. Managing patients with dyslipidemia, begins with implementation of lifestyle changes including nutrition therapy, physical activity, smoking cessation, and assessment of sleep issues.

Nutrition	For primary and secondary prevention of ASCVD, encourage patients to consume CDCR Heart Healthy Diet with a focus on fruits and vegetables, whole grains, legumes, high soluble fiber, and moderate intake of low-fat dairy products while avoiding processed foods and saturated fat. Encourage patients to make healthier canteen choices. Goals of these dietary recommendations: • Reduce percentage of calories from saturated fat and trans-fat for LDL-C lowering • Lower sodium intake (no more than 2400 mg of sodium per day) for blood pressure lowering • For overweight or obese patients who would benefit from weight loss, reduced calorie intake for weight loss
Physical Activity	For primary and secondary prevention of ASCVD, recommend regular aerobic physical activity. Specific lipid-level improvements associated with regular exercise include: • Reduced triglyceride and VLDL-C levels • Reduction in total cholesterol • Increased HDL-C • Reductions in hsCRP • Reduced LDL-C levels • Reduced non-HDL cholesterol Evidence suggests cardiovascular benefits at various levels of physical activity: At least 150 minutes (2 hours and 30 minutes) of aerobic activity of moderate or greater intensity per week and 2 days of muscle-strengthening activity. Consider recommending 30 minutes of activity at least 5 days a week, and providing examples to patients (i.e., brisk walking, jogging, push-ups, sit-ups, body-weight squats). For patients with a recent occurrence of coronary heart disease (CHD) (i.e., myocardial infarction, diagnosis of coronary artery disease, coronary artery bypass grafting, or percutaneous coronary intervention), a structured, exercise-based cardiac rehabilitation program should be considered to reduce cardiovascular morbidity and mortality. Development of a safe and effective program may require coordination with specialists.
Sleep	Sleep deprivation aggravates insulin resistance, HTN, hyperglycemia, and dyslipidemia, and increases inflammatory cytokines. Encourage patients to get 6-8 hours/night and avoid sleeping pills.
Smoking	Smoking is a major risk factor for ASCVD and may triple the risk of death due to atherosclerosis. Encourage abstinence from tobacco or nicotine related products; avoid passive exposure tobacco smoke.

Treatment Cont'd

FIRST-LINE THERAPY: STATINS

Statins (HMG-CoA reductase inhibitors) should be used as first-line cholesterol-lowering therapy, unless contraindicated. However, considerable residual risk often persists even after aggressive statin monotherapy in primary prevention in patients with multiple cardiovascular risk factors and in secondary prevention patients with stable clinical ASCVD.

Statin Groups: Recommend treatment with a statin for four different groups of patients as listed in the following table:

STATIN BENEFIT GROUPS AND TREATMENT RECOMMENDATIONS			
PREVENTION STRATEGY	**GROUP**	**RISK LEVEL**	**RECOMMENDATIONS**
SECONDARY PREVENTION AGE 18+	1	**Very high risk ASCVD** See algorithm page 9	Maximally tolerated statin is recommended: • If LDL-C ≥ 70 mg/dL, consider adding ezetimibe • If persistent LDL-C ≥ 70 mg/dL or non-HDL–C ≥ 100, consider adding PCSK9 monoclonal antibody (PCSK9 mAb). Specialty referral advised. *If LDL–C ≥ 55 mg/dL and/or LDL-C ≥ 50% reduction not achieved, consider adding ezetimibe or PCSK9 mAb.**
		Stable ASCVD See algorithm page 9	High- or moderate-intensity statin is recommended: • If high-intensity statin, goal LDL-C lowering ≥ 50% • If moderate-intensity statin, goal LDL-C lowering 30-49% • Increase statin intensity and/or add ezetimibe with inadequate LDL-C reduction. Specialty referral advised.
		Diabetes mellitus with ASCVD	Maximally tolerated statin is recommended: *If LDL–C ≥ 55 mg/dL and/or LDL-C ≥ 50% reduction not achieved, consider adding ezetimibe or PCSK9 mAb. Specialty referral advised.*†
	2	**Severe hypercholesterolemia** Requires no ASCVD risk assessment	LDL-C ≥ 190 mg/dL, maximally tolerated statin. Specialty referral advised. • If LDL-C ≥ 50% reduction not achieved, consider adding ezetimibe • If LDL-C ≥ 50% reduction still not achieved, consider adding PCSK9 mAb If LDL-C ≥ 50% reduction still not achieved and fasting TG's ≥ 300 mg/dL, consider bile acid sequestrant Heterozygous FH aged 30-75 years, maximally tolerated statin. Specialty referral advised. • If LDL-C ≥ 100 mg/dL, consider adding ezetimibe • If persistent LDL–C ≥ 100 mg/dL, consider adding PCSK9 mAb
PRIMARY PREVENTION ***Assess ASCVD risk in each age group. Emphasize adherence to heart healthy lifestyle.***	3	**Diabetes mellitus without clinical ASCVD** Age 20-39	• For patients with diabetes aged 20–39 years with additional ASCVD risk factors, it may be reasonable to initiate statin therapy in addition to lifestyle therapy
		Diabetes mellitus without clinical ASCVD Age 40-75	• Irrespective of estimated 10-year ASCVD risk percentage, use at least moderate-intensity statin therapy • For patients with higher cardiovascular risk, including those with one or more ASCVD risk factors, use high-intensity statin therapy to reduce LDL cholesterol by ≥ 50% of baseline and to target LDL goal of < 70 mg/dL For patients with higher cardiovascular risk, especially those with multiple ASCVD risk factors and an LDL cholesterol > 70 mg/dL, add ezetimibe or a PCSK9 mAb to maximum tolerated statin therapy

Treatment Cont'd

STATIN BENEFIT GROUPS AND TREATMENT RECOMMENDATIONS			
PREVENTION STRATEGY	**GROUP**	**RISK LEVEL**	**RECOMMENDATIONS**
PRIMARY PREVENTION, cont'd		**Diabetes mellitus without clinical ASCVD** Age > 75	• Reasonable to continue statin treatment if already on statin • Reasonable to initiate moderate-intensity statin therapy with shared decision making
	4	**Primary prevention in nondiabetics** Age 20-39 See algorithm page 8	• Patient with family history of premature ASCVD and LDL-C ≥ 160 mg/dL, consider adding statin
		Primary prevention in nondiabetics Age 40-75 See algorithm page 8	LDL-C between 70 and 190 mg/dL, calculate estimated 10-year ASCVD risk percentage and engage in risk discussion • Low risk (<5%): emphasize therapeutic lifestyle changes • Borderline risk (5% to < 7.5%): consider starting moderate-intensity statin. Consider measuring CAC if risk decision uncertain • Intermediate risk (≥ 7.5% to < 20%): recommend starting moderate-intensity statin with goal LDL-C lowering 30-49%. Consider measuring CAC if risk decision uncertain • High risk (≥ 20%): recommend starting high-intensity statin with goal LDL-C lowering ≥ 50%. Also reasonable is maximally tolerated statin with ezetimibe or bile acid sequestrant with goal LDL-C lowering ≥ 50%

* Since the 2018 publication of AHA/ACC Guideline on the Management of Blood Cholesterol, the FDA approved several new LDL lowering therapies that were not included in the 2018 national guideline so the ACC published the 2022 ECDP on the Role of Non-statin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk. Please note that the ECDP process did not involve formal systematic reviews, grading of evidence, or synthesis of evidence. The goal of the 2022 publication was to provide practical guidance in clinical situations not covered by the 2018 AHA/ACC cholesterol guideline.

† Based on the 2022 ECDP on the Role of Non-statin Therapies for LDL-Cholesterol Lowering in the Management of Atherosclerotic Cardiovascular Disease Risk and the 2023 American Diabetes Association (ADA) Standards of Care in Diabetes recommendations.

Statin Intensity: Based on the defined statin group above, select the appropriate statin intensity as listed below and options for specific medications.

	High-intensity	Moderate-intensity	Low-intensity
LDL-C Lowering *	≥ 50%	30-49%	< 30%
First-line Statins	**Atorvastatin 40 – 80 mg** **Rosuvastatin 20 – 40 mg**	**Atorvastatin 10 – 20 mg** **Rosuvastatin 5 – 10 mg** **Pravastatin 40 – 80 mg**	**Pravastatin 10 – 20 mg**
Other Statins		Simvastatin 20 – 40 mg Lovastatin 40 – 80 mg Fluvastatin XL 80 mg Fluvastatin 40 mg BID Pitavastatin 2 – 4 mg	Simvastatin 10 mg Lovastatin 20 mg Fluvastatin 20 – 40 mg Pitavastatin 1 mg

* Percentage reductions are estimates from data across large populations. Individual responses to therapy varied in randomized controlled trials.

Statin Selection: Once the intensity of the statin is determined, select a preferred statin based on other factors. See table below with CCHCS formulary agents followed by non-formulary. See medication pages 17 for non-statin therapy.

Treatment Cont'd

Factors to Consider in Statin Selection					
	Greatest LDL-C Reduction	Preferred in CKD	Preferred in liver disease	Fewer DDI	Possibly less myopathy
Atorvastatin	Yes	Yes			
Rosuvastatin	Yes			Yes	
Pravastatin			Yes	Yes	Yes
Simvastatin					
Lovastatin					
Fluvastatin		Yes		Yes	Yes
Pitavastatin				Yes	

Bold = Formulary

Statin Contraindications: For the majority of patients, statins are safe, however:

- Patients with active hepatic disease or unexplained persistent elevations in aminotransferase levels.
- Pregnancy and breastfeeding because of effects on the cholesterol pathway.
- CKD patients NOT on dialysis may be started and continued on statins.
  - Patients starting dialysis may continue a statin if already on a statin.
  - Patients on dialysis should NOT be started on statins.

Statin-Associated Side Effects: For most patients, statins are safe and well-tolerated. However, statin-associated side effects (SASE) may occur in a significant subset of statin-treated patients, which are discussed in detail on pages 33-34.

- **Statin-associated muscle symptoms (SAMS):** Most frequent SASE
- **New onset DM**: More frequent if associated with BMI ≥ 30, FBS ≥ 100 mg/dL, or A1c ≥ 6%
- **Hepatic dysfunction**: Transaminase elevation 3x ULN – Infrequent
- **CKD/ESRD:** Patients on hemodialysis should **NOT** be started on a statin

Statin-Associated Muscle Symptoms		
Myalgias	CK normal	Musculoskeletal manifestations are well-recognized side effects of treatment with statins. These side effects are reported to occur in about 10% of people prescribed statins. See page 33-34 for details.
Myopathy	CK > 10x ULN	Characterized most of the time by mild to moderate hypercreatinemia. Rare incidence. See page 33-34 for details.
Rhabodomyolysis	CK > 40x ULN	Defined by the Task Force as myonecrosis, which can cause myoglobinuria and acute renal failure. Patients with symptomatic or asymptomatic rhabdomyolysis from a statin should discontinue therapy immediately. See page 33-34 for details.
Statin-Induced Necrotizing Autoimmune Myopathy (SINAM)	CK > 40x ULM Positive anti-HMGCR antibody	Proximal muscle weakness, which can cause myalgia, dysphagia, and/or respiratory failure. Persistent/progressive symptoms even with discontinuation of statin. Treated with immunosuppression. Rare incidence but potentially life-threatening.

Treatment Cont'd

NON-STATIN LDL-C LOWERING MEDICATIONS

Non-statin medications may be useful in combination with statin therapy. For secondary prevention: LDL-C ≥ 70 mg/dL is the threshold for non-statin drug consideration.

Ezetimibe is the most used non-statin agent. It lowers LDL-C levels by 18% to 20% and has a low incidence of side effects.

- Recommended in combination with statin therapy in patients who do not meet LDL-C goals (threshold levels) with dietary modification and maximally tolerated statin therapy.
- Ezetimibe may also be used in situations of statin-associated muscle symptoms. Combination therapy with statins is relatively safe.

Bile acid sequestrants reduce LDL-C levels by 15% to 30% depending on the dose. Bile acid sequestrants are not absorbed and do not cause systemic side effects, but they are associated with gastrointestinal complaints (e.g., constipation) and can cause severe hypertriglyceridemia when fasting triglycerides are ≥ 300 mg/dL (≥ 3.4 mmol/L).

- Recommended use in patients 20 to 75 years of age with a baseline LDL-C 190 mg/dL or higher (4.9 mmol/L), who achieve less than a 50% reduction in LDL-C levels and have fasting triglycerides 300 mg/dL or lower (-3.4 mmol/L) while taking maximally tolerated statin and ezetimibe therapy, the addition of a bile acid sequestrant may be considered. Cholestyramine is the bile acid sequestrant that is on formulary.

PCSK9 monoclonal antibodies (PCSK9 mAb) are powerful LDL-lowering drugs. PCSK9 mAbs (previously referred to as PCSK9 inhibitors or PCSK9i) bind to PCSK9 proteins and increase the number of LDL receptors available to clear circulating LDL-C. There are 2 FDA approved PCSK9 mAbs: alirocumab (Praluent®) and evolocumab (Repatha®). They generally are well-tolerated, but long-term safety reduction requires continued research. PCSK9 mAbs, when used alone or in combination with other lipid-lowering therapy, are administered subcutaneously every 2-4 weeks and are non-formulary. See restrictions on page 28. Adding a PCSK9 mAb may be considered in the following:

- In patients with very high-risk ASCVD (history of multiple major ASCVD events or 1 major ASCVD event and multiple high-risk conditions) and LDL-C level remains ≥ 70 mg/dL on maximally tolerated statin plus ezetimibe therapy.
- In patients with severe primary hypercholesterolemia (LDL-C level ≥ 190 mg/dL) and the LDL-C level on high-intensity or maximally tolerated statin plus ezetimibe remains ≥ 100 mg/dL and the patient has multiple factors that increase subsequent risk of ASCVD events.
- In patients 30 to 75 years of age with Familial Hypercholesterolemia and with an LDL-C level of 100 mg/dL or higher while taking maximally tolerated statin and ezetimibe therapy.
- In patients 40 to 75 years of age with a baseline LDL-C level of 220 mg/dL or higher who have an on-treatment LDL-C level of 130 mg/dL or higher while receiving maximally tolerated statin and ezetimibe therapy.

Inclisiran* (Leqvio®) is an **antilipemic small interfering ribonucleic acid (siRNA)** targeting PCSK9, one of the newest LDL-lowering drugs. This can be used in combination with lifestyle changes and maximally tolerated statin therapy to further decrease LDL cholesterol in adults with heterozygous familial hypercholesterolemia (HeFH) and patients with clinical ASCVD that need to further lower their LDL. Inclisiran is administered by subcutaneous injection every 6 months and is non-formulary. This medication should be prescribed and managed by a specialist.

*Inclisiran is not in the 2018 Guideline on the Management of Blood Cholesterol, as it was FDA approved in 2021.

Adenosine-triphosphate citrate lyase (ACL) inhibitors* are another new class of LDL-lowering drugs. ACL inhibitors are used in combination with lifestyle changes, maximally tolerated statin therapy, and ezetimibe to further decrease LDL cholesterol in adults with HeFH and patients with clinical ASCVD that need to further lower their LDL. The FDA approved bempedoic acid (Nexletol®) and bempedoic acid with ezetimibe (Nexlizet®). Bempedoic acid is administered orally and activated by an enzyme present in liver cells, but not muscle cells. Therefore, bempedoic acid has been considered a possible advantage in patients with statin-associated muscle symptoms. This medication should be prescribed and managed by a specialist.

*ACL inhibitors are not in the 2018 Guideline on the Management of Blood Cholesterol, as they were FDA approved in 2020.

Treatment Cont'd

TREATMENT OPTIONS FOR HOMOZYGOUS FAMILIAL HYPERCHOLESTEROLEMIA

Microsomal Triglyceride Transfer Protein (MTP) inhibitor is one of the treatment options for homozygous familial hypercholesterolemia (HoFH). Lomitapide (Juxtapid®) is administered orally for a daily dose. This medication should be prescribed and managed by a specialist.

Angiopoietin-Like Protein 3 (ANGPTL3) inhibitors* are one of the newest classes of LDL-lowering drugs. ANGPTL3 inhibitors can be used in combination with lifestyle changes and maximally tolerated statin therapy to further decrease LDL cholesterol in patients with HoFH. The first-in-class ANGPTL3 inhibitor is evinacumab (Evkeeza®), which is administered by intravenous infusion every 4 weeks. This medication should be prescribed and managed by a specialist.

*ANGPTL3 inhibitors are not in the 2018 Guideline on the Management of Blood Cholesterol, as they were FDA approved in 2021.

ADDITIONAL MEDICATIONS FOR HYPERTRIGLYCERIDEMIA

Patients occasionally have isolated hypertriglyceridemia or persistent hypertriglyceridemia despite maximally tolerated therapy. See Appendix C. AHA/ACC guidelines recognize two categories:

- Moderate hypertriglyceridemia is defined as TG (fasting or nonfasting) 175 - 499 mg/dl
- Severe hypertriglyceridemia is defined as fasting TG ≥ 500 mg/dl

Statins remain first line therapy. In cases where ASCVD risk is high or other complications exist (e.g., pancreatitis) and patient requires more than lifestyle/diet change and statin, the following medications can be considered.

Fibrates: Triglyceride-lowering medication may also mildly lower LDL-C levels in patients with normal TGs.

- Fenofibrate (formulary) may be considered concomitantly with a low or moderate-intensity statin **only if** the patient benefits from ASCVD risk reduction or TG lowering when TGs are > 500 mg/dl. They may be useful in some patients with severe hypertriglyceridemia.
- Gemfibrozil (nonformulary) should not be initiated in patients on statin therapy because of an increased risk for muscle symptoms and rhabdomyolysis.

Omega-3 polyunsaturated fatty acid (PUFA): There is evidence that omega-3 PUFAs can reduce blood TG levels (third- line therapy) and are currently CCHCS nonformulary. Omega-3 PUFAs available in the U.S. include Lovaza®, Vascepa™, and Omtryg®, which are indicated for use as an adjunct to lifestyle to treat persistent hypertriglyceridemia.

Vascepa™ can be used with maximally tolerated statin therapy to lower the risk of cardiovascular events (myocardial infarction, stroke, coronary revascularization, and unstable angina requiring hospitalization) in adults with persistent hypertriglyceridemia who have clinical ASCVD or who have diabetes mellitus and two or more other cardiovascular risk factors for heart disease. Additionally, Vascepa™, which contains only eicosapentaenoic acid (EPA), does not increase LDL-C levels.

Marine-derived omega-3 dietary supplements: Omega-3 dietary supplements or fish oil are one of the most commonly used over the counter dietary supplements used in the United States. They are taken in large doses to help decrease triglyceride secretion and clear triglycerides. The amount of marine-derived omega-3 fatty acids needed to significantly lower triglyceride (2 to 4 g per meal) is hard to get from a daily diet alone.

Use of these dietary supplements at the required large doses may cause serious side effects. These can include increased bleeding, hemorrhagic stroke, and reduced blood sugar control in diabetics. Negative interactions with other medications, herbal preparations and nutritional supplements are also possible. People with allergies to fish, shellfish, or both may have a severe adverse reaction to using these supplements.

Importantly, dietary supplements are not FDA-regulated and are classified as "food" not "drug", so their content and efficacy often remain unverified. Moderate-to-high evidence exists to indicate that fish oil dietary supplement intake does not affect major cardiovascular events, total coronary heart disease, coronary revascularization, all-cause mortality, sudden cardiac death, atrial fibrillation, or blood pressure.

Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
HMG CoA Reductase inhibitors (statins) - Risk of skeletal muscle effects (e.g., myopathy, rhabdomyolysis) increases with higher doses and concomitant use of certain drugs. Predisposing factors include age > 65, female, uncontrolled hypothyroidism, and renal impairment			
Atorvastatin (Lipitor®) **Tablet: 10 mg, 20 mg, 40 mg, 80 mg** **$**	Usual dose: 10-80 mg orally once daily MODERATE-INTENSITY 30% to < 50% reduction in LDL: 10-20 mg orally once daily HIGH-INTENSITY ≥ 50% reduction in LDL: 80 mg orally once daily; 40 mg orally once daily if 80 mg not tolerated Dose adjustments: Max dose 20 mg/day: concomitant use with clarithromycin, erythromycin, itraconazole, ketoconazole, voriconazole, saquinavir plus ritonavir, darunavir plus ritonavir, fosamprenavir, atazanavir plus ritonavir, elbasvir/grazoprevir Max dose 40 mg/day: concomitant use with nelfinavir Renal Impairment: No adjustment needed Hepatic Impairment: Contraindicated inactive liver disease or unexplained/ persistent transaminase elevations	Adverse Reactions: myopathy, rhabdomyolysis, elevated liver enzymes, diarrhea, arthralgia, myalgia, nasopharyngitis, nausea,dyspepsia, urinary tract infection, insomnia, rhabdomyolysis Drug interactions: • Contraindicated with cyclosporine, posaconazole, certain HIV protease inhibitors (tipranavir plus ritonavir), glecaprevir/pibrentasvir • Use caution and lowest dose necessary with HIV protease inhibitor (lopinavir plus ritonavir) • Use caution with niacin, fibrates(avoid gemfibrozil if possible) • Use caution with digoxin, oralcontraceptives, warfarin, colchicine • Consider temporarily suspending use while patient is on daptomycin	CCHCS PREFERRED AGENT Contraindications: hypersensitivity to atorvastatin or any component of the formulation, active liver disease (including cholestasis, hepatic encephalopathy, hepatitis, and jaundice), unexplained/persistent elevations of serum transaminases, pregnancy, breast-feeding, concomitant use with certain HIV protease inhibitors (tipranavir plus ritonavir), glecaprevir/pibrentasvir, cyclosporine Caution in the following: heavy alcohol use (alcoholism), history of liver disease/renal impairment, uncontrolled hypothyroidism, sepsis or severe infection, uncontrolled seizure disorder, recent stroke or transient ischemic attack, electrolyte imbalance, preexisting amyotrophic lateral sclerosis (ALS), the elderly

Bold = Formulary *See prescribing information for complete description of dosing, adverse effects, drug interactions, precautions and contraindications. The cost scale $-$$$$$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment.

Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
HMG CoA Reductase inhibitors (statins) - Risk of skeletal muscle effects (e.g., myopathy, rhabdomyolysis) increases with higher doses and concomitant use of certain drugs. Predisposing factors include age > 65, female, uncontrolled hypothyroidism, and renal impairment			
Rosuvastatin (Crestor®) **Tablet:** **5 mg, 10 mg,** **20 mg, 40 mg** **$**	Usual dose: 5-40 mg orally once daily MODERATE-INTENSITY 30% to < 50% LDL reduction: 5-10 mg orally once daily HIGH-INTENSITY ≥ 50% LDL reduction: 20-40 mg orally once daily Dose adjustments: Asian patients: consider lower starting (5 mg/day) and maximum doses Max dose 5 mg/day: concomitant use with cyclosporine Max dose 10 mg/day: concomitant use with lopinavir plus ritonavir, atazanavir plus ritonavir, elbasvir/grazoprevir, glecaprevir/ pibrentasvir, sofosbuvir/velpatasvir, gemfibrozil Renal Impairment: CrCl < 30 mL/min (not on hemodialysis): Initial dose: 5 mg/day; Max dose 10 mg/day Rosuvastatin levels in hemodialysis patients are about 50% higher than in normal renal function. Hepatic Impairment: Contraindicated inactive liver disease or unexplained/ persistent transaminase elevations	Adverse Reactions: myopathy, rhabdomyolysis, elevated liver enzymes, diarrhea, arthralgia, myalgia, headache, dizziness, constipation, nausea, dyspepsia, rash, rhabdomyolysis Drug interactions: • Contraindicated with sofosbuvir/velpatasvir/voxilaprevir, lasmiditan • Rosuvastatin may be less likely to interact with other drugs • Use caution with niacin, fibrates(avoid gemfibrozil if possible) • Use caution with protease inhibitor/ritonavir combinations, warfarin, colchicine • Consider temporarily suspending use while patient is on daptomycin	Contraindications: hypersensitivity to rosuvastatin or any component of the formulation, active liver disease (including cholestasis, hepatic encephalopathy, hepatitis, and jaundice), unexplained/persistent elevations of serum transaminases, pregnancy, breast-feeding, concomitant use with sofosbuvir/ velpatasvir/voxilaprevir, lasmiditan For Asian patients, consider 5 mg/day starting dose Caution in the following: patients of Asian descent, heavy alcohol use (alcoholism), history of liver disease/renal impairment, uncontrolled hypothyroidism, sepsis or severe infection, uncontrolled seizure disorder, recent stroke or transient ischemic attack, electrolyte imbalance, preexisting amyotrophiclateral sclerosis (ALS), the elderly

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
HMG CoA Reductase inhibitors (statins) - Risk of skeletal muscle effects (e.g., myopathy, rhabdomyolysis) increases with higher doses and concomitant use of certain drugs. Predisposing factors include: age >65, female, uncontrolled hypothyroidism, and renal impairment			
Pravastatin (Pravachol®) **Tablet: 10 mg, 20 mg, 40 mg** **$**	Usual dose: 20-80 mg orally once daily MODERATE-INTENSITY 30% to < 50% reduction in LDL: 40-80 mg orally once daily Dose adjustments: Max dose 20 mg/day: concomitant usewith cyclosporine Max dose 40 mg/day: concomitant usewith clarithromycin, sofosbuvir/ velpatasvir/voxilaprevir Reduce dose by 50%: concomitant usewith glecaprevir/pibrentasvir Renal Impairment: Severe renalimpairment (CrCl < 30 mL/min): Initial dose: 10 mg/day Hepatic Impairment: Contraindicated inactive liver disease or unexplained/ persistent transaminase elevations	Adverse Reactions: myopathy, rhabdomyolysis, elevated liver enzymes, diarrhea, constipation, nausea, vomiting, musculoskeletalpain, myalgia, rash, headache, dizziness, cough, rhinitis, upper respiratory infection Drug interactions: • Pravastatin may be less likely to interact with other drugs • Use caution with niacin, fibrates (avoid gemfibrozil) • Consider dose reduction with niacin dose ≥ 1000 mg/day • Consider temporarily suspending use while patient is on daptomycin	Contraindications: hypersensitivity to pravastatin or any component of the formulation, active liver disease (including cholestasis, hepatic encephalopathy, hepatitis, and jaundice), unexplained/persistent elevation serum transaminases, pregnancy, breast-feeding Caution in the following: heavy alcohol use (alcoholism), history of liver disease/renal impairment, uncontrolled hypothyroidism, sepsis or severe infection, uncontrolled seizure disorder, recent stroke or transient ischemic attack, electrolyte imbalance, preexisting amyotrophic lateral sclerosis (ALS), the elderly

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
HMG CoA Reductase Inhibitors (Statins) - Risk of skeletal muscle effects (e.g., myopathy, rhabdomyolysis) increase with higher doses and concomitant use of certain drugs. Predisposing factors include: age >65, female, uncontrolled hypothyroidism, and renal impairment			
Simvastatin (Zocor®) Tablet: 5 mg, 10 mg, 20 mg, 40 mg Note: 80 mg tabs unavailable within CCHCS - use 40 mg x 2 tabs (see comments) $	Usual dose: 10-40 mg once daily in evening MODERATE-INTENSITY 30% to < 50% reduction in LDL: 20-40 mg once daily in evening Dose adjustments: Max dose 10 mg/day: concomitant use with verapamil, diltiazem, dronedarone Max dose 20 mg/day: concomitant use with amiodarone, amlodipine, ranolazine, lomitapide Renal Impairment: CrCl < 30 mL/min: initial dose, 5 mg/day, close monitoring is advised Hepatic Impairment: Contraindicated inactive liver disease or unexplained/persistent transaminase elevations	Adverse Reactions: myopathy, rhabdomyolysis, elevated liver enzymes, arthralgia, myalgia, vertigo, headache, abdominal pain, gastritis, nausea, constipation, edema, upper respiratory infection, atrial fibrillation, insomnia, sinusitis Drug interactions: • Contraindicated with strong CYP3A4 inhibitors (e.g., erythromycin, clarithromycin, itraconazole, ketoconazole, posaconazole, voriconazole, HIV protease inhibitors, nefazodone, cobicistat), cyclosporine, danazol, gemfibrozil • Use caution with warfarin, niacin, fibrates, digoxin, colchicine • Consider temporarily suspending use while patient is on daptomycin	Note: 80 mg dose is associated with elevated risk of muscle injury. FDA recommends 80 mg dose only for patients who have been taking this dose for at least 12 months without evidence of muscle injury. 80 mg dose should not be started in new patients, including patients already taking lower doses Contraindications: hypersensitivity to simvastatin or any component of the formulation, active liver disease (including cholestasis, hepatic encephalopathy, hepatitis, and jaundice), unexplained/persistent elevations of serum transaminases, pregnancy, breast-feeding, concomitant use with strong CYP3A4 inhibitors (e.g., erythromycin, clarithromycin, itraconazole, ketoconazole, posaconazole, voriconazole, HIV protease inhibitors, nefazodone, cobicistat), cyclosporine, danazol, gemfibrozil Caution in the following: heavy alcohol use (alcoholism), history of liver disease/renal impairment, uncontrolled hypothyroidism, patientsof Chinese descent, sepsis or severe infection, uncontrolled seizure disorder, recent stroke or transient ischemic attack, electrolyte imbalance, preexisting amyotrophic lateral sclerosis (ALS), the elderly

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The cost scale $-$$$$$ represents the relative cost of acquisition of medication only. Frequency and complexity of medication administration (institution workload, effect on adherence) should be considered when determining overall cost-effectiveness of treatment.

Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
HMG CoA Reductase Inhibitors (Statins) - Risk of skeletal muscle effects (e.g., myopathy, rhabdomyolysis) increase with higher doses and concomitant use of certain drugs. Predisposing factors include: age >65, female, uncontrolled hypothyroidism, and renal impairment			
Fluvastatin Lescol®, Lescol® XL Capsule, immediate-release (IR): 20 mg, 40 mg Tablet, extended-release (XL): 80 mg $$$-$$$$	Usual dose: 20-40 mg orally once daily in the evening, titrated up to 40 mg orally twice daily MODERATE-INTENSITY 30% to < 50% reduction in LDL: 40 mg (IR) orally twice daily or 80 mg (XL) orally once daily Dose adjustments: Max dose 20 mg orally twice daily: concomitant use with cyclosporine, fluconazole Renal Impairment: Mild-moderate impairment: no adjustment; Severe impairment: doses > 40 mg/day have not been evaluatedHD/PD: not defined Hepatic Impairment: Contraindicated in active liver disease or unexplained/persistent transaminase elevations	Adverse Reactions: myopathy, rhabdomyolysis, elevated liver enzymes, arthralgia, headache, atrial fibrillation, HTN, nausea, dyspepsia, fatigue, myalgia, sinusitis, pharyngitis, peripheral edema, rash, syncope, urinary tract infection Drug interactions: • Fluvastatin may be less likely to interact with other drugs • Use caution with niacin, fibrates (avoid gemfibrozil), glyburide, phenytoin, warfarin, colchicine • Consider temporarily suspending use while patient on daptomycin	Contraindications: hypersensitivity to fluvastatin or any component of the formulation, active liver disease (including cholestasis, hepatic encephalopathy, hepatitis, and jaundice), unexplained/persistent elevation serum transaminases, pregnancy, breast-feeding Caution in the following: heavy alcohol use (alcoholism), history of liver disease/renal impairment, uncontrolled hypothyroidism, sepsis or severe infection, uncontrolled seizure disorder, recent stroke or transient ischemic attack, electrolyte imbalance, preexisting amyotrophic lateral sclerosis (ALS), the elderly

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
HMG CoA Reductase Inhibitors (Statins) - Risk of skeletal muscle effects (e.g., myopathy, rhabdomyolysis) increase with higher doses and concomitant use of certain drugs. Predisposing factors include: age >65, female, uncontrolled hypothyroidism, and renal impairment			
Pitavastatin Livalo® Tablet: 1 mg, 2 mg, 4 mg $$$$$	Usual dose: 1-4 mg orally once daily MODERATE-INTENSITY 30% to < 50% reduction in LDL: 2-4 mg orally once daily in the evening Dose adjustments: Max dose 1 mg/day: concomitant use with erythromycin Max dose 2 mg/day: concomitant use with rifampin Renal Impairment: CrCl < 60 mL/min or HD: start 1 mg orally daily, max 2 mg/day Hepatic Impairment: Contraindicated in active liver disease or unexplained/persistent transaminase elevations	Adverse Reactions: myopathy, rhabdomyolysis, elevated liver enzymes, headache, nausea, dyspepsia, constipation, diarrhea, backache, myalgia, limb pain, arthralgia, rash, dizziness, memory impairment, peripheral neuropathy Drug interactions: • Pitavastatin may be less likely to interact with other drugs • Contraindicated with cyclosporine • Use caution with fibrates (avoid gemfibrozil), niacin, colchicine • Consider dosage reduction with niacin • Consider temporarily suspending use while patient on daptomycin	Contraindications: hypersensitivity to pitavastatin or any component of the formulation, active liver disease (including cholestasis, hepatic encephalopathy, hepatitis, and jaundice), unexplained/persistent elevation serum transaminases, pregnancy, breast-feeding, concomitant use with cyclosporine Caution in the following: heavy alcohol use (alcoholism), history of liver disease/renal impairment, uncontrolled hypothyroidism, sepsis or severe infection, uncontrolled seizure disorder, recent stroke or transient ischemic attack, electrolyte imbalance, the elderly

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Ezetimibe use may be considered in patients who do not meet cholesterol treatment goals with dietary modification and maximally tolerated statin therapy. 2018 Guideline on the Management of Blood Cholesterol from the ACC/American Heart Association Task Force found no proof that adding a non-statin to a statin prevents ASCVD events in patients without clinical ASCVD.			
Ezetimibe Zetia® Table: 10 mg $	Dose: 10 mg orally once daily Renal Impairment: No dose adjustment needed. Hepatic Impairment: Mild hepatic impairment: No dose adjustment needed Moderate to severe hepatic impairment: Not recommended	Adverse Reactions: diarrhea, abdominal pain, myalgia, arthralgia, back pain, fatigue, cough, sinusitis, pharyngitis, upper respiratory infection, hepatitis, elevated hepatic enzymes, rhabdomyolysis, anaphylaxis, pruritus Drug interactions: • Use caution with fibrates (avoid gemfibrozil), cyclosporine, warfarin, antacids, bile acid sequestrants	Contraindications: hypersensitivity to ezetimibe or any component of the formulation, concomitant use with statin in patients with active hepatic disease or unexplained persistent elevations in serum transaminases; pregnancy and breastfeeding (when used with a statin) Caution in the following: hepatic impairment (use not recommended in moderate or severe impairment), renal impairment, concomitant use with statins, concomitant use with fibrates, pregnancy, breast-feeding, the elderly Use Restrictions: For the addition of Ezetimibe see "Memorandum—Ezetimibe" located on Pharmacy Lifeline page under Memos tab. Pharmacy Lifeline

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Bile Acid Sequestrants:			
Cholestyramine QUESTRAN® Powder: 4 g $$$	Initial dose as an adjunct to diet with maximally tolerated statin for additional LDL-lowering therapy in patients who are ezetimibe-intolerant and triglycerides < 300: 4 g orally 1 to 2 times daily before meals (mixed in 60-180 ml of noncarbonated beverage) Maintenance dose: 8-16 g/day in 2 divided doses before meals Max dose: 24 g/day Renal Impairment: No adjustment needed Hepatic Impairment: No adjustment needed	Adverse Reactions: constipation, abdominal discomfort, flatulence, nausea, vomiting, dental caries, anxiety, dizziness, drowsiness, fatigue, headache, osteoporosis Drug interactions: • Decreases absorption of other drugs • Administer other drugs at least 1 hour before or at least 4-6 hours after each dose	NONFORMULARY Contraindications: Complete biliary obstruction (bile is not secreted into the intestine), patients with TG > 400 mg/dl, GI obstruction, hypersensitivity to cholestyramine or any component of the product Caution in the following: primary biliary cirrhosis, hypertriglyceridemia, renal impairment, patients susceptible to fat-soluble vitamin deficiencies, constipation, coagulopathy, dysphagia, cholelithiasis, hemorrhoids, pregnancy, breast-feeding, the elderly

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) monoclonal antibody - Recommended as third-line therapy in addition to maximally tolerated statin therapy and ezetimibe in patients with clinical ASCVD and comorbidities that still require greater LDL reduction. The addition of PCSK9 mAb may be considered for patients that meet the non-formulary criteria[1] upon the recommendation of a specialist (cardiologist or endocrinologist).			
PCSK9 mAb Alirocumab Praluent® Solution for Injection, Prefilled pen device: 75 mg/mL 150 mg/mL $$$$$ **Non-Formulary Preferred** **USE RESTRICTIONS APPLY– SEE COMMENTS**	Usual dose: Treatment of primary hyperlipidemia, including heterozygous familial hypercholesterolemia, to lower LDL-C: 75 mg subcutaneously once every 2 weeks. If response is inadequate, increase to 150 mg subcutaneously once every 2 weeks Treatment of patients with heterozygous familial hypercholesterolemia who are undergoing LDL apheresis: 150 mg subcutaneously every 2 weeks; administer without regard to timing of apheresis. For myocardial infarction prophylaxis, stroke prophylaxis, and to reduce the risk of unstable angina requiring hospitalization in patients with established cardiovascular disease: 75 mg subcutaneously once every 2 weeks. If response is inadequate, increase to 150 mg subcutaneously once every 2 weeks Note: Alirocumab is recommended as second-line therapy in addition to maximally tolerated statin therapy in patients with clinical ASCVD and comorbidities that still require 25% or greater LDL reduction. Factors to consider include cost, benefit of ASCVD risk reduction, dosing frequency requirements, and administration by subcutaneous injection Renal Impairment: Mild to moderate impairment: No dose adjustment needed Severe impairment: specific guidelines for dose adjustment not available Hepatic Impairment: Mild to moderate impairment: No dose adjustment needed Severe impairment: Specific guidelines for dose adjustment not available	Adverse Reactions: chest pain, pharyngitis, antibody formation, injection site reaction, cough, diarrhea, myalgia, elevated hepatic enzymes, influenza, sinusitis, allergic reaction, angioedema,anaphylaxis Drug interactions: • No known significant interactions	Contraindications: hypersensitivity to alirocumab or any component of the formulation Caution in the following: pregnancy, breast-feeding Non-Formulary Use Criteria: Addition of a PCSK9 mAb may be considered in the following upon the recommendation of a specialist (cardiologist or endocrinologist): In patients with very high-risk ASCVD (history of multiple major ASCVD events or 1 major ASCVD event and multiple high-risk conditions) and LDL-C level remains ≥ 70 mg/dL on maximally tolerated statin PLUS ezetimibe therapy. In patients with severe primary hypercholesterolemia (LDL-C level ≥190 mg/dL) and the LDL-C level remains ≥100 mg/ dL on high-intensity or maximally tolerated statin PLUS ezetimibe AND the patient has multiple factors that increase subsequent risk of ASCVD events. In patients 30 to 75 years of age with heterozygous FH and with an LDL-C level of 100 mg/dL or higher while taking maximally tolerated statin PLUS ezetimibe therapy. In patients 40 to 75 years of age with a baseline LDL-C level of 220 mg/dL or higher who achieve an on-treatment LDL-C level of 130 mg/dL or higher while receiving maximally tolerated statin PLUS ezetimibe therapy.

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) monoclonal antibody - Recommended as third-line therapy in addition to maximally tolerated statin therapy and ezetimibe in patients with clinical ASCVD and comorbidities that still require greater LDL reduction. The addition of PCSK9 mAb may be considered for patients that meet the non-formulary criteria[1] upon the recommendation of a specialist (cardiologist or endocrinologist).			
PCSK9 mAb Evolocumab Repatha® Solution for Injection, Prefilled syringe: 140 mg/mL Prefilled pen device: 140 mg/mL $$$$$ **USE RESTRICTIONS APPLY– SEE COMMENTS**	Usual dose: Treatment of primary hypercholesterolemia (including heterozygous familial hypercholesterolemia): 140 mg subcutaneously every 2 weeks or 420 mg subcutaneously once monthly Adjunct to diet and other LDL-C lowering therapies for the treatment of homozygous familial hypercholesterolemia in patients who require additional lowering of LDL-C: 420 mg subcutaneously once monthly Myocardial infarction prophylaxis, stroke prophylaxis, and to reduce the risk of coronary revascularization in patients with established cardiovascular disease: 140 mg subcutaneously every 2 weeks or 420 mg subcutaneously once monthly Note: Evolocumab is recommended as second-line therapy in addition to maximally tolerated statin therapy in patients with clinical ASCVD and comorbidities that still require 25% or greater LDL reduction. Factors to consider include cost, benefit of ASCVD risk reduction, dosing frequency requirements, and administration by subcutaneous injection Renal Impairment: Mild to moderate impairment: No dose adjustment needed Severe impairment: specific guidelines for dose adjustment not available Hepatic Impairment: Mild to moderate impairment: No dose adjustment needed Severe impairment: specific guidelines for dose adjustment not available	Adverse Reactions: back pain, pharyngitis, antibody formation, injection site reaction, diarrhea, myalgia, arthralgia, cough, dizziness, HTN, headache, hyperglycemia, influenza, sinusitis, allergic reaction, angioedema, anaphylaxis Drug interactions: • No known significant interactions	Contraindications: hypersensitivity to evolocumab or any component of the formulation Caution in the following: latex hypersensitivity, pregnancy, breast-feeding Non-Formulary Use Criteria: Addition of a PCSK9 mAb may be considered in the following up on the recommendation of a specialist (cardiologist or endocrinologist): • In patients with very high-risk ASCVD (history of multiple major ASCVD events or 1 major ASCVD event and multiple high-risk conditions) and LDL-C level remains ≥ 70 mg/dL on maximally tolerated statin PLUS ezetimibe therapy • In patients with severe primary hypercholesterolemia (LDL-C level ≥190 mg/dL) and the LDL-C level remains ≥100 mg/dL on high-intensity or maximally tolerated statin PLUS ezetimibe AND the patient has multiple factors that increase subsequent risk of ASCVD events • In patients 30 to 75 years of age with heterozygous FH and with an LDL-C level of 100 mg/dL or higher while taking maximally tolerated statin PLUS ezetimibe • In patients 40 to 75 years of age with a baseline LDL-C level of 220 mg/dL or higher who achieve an on-treatment LDL-C level of 130 mg/dL or higher while receiving maximally tolerated statin PLUS ezetimibe.

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Small interfering ribonucleic acid (siRNA) – In addition to maximally tolerated statin therapy and ezetimibe in patients with clinical ASCVD and comorbidities that still require greater LDL reduction. The addition of the siRNA, inclisiran, may be considered for patients that meet the non-formulary criteria[1] upon the recommendation of a specialist (cardiologist or endocrinologist).			
siRNA Inclisiran Leqvio® Solution for Injection, Prefilled syringe: 284 mg/1.5 mL Prefilled pen device: 284 mg/1.5 mL $$$$$ **USE RESTRICTIONS APPLY– SEE COMMENTS**	Usual dose: Treatment of primary hypercholesterolemia (including heterozygous familial hypercholesterolemia) as adjunctive therapy to diet and maximally-tolerated statin therapy: 284 mg as a single injection, again at 3 months, and then every 6 months thereafter Myocardial infarction prophylaxis, stroke prophylaxis, and to reduce the risk of coronary revascularization in patients with established cardiovascular disease: 284 mg as a single injection, again at 3 months, and then every 6 months thereafter Factors to consider include cost, benefit of ASCVD risk reduction, dosing frequency requirements, and administration by subcutaneous injection Renal Impairment: Mild to moderate impairment: No dose adjustment needed Severe impairment: there are no dosage adjustments provided in the manufacturer's labeling (has not been studied) Hepatic Impairment: Mild to moderate impairment: No dose adjustment needed Severe impairment: there are no dosage adjustments provided in the manufacturer's labeling (has not been studied)	Adverse Reactions: antibody development, injection site reactions, arthralgia, bronchitis, urinary tract infection, diarrhea, dyspnea, pain in extremities Drug interactions: • No known significant interactions	Contraindications: hypersensitivity to inclisiran or any component of the formulation Caution in the following: pregnancy, breast-feeding Non-Formulary Use Criteria: Addition of inclisiran may be considered in addition to statin and ezetimibe therapy and in lieu of PCSK9 mAb upon the recommendation of a specialist (cardiologist or endocrinologist). According to the 2022 ECDP, PCSK9 mAb is preferred as the initial PCSK9 inhibitor of choice in view of its demonstrated safety, efficacy, and benefits for cardiovascular outcomes. Inclisiran may be considered in lieu of PCSK9 mAb in patients with demonstrated poor adherence to PCSK9 mAbs or in patients with adverse effects from both PCSK9 mAbs. There is currently no evidence or mechanistic plausibility for additional efficacy in LDL-C lowering or cardiovascular outcomes benefit for combination therapy of PCSK9 mAb and inclisiran when added to maximally tolerated statin therapy with or without ezetimibe. Therefore, if inclisiran is prescribed, it should be in place of a PCSK9 mAb and recommended by a specialist, like cardiologist or endocrinologist.

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Adenosine triphosphate-citrate lyase (ACL) inhibitor - May be considered for patients upon the recommendation of a specialist (cardiologist or endocrinologist) in addition to maximally tolerated statin therapy and ezetimibe or in patients who do not tolerate statin therapy (after trying 2-3 different statins) due to statin-associated muscle symptoms.			
Bempedoic acid (Nexletol®) Tablet: Nexletol® 180 mg $$$$ **USE RESTRICTIONS APPLY– SEE COMMENTS**	Clinical ASCVD not at goal on maximally tolerated LDL-C lowering therapy: Consider for use in patients who do not meet cholesterol treatment goals with dietary modification and other lipid-lowering therapies (e.g., maximally tolerated statin plus ezetimibe and/or a PCSK9 mAb) Nexletol® 180 mg orally once daily Renal Impairment: Nexletol®: CrCl < 30 mL/min: There are no dosage adjustments provided in the manufacturer's labeling (limited experience) ESRD on dialysis: There are no dosage adjustments provided in the manufacturer's labeling (limited experience) Hepatic Impairment: There are no dosage adjustments provided in the manufacturer's labeling (has not been studied) for severe impairment (Child-Pugh class C)	Adverse Reactions: elevated hepatic enzymes, abdominal pain, increased serum creatinine, back pain, limb pain, muscle spasm, gout, hyperuricemia, tendon rupture, atrial fibrillation/flutter, increased total CK, thrombocytopenia, leukopenia Drug interactions: Use caution with pravastatin and simvastatin	Contraindications: No contraindications listed on manufacturer's labeling Caution in the following: pregnancy and breastfeeding, in which instances bempedoic acid should be discontinued
Bempedoic acid-ezetimibe (Nexlizet®) Tablet: Nexlizet® 180 mg/10 mg $$$$ **USE RESTRICTIONS APPLY– SEE COMMENTS**	Clinical ASCVD not at goal on maximally tolerated LDL-C lowering therapy: Consider for use in patients who do not meet cholesterol treatment goals with dietary modification and other lipid-lowering therapies (e.g., maximally tolerated statin plus ezetimibe and/or a PCSK9 mAb) Nexlizet® 180 mg/10 mg orally once daily Renal Impairment: Nexlizet®: CrCl < 30 mL/min: There are no dosage adjustments provided in the manufacturer's labeling (limited experience) ESRD on dialysis: There are no dosage adjustments provided in the manufacturer's labeling (limited experience) Hepatic Impairment: Mild impairment (Child-Pugh class A) No dosage adjustment necessary. Moderate-to-severe impairment (Child-Pugh class B and C) Use is not recommended	Adverse Reactions: constipation, urinary tract infection, nasopharyngitis, elevated hepatic enzymes, abdominal pain, increased serum creatinine, back pain, limb pain, muscle spasm, gout, hyperuricemia, tendon rupture, atrial fibrillation/flutter, increased total CK, thrombocytopenia, leukopenia Drug interactions: Use caution with pravastatin, simvastatin, bile acid sequestrants, cyclosporin	Contraindications: Hypersensitivity (e.g., anaphylaxis, angioedema, rash, urticaria) to ezetimibe or any component of the formulation Caution in the following: pregnancy and breastfeeding, in which instances bempedoic acid-ezetimibe should be discontinued

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Fibric Acid Derivatives: Increases in CK, increased risk of rhabdomyolysis and myoglobinuria leading to acute renal failure are associated with concurrent use of fibrates and statins (significantly higher rate observed with gemfibrozil), particularly in the elderly, patients with diabetes, renal failure, or hypothyroidism			
Fenofibrate (Lofibra®, Tricor®) Tablet: Generic Lofibra® **54 mg, 160 mg** Generic Tricor® 48 mg, 145 mg, $$-$$$	Primary hypercholesterolemia or mixed dyslipidemia: Generic Lofibra®: • **160 mg** orally once daily Generic Tricor®: • 145 mg orally once daily Withdraw therapy in patients who do not have an adequate response after two months of treatment with the maximum recommended dose of **160 mg/**day (Generic Lofibra®); 145 mg/day (GenericTricor®) Severe hypertriglyceridemia: Generic Lofibra®: • **54-160 mg** orally once daily Generic Tricor®: • 48-145 mg orally once daily Consider dosage reduction if serum lipid concentrations fall significantly below target goals. Max dose: Generic Lofibra: • **160 mg/**day Generic Tricor®: • 145 mg/day Renal Impairment: CrCl 30-80 mL/min: Initially Generic Lofibra®: • **54 mg** orally once daily Generic Tricor®: • 48 mg orally once daily Do not increase dose until effects of the initial dose on renal function and serum lipid concentrations have been fully evaluated. CrCl < 30 mL/min: Contraindicated Hepatic Impairment: Contraindicated in hepatic dysfunction, including primary biliary cirrhosis or unexplained, persistent liver function abnormality	Adverse Reactions: elevated hepatic enzymes, abdominal pain, nausea, constipation, diarrhea, asthenia, dyspepsia, gallstones, myopathy, backache, rhinitis, urticarial rash, pulmonary embolism, thrombosis Drug interactions: • Use caution with warfarin, statins, bile acid sequestrants, immunosuppressants (e.g., cyclosporine, tacrolimus), colchicine	FENOFIBRATE AND KIDNEYS • Renal status should be evaluated before fenofibrate initiation, within 3 months after initiation, and every 6 months thereafter • Assess renal safety with both serum creatinine level and estimated GFR based on creatinine Contraindications: hypersensitivity to fenofibrate, fenofibric acid or any component of the formulation, severe renal dysfunction (CrCl <30 ml/min) including patients receiving dialysis, hepatic disease including primary biliary cirrhosis or unexplained persistent liver function abnormalities, gallbladder disease, breast-feeding Caution in the following: diabetes, hypothyroidism, mild to moderate renal impairment, thromboembolic disease, pregnancy, the elderly

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Treatment Cont'd

MEDICATION	DOSING	ADVERSE EFFECTS DRUG INTERACTIONS	COMMENTS
Prescription Omega-3 Polyunsaturated Fatty Acid (PUFA) - Recommended as therapy for persistent hypertriglyceridemia. Nonprescription fish oil products, or marine-derived omega-3 fatty acid preparations, are classified as dietary supplements and are not interchangeable with prescription omega-3 PUFAs.			
Omega-3 ethyl esters Lovaza® Tablet: Generic Lovaza® 1 g $$$ **USE RESTRICTIONS APPLY– SEE COMMENTS**	Usual dose: Treatment of persistent fasting hypertriglyceridemia: 4 g orally once daily with meals or 2 g orally BID with meals Severe hypertriglyceridemia: 4 g orally once daily with meals or 2 g orally BID with meals Renal Impairment: No dose adjustment provided in the manufacturer's labeling (has not been studied). Not renally eliminated Hepatic Impairment: No dose adjustment provided in the manufacturer's labeling (has not been studied).	Adverse Reactions: atrial fibrillation/flutter, dysgeusia, dyspepsia, eructation, dyspepsia, vomiting, pruritus, skin rash, urticaria, anaphylaxis, bleeding tendency disorder, increased LDL cholesterol, transaminitis Drug interactions: • Agents with antiplatelet properties (e.g., P2Y12 inhibitors, NSAIDs, SSRIs, etc.): Omega-3 fatty acids may enhance the antiplatelet effect of these agents • Anticoagulants: Omega-3 fatty acids may enhance anticoagulant effects • Ibrutinib: Omega-3 fatty acids may enhance the antiplatelet effects	Contraindications: hypersensitivity to omega-3 fatty acids or any component of formulation Caution in the following: bleeding risk, fish allergy, hypersensitivity, hepatic impairment, excessive alcohol intake, breastfeeding Non-Formulary Use Criteria: Reserved for patients who fail or are intolerant to fibrate/statin combination after a six-month trial
Icosapent ethyl Vascepa® Tablet: Vascepa® 1 g $$$$ **USE RESTRICTIONS APPLY– SEE COMMENTS**	Usual dose: Treatment of persistent fasting hypertriglyceridemia: 2 g orally BID with meals Severe hypertriglyceridemia: 2 g orally BID with meals Renal Impairment: No dose adjustment provided in the manufacturer's labeling (has not been studied). Eicosapentaenoic acid is not renally eliminated. Hepatic Impairment: No dose adjustment provided in the manufacturer's labeling (has not been studied).	Adverse Reactions: hemorrhage including major hemorrhage, atrial fibrillation/flutter, peripheral edema, gout, musculoskeletal pain, constipation Drug interactions: • Agents with antiplatelet properties (e.g., P2Y12 inhibitors, NSAIDs, SSRIs, etc.): Omega-3 fatty acids may enhance the antiplatelet effect these agents • Anticoagulants: Omega-3 fatty acids may enhance anticoagulant effects • Ibrutinib: Omega-3 fatty acids may enhance the antiplatelet effects	Contraindications: hypersensitivity to icosapent ethyl or any component of formulation Caution in the following: bleeding risk, atrial fibrillation/flutter, fish allergy, hypersensitivity, diabetes, hypothyroidism, hepatic impairment, excessive alcohol intake, breastfeeding Non-Formulary Use Criteria: Reserved for patients who fail or are intolerant to fibrate/statin combination after a six-month trial

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MONITORING

Starting at 20 years of age, it is reasonable to assess traditional ASCVD risk factors every 4 to 6 years with either a fasting or a nonfasting lipid panel to document baseline LDL-C for patients who have low ASCVD risk. For patients with elevated risk who decide not to start lipid-lowering therapy, it is reasonable to reassess ASCVD risk factors and recheck a nonfasting lipid panel annually. If the nonfasting lipid panel reveals triglycerides of 400 mg/dL or higher, do a fasting lipid panel for assessment of fasting triglyceride levels and baseline LDL-C. For adults with LDL-C < 70 mg/dL, measurement of direct LDL-C or modified LDL-C estimate may be ordered for improved accuracy. Fasting and nonfasting TC and HDL-C levels appear to have fairly similar prognostic value and associations with CVD outcomes.

Two lipoprotein entities related to LDL-C are apoB and lipoprotein (a) [Lp(a)]. Under certain circumstances, particularly in patients with hypertriglyceridemia (persistent fasting TG ≥ 200 mg/dL), measurement of apoB may have advantages. ApoB level > 130 mg/dL corresponds to LDL-C ≥ 160 mg/dL and constitutes a risk-enhancing factor.

Lp(a) is a modified form of LDL. Relative indications to measure Lp(a) are family history of premature ASCVD or personal history of ASCVD non-explained by major risk factors. Lp(a) level > 50 mg/dL constitutes a risk-enhancing factor.

Once lipid-lowering drug therapy has started, check patient's fasting lipids:

- 1-3 months after starting treatment
- 1-3 months after adjustment of treatment until within the threshold range.

Once a patient has reached the appropriate threshold or optimal lipid level:

- Check fasting lipids every 12 months, unless there are adherence problems or other reasons for more frequent testing, such as efficacy checks, changes in therapy, etc.
    - Discontinuation/non-adherence remains a major gap in the primary/secondary prevention of ASCVD
    - The major reason for statin discontinuation is because of the development of SAMS. See below.

Statin-Associated Side Effects: Adverse reactions occur less frequently with statins than with most other classes of lipid-lowering agents. However, muscle events remain important side effects. Total CK and transaminase levels should NOT be routinely measured given the unlikely impact on clinical outcomes and the lack of established cost effectiveness. Instead of the label *statin intolerance*, the present AHA guideline prefers *statin-associated side effects* because the large majority of patients are able to tolerate statin rechallenge with an alternative statin or alternative regimen, such as reduced dose or in combination with non-statins. Consider referring to a lipid specialist for patients with statin-associated side effects to at least 2 statin therapies and 1 attempt at the lowest FDA-approved dose and a trial of a non-statin LDL-lowering medication.

Statin Associated Muscle Symptoms (SAMS)
The most frequent SASE, SAMS are usually subjective myalgia, reported observationally in 5% to 20% of patients. SAMS often result in nonadherence and can adversely impact ASCVD outcomes. An elevated creatine kinase (CK) level is not required for establishing the diagnosis, although SAMS may be corroborated by CK ≥ 4 x the upper limit of normal (ULN).

Factors that may increase the risk for statin-induced myopathy include:

- Female sex
- Small body size
- Age > 65 years
- Frailty
- Asian ancestry
- Personal or family history of myopathy
- Poorly controlled hypothyroidism
- Subnormal vitamin D
- Use of medications that raise circulating levels of statins and/or their active metabolites (e.g., erythromycin, fluconazole, amlodipine, etc.)

Muscle adverse events remain important side effects usually manifested by muscle discomfort, including muscleaches, soreness, stiffness, and weakness**.** For symptomatic cases, current guidelines recommend checking total CK.

Myalgia: Measure total creatinine kinase levels (total CK) in patients with statin-associated muscle symptoms and/or objective muscle weakness. If total CK remains normal, statin therapy may continue if the symptoms are acceptable to the patient. Otherwise, the patient can temporarily discontinue the statin to reassess for any improvement with muscle symptom. Then the patient can rechallenge to achieve maximal LDL-C lowering by a modified dosing regimen on the same statin, a reduced dose of statin in combination with non-statin therapy, or an alternative statin.

Monitoring Cont'd

Myositis/Myopathy: Statin treatment should be discontinued immediately. Monitor total CK levels weekly and seek specialist advice if:

- There is a moderate rise in the CK level (i.e., 3-10 x upper limit of normal)
- An elevated CK level is found (i.e., CK > 10 x upper limit of normal), or where myopathy is suspected or diagnosed

Myonecrosis with myoglobinuria (rhabdomyolysis): Stop statin immediately and refer to specialist.

Statin-Associated Muscle Symptoms		
Myalgias	CK normal	Musculoskeletal manifestations are well-recognized side effects of treatment with statins. These side effects are reported to occur in about 10% of people prescribed statins.
Myopathy	CK > 10x ULN	Characterized most of the time by mild to moderate hypercreatinemia. Rare incidence.
Rhabodomyolysis	CK > 40x ULN	Defined by the Task Force as myonecrosis, which can cause myoglobinuria and acute renal failure. Patients with symptomatic or asymptomatic rhabdomyolysis from a statin should discontinue therapy immediately.
Statin-Induced Necrotizing Autoimmune Myopathy (SINAM)	CK > 40x ULM Positive anti-HMGCR antibody	Proximal muscle weakness, which can cause myalgia, dysphagia, and/or respiratory failure. Persistent/progressive symptoms even with discontinuation of statin. Treated with immunosuppression. Rare incidence but potentially life-threatening.

Hepatic dysfunction: Measure transaminases (AST, ALT), total bilirubin, and alkaline phosphatase if there are symptoms suggesting hepatotoxicity. In patients with chronic, stable liver disease, including non-alcoholic fatty liver disease (NAFLD), check a baseline measurement of transaminases (AST, ALT), total bilirubin, and alkaline phosphatase, then schedule monitoring labs. An asymptomatic increase in transaminases more than 3 times the upper limit of normal is an infrequent statin-associated side effect that is often resolved with dose reduction or rechallenge with an alternative statin. Borderline elevations are almost always benign (LFTs < 2x ULN). Severe statin-associated hepatotoxicity is rare and not impacted by routine monitoring of transaminases. Stop statin if signs or symptoms of liver disease occur or LFTs >3x ULN. Measure liver function and monitor for select patients. Transaminase levels should NOT be routinely measured given the unlikely impact on clinical outcomes and the lack of established cost effectiveness.

Type 2 DM: Patients at increased risk of type 2 DM are recommended to continue statin therapy with added emphasis given to lifestyle changes.

Behavioral and cognitive: Concerns have been raised about increased suicide in patients treated with some lipid-lowering therapies, but statins do not appear to be associated with an increased risk of suicide or depression. Some reports indicate a possible correlation between statins and short-term memory loss. Cognitive impairment is usually reversible upon discontinuation of statin. In adults 75 years of age or older, it may be reasonable to stop statin therapy when cognitive functional decline limits the potential benefits of statin therapy.

Patient Adherence: Despite the well-documented benefits from statins, patient adherence to therapy is frequently challenged by adverse effects and it is important to discuss these with each patient prior to prescribing (see PE-3 for details). The clinician should reassess, rediscuss, and encourage rechallenge as the initial approach unless side effects are severe. Ongoing communication is integral to patient care, as is regular monitoring to check for adherence, adequacy of response, new associated symptoms, and reaffirmation of benefit.

- In such cases, consider using smaller statin doses and/or less potent statins with lower incidence of myopathy, along with cautious up-titration of dose.

Some lipid specialists prescribe fluvastatin or pitavastatin in patients intolerant to other members of the HMG-CoA reductase inhibitor class.

APPENDIX A: GENETIC CAUSES OF DYSLIPIDEMIA

The terminology surrounding the hereditary disorders of LDL-C metabolism can be confusing. Individual patients may have one (monogenetic) or more (polygenetic) genetic defects that lead to a particular phenotype.

Below is a brief summary of some of the more common genetic causes, see references for more information.

Genetic Cause	Description
Familial Combined Hyperlipidemia (FCHL)	FCHL is a common genetic lipid disorder (1 to 2 percent of the population). Patients present with elevated levels of plasma total cholesterol, LDL-C, triglycerides, and apoB. Premature atherosclerotic cardiovascular disease (ASCVD) is not uncommon. When not initially present, there is an increased future likelihood of impaired glucose tolerance or type 2 diabetes. • **Clinical manifestations** include premature CHD (particularly in patients with concurrent hypertriglyceridemia), xanthelasma (in 10 percent of cases), and obesity • **Diagnosed** in patients with a family history of premature CHD and whose apoB concentration is > 120 mg/dL in combination with either elevations in both LDL-C and triglycerides or either elevated LDL-Col or triglycerides • **Treatment** of FCHL (first line) is a statin, irrespective of whether or not triglyceride levels are elevated, which is effective therapy for lowering apoB levels. The potency and dose of statin should be chosen with the aim of achieving LDL-C < 100 mg/dL in primary prevention and < 70 mg/dL in secondary prevention. If the LDL-C or non-HDL-C therapeutic goals are not reached, addition of ezetimibe can be considered.

Familial Hypertriglyceridemia	Familial Hypertriglyceridemia is a disorder characterized by the overproduction of V-LDL from the liver. As a result, the patient will have an excessive number of triglycerides and V-LDL on the lipid profile.
	
	• **Clinical manifestations**: Mild to moderate elevations in triglyceride concentration. familial hypertriglyceridemia is typically accompanied by other co-morbidities: obesity, hyperglycemia, and HTN. Patients with this disorder are often heterozygous for inactivating mutations of the lipoprotein lipase (LPL) gene. While this mutation can alone raise triglyceride levels significantly, the combination of other medications or pathology can further increase serum triglyceride to pathologic levels. Significant increases in triglyceride levels can lead to the development of clinical signs and acute pancreatitis. A common sign of severe triglyceride elevation is xanthoma formation on skin exams.
	  ◦ Typically, familial hypertriglyceridemia is differentiated with significantly high triglycerides and low HDL-C levels in comparison to others in the Fredrickson, Levy and Lees (FLL) phenotypes.
	  ◦ However, the influence of comorbid conditions that often accompany the pathology can skew a patient's lipid profile.
	• **Diagnosed** with routine fasting LP in the appropriate individuals. If triglyceride abnormalities are still present in the absence of secondary causes, further investigation could be suggested to screen first degree relatives due to the high prevalence of familial causes.
	• **Treatment** for familial hypertriglyceridemia focuses on reducing the triglyceride levels. Furthermore, if the patient has pathologic triglyceride levels, treating or removing secondary causes would be vital to maintain relatively normal lipid levels. See Appendix C.

- UpToDate: Inherited disorders of LDL-cholesterol metabolism other than familial hypercholesterolemia
- NCBI: Familial Hypertriglyceridemia

APPENDIX B: COMPONENTS OF A LIPID PANEL

Diagnosis of dyslipidemia is made by measuring plasma levels of total cholesterol, triglycerides (TG), and individual lipoproteins. Key components of a standard lipid panel include the following:

Component	Details
Cholesterol/HDL-C ratio (calculated)	To calculate ratio, divide total cholesterol number by HDL-C (e.g., if total cholesterol is 200 mg/dL and HDL is 50 mg/dL the ratio would be 4-to-1). Higher ratios mean a higher risk of heart disease.
HDL-C	HDL cholesterol is known as the "good" cholesterol because it helps remove other forms of cholesterol from the bloodstream. Higher levels of HDL cholesterol are associated with a lower risk of heart disease.
LDL-C (calculated)	The equation calculates LDL-C as total cholesterol minus HDL-C minus TGs divided by five. The result is expressed in milligrams per deciliter. The direct LDL-C test measures the amount of LDL cholesterol, sometimes called "bad" cholesterol, in the blood. Calculated LDL-C is about as accurate as direct LDL-C when TG levels are normal.
Non-HDL-C (calculated)	Non-HDL-C is calculated as total cholesterol minus HDL cholesterol. According to the European Society of Cardiology cholesterol guideline, non-HDL-C evaluation is recommended for risk assessment, particularly in people with high TG levels, diabetes, obesity, or very low LDL-C levels.
Non-HDL cholesterol (non-HDL-C)	Represents the cholesterol components carried by atherogenic lipoproteins such as LDL, very low-density lipoprotein (VLDL) and intermediate density lipoprotein (IDL). Higher non-HDL-C levels indicate increased risk of atherosclerosis. Non-HDL-C is calculated as total cholesterol minus HDL cholesterol[2]
Total cholesterol	Total cholesterol is a measure of the total amount of cholesterol in the blood. It includes LDL cholesterol, VLDL, and HDL cholesterol.
Triglyceride	TGs (excess calories are transformed into TGs) are a type of fat (lipid) found in the blood.

APPENDIX C: EVALUATING AND TREATING HYPERTRIGLYCERIDEMIA

Elevated fasting plasma TG levels are associated with ASCVD burden and events such as myocardial infarction and stroke. The risk of ASCVD events begins to rise with TG level above 150 mg/dL (1.7 mmol/L). Higher TG levels also increase the risk of pancreatitis.

This section will assist clinicians in recognizing and treating patients with hypertriglyceridemia to reduce ASCVD risk, as well as reduce the risk of pancreatitis.[14]

Risk Factors for Hypertriglyceridemia

- Men ≥ 55y, Women ≥ 65y
- Current/Recent smoker
- HTN
- HDL ≤ 40 mg/dL for men, ≤ 50 mg/dL for women
- Hs-CRP > 3 mg/L
- Renal dysfunction, CrCl 30-60 ml/min
- Retinopathy
- Micro- or Macro-albuminuria
- ABI < 0.9 without claudication

Diagnosing Hypertriglyceridemia

ACC/AHA guidelines recognize two categories of hypertriglyceridemia:

- Moderate hypertriglyceridemia is defined as TG (fasting or non-fasting) 175-499 mg/dl
- Severe hypertriglyceridemia is defined as fasting TG ≥ 500 mg/dl (at risk for pancreatitis, patients should be evaluated for genetic disorder of lipid metabolism).

Secondary Factors for Hypertriglyceridemia

The table below lists lifestyle, secondary disorders, and medications that influence hypertriglyceridemia.

LIFESTYLE	Obesity	Metabolic syndrome	
2° DISORDERS	DM or Hypothyroidism	Chronic liver disease	CKD and/or nephrotic syndrome
MEDICATIONS	Hormone related: • Oral estrogens • Tamoxifen • Raloxifene • Retinoids • Glucocorticoids	Immune related: • Cyclosporine • Tacrolimus • Sirolimus • Cyclophosphamide • Interferon	Other: • Beta blockers • Thiazides • Atypical antipsychotics • Rosiglitazone • Bile acid sequestrants

Treatment of Hypertriglyceridemia

The table below includes treatment recommendations and goals for moderate and severe hypertriglyceridemia.

Triglyceride Level		Goal	Intervention
Moderate Hypertriglyceridemia	**150-499 mg/dL**	Reduce VLDL (atherogenic) and ASCVD risk	• Lifestyle changes: especially appropriate diet composition, physical activity, weight reduction in overweight and obese • Evaluate all patients for secondary factors/causes • Statin recommended in adults aged 40-75 with moderate hypertriglyceridemia and an ASCVD risk of 7.5% or higher (proven to be of most benefit to reduce ASCVD risk)
Severe Hypertriglyceridemia	**≥ 500 mg/dL**	Reduce ASCVD risk (↑VLDL) Reduce pancreatitis risk (↑chylomicrons)	• Evaluate and address secondary factors/causes • Initiation of statins is recommended in patients with severehypertriglyceridemia and ASCVD risk equal to or greater than 7.5%. • Recommend very low fat diet. • Avoid refined carbohydrates and alcohol • Fenofibrate may be considered concomitantly with a low or moderate-intensity statin **only if** the benefits from ASCVD risk reduction or triglyceride lowering when benefits are judged to outweigh the potential risk for adverse effects (Renal status should be evaluated before fenofibrate initiation (see page 17). • Consider Omega-3 PUFAs as needed. Omega-3-PUFA (Lovaza®) is non-formulary with use criteria (Reserved for patients who fail or are intolerant to fibrate/statin combination after a six month trial).

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PATIENT EDUCATION/SELF-MANAGEMENT

Dyslipidemia (dis-lip-i-deem-ee-a)

What You Should Know

What is dyslipidemia? (Also called "high cholesterol")

- Your blood contains three main types of lipids (fat):lipids high-density lipoprotein, low-density lipoprotein, and triglycerides (tri-gli-cer-ides).
- Dyslipidemia is unhealthy levels of one or more kinds of lipids in your blood.

Why is treating dyslipidemia important?

- Dyslipidemia is a risk factor for heart attacks and strokes
- Treating dyslipidemia will help you avoid a heart attack or stroke

How can I tell if I have dyslipidemia?

- When you have dyslipidemia, you may not have symptoms
- Your health care provider will order a test that measures the amount of lipids in your blood

How do I know if I need the test?

Your health care provider will check your lipids/cholesterol if:

You have a history of:

- Previous heart attack
- Diabetes mellitus
- High blood pressure
- Cigarette smoking

You are:

- Overweight or obese
- Physically Inactive

You have:

- Family history of early heart disease

How is dyslipidemia treated?

Treatment depends on your:

- Lipid (cholesterol) levels
- Your risk of heart attack is based on other things like if you smoke, have high blood pressure, or diabetes
- General health
- Your primary care provider may give you a lipid-lowering medication to lower your cholesterol

What You Should Do

Change your daily routine and activities to lower your cholesterol:

- Lose weight if you are overweight
- Exercise
- Stop smoking
- Eat more fruits and vegetables
- Reduce fat in your diet (meat, milk, eggs, butter, cheese, packaged foods and snack items like cookies, crackers, and chips)
- Take any medications you are given as directed
- Report medication side effects
  - ➢ Muscle aches are commonly reported and may or may not be due to your medicine
- Get blood tests as recommended by your health care team

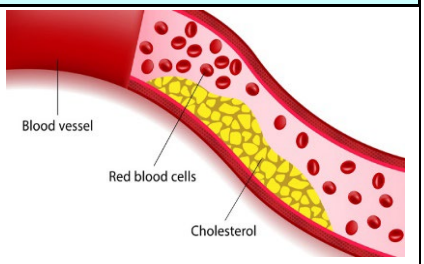
PATIENT EDUCATION/SELF MANAGEMENT

High Cholesterol– Dyslipidemia

What You Should Know

What is cholesterol? (kol-es-ter-ol)

- **Blood cholesterol** is waxy and fat-like and is made by your liver.
- Your body needs it to make hormones, Vitamin D, and for protecting nerves.
- Your body makes all the blood cholesterol it needs.
  - You should try to eat as little cholesterol in your food as possible while on a healthy eating plan.



- **Dietary cholesterol** is found in animal foods, including meat, seafood, poultry, eggs, and dairy products.

What is the difference between "good cholesterol" and "bad cholesterol"?

• "Good cholesterol" absorbs bad cholesterol and carries it back to the liver where it is flushed from the body. High levels of good cholesterol can lower your risk for heart disease and stroke.	High levels of "bad cholesterol" can build up on the walls of your blood vessels (called plaque) and raise your risk for heart disease and stroke.

How do I find out my cholesterol levels?

- Your health care team can do a simple blood test, called a "lipid profile," to measure your cholesterol levels.
- This will help your health care team determine your risk for heart disease or stroke.

How do I know my risk for high cholesterol?

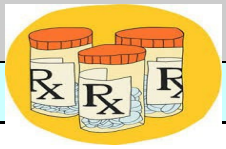
- Certain health conditions, your lifestyle, and your family history can raise your risk for high cholesterol.
- These are called "risk factors." These are listed below in detail.

Risk Factors for High Cholesterol

Health Conditions	• Type 2 Diabetes and obesity can raise cholesterol and blood fat levels.
Behaviors	Diet high in saturated fat and trans-fat (packaged foods, cookies, honey buns, etc.), not getting enough exercise, smoking, heavy drinking.
Family History	• Some people have a rare, genetic condition called familial hypercholesterolemia (FH). • This condition causes very high "bad cholesterol" levels beginning at a young age. If not treated, it will get worse with age. • If someone in your family had a heart attack early in life, talk to your health care team about your risk for FH. • If you have a family history of high cholesterol, you are more likely to have high cholesterol. You may need to get your cholesterol levels checked more often than people who do not have a family history of high cholesterol. • The risk for high cholesterol can increase even more when a family history of high cholesterol combines with unhealthy lifestyle choices, such as eating an unhealthy diet.

PATIENT EDUCATION/SELF MANAGEMENT

Statin Medications



What You Should Know

What are statins?

- Statin medications are usually the first type of drug that your health care provider will prescribe to lower bad cholesterol and triglycerides. They can also mildly raise your good cholesterol level.

What happens when I take this medication?

- You should have a blood test before starting treatment to check the level of cholesterol. It also checks if your liver is working properly.
- After starting treatment, you should have a blood test within 1-3 months and again at 12 months.
- The blood is checked to measure the cholesterol level to see how well the statin is working.

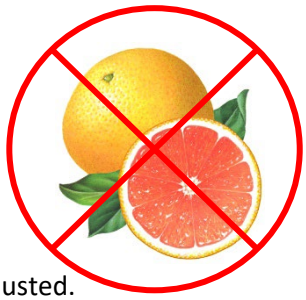
What are side effects of this medication?

- Most people who take a statin medication have no side effects, or only minor ones.
- Possible side effects include:
  - Headache
  - Muscle aches
  - Stomach pain
  - Bloating
  - Diarrhea
  - Feeling sick (nausea)
  - A rash
  - Pins and needles feeling
- Tell your doctor if you have any of these, especially muscle pains, tenderness, cramps, or weakness. This may happen as a rare side effect of statins and is a severe form of muscle inflammation.
- Muscle pains may be more likely if you are also taking a medicine called amlodipine or diltiazem.
- Your doctor may need to adjust your dose of statin to reduce the risk of muscle damage.

How do I know if I should not take this medication?

- You should not take a statin if you have active liver disease, if you are pregnant or intend to be pregnant, or if you are breastfeeding.
- You should stop a statin if you develop liver disease.

What should I avoid when taking this medication?



- Do not eat grapefruit or drink grapefruit juice if you are taking some statins.
- A chemical in grapefruit can increase the level of statin in the bloodstream, which can make side effects from the statin more likely. This is only a problem with simvastatin, atorvastatin and lovastatin. Other statins, such as pravastatin, do not interact with grapefruit.
- Medicines can sometimes interfere with statins. For example, some antibiotics and cyclosporin. The doses of either the statin or the other medicine may need to be adjusted.
- If you are prescribed (or buy) another medicine, remind the doctor or pharmacist that you are on a statin in case an interaction is likely.

EDUCACIÓN PARA EL PACIENTE/ CONTROL PERSONAL DEL CASO

Dislipidemia

Lo que debe saber

¿Qué es la dislipidemia? (También llamada "colesterol alto")

- La dislipidemia se refiere a niveles poco saludables de uno o más tipos de lípidos (grasas) en la sangre. Su sangre contiene tres tipos principales de lípidos: lipoproteínas de alta densidad (high-density lipoprotein, HDL), lipoproteínas de baja densidad (low-density lipoprotein, LDL) y triglicéridos.

¿Por qué es importante tratar la dislipidemia?

- La dislipidemia es un factor de riesgo para ataques cardíacos y derrames cerebrales.
- El tratamiento de la dislipidemia lo ayudará a evitar un ataque cardíaco o un derrame cerebral.

¿Cómo puedo saber si tengo dislipidemia?

- Cuando se tiene dislipidemia, es posible no presentar síntomas.
- Su proveedor de atención médica ordenará una prueba que mide la cantidad de lípidos en la sangre.

¿Cómo sé si necesito la prueba?

- Su proveedor de atención médica revisará sus lípidos/colesterol:

Si tiene antecedentes de:

- Ataque cardíaco previo
- Diabetes mellitus
- Hipertensión
- Tabaquismo

Si usted:

- Tiene sobrepeso o es obeso
- Es físicamente inactivo

Si tiene:

- Antecedentes familiares de una enfermedad cardíaca a temprana

¿Cómo se trata la dislipidemia?

El tratamiento depende de:

- Sus niveles de lípidos (colesterol)
- Su riesgo de sufrir ataques cardíacos con base en otros aspectos: si fuma, si tiene hipertensión o diabetes
- Salud general

Su proveedor de atención primaria puede proporcionarle un medicamento que reduce los lípidos para bajar su colesterol.

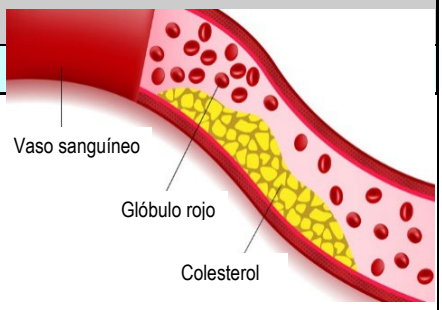
Lo que debe hacer

Cambie su rutina y actividades diarias para bajar su colesterol:

- Pierda peso si tiene sobrepeso
- Ejercítese
- Deje de fumar
- Coma más frutas y verduras
- Reduzca la grasa en su dieta (carne, leche, huevos, mantequilla, queso, alimentos envasados y meriendas como galletas dulces o saladas y papas fritas)
- Tome cualquier medicamento que se le proporcione según lo indicado
- Informe sobre cualquier efecto secundario de los medicamentos
  - ➢ Se reportan comúnmente dolores musculares y pueden deberse o no a su medicamento
- Realícese análisis de sangre según las recomendaciones de su equipo de atención médica

EDUCACIÓN PARA EL PACIENTE/ CONTROL PERSONAL DEL CASO

Colesterol alto – Dislipidemia



Lo que debe saber

¿Qué es el colesterol?

- **El colesterol en sangre** es ceroso y similar a la grasa y es producido por el hígado. Es necesario para una buena salud. Su cuerpo lo necesita para realizar trabajos importantes, como producer hormonas, vitamina D y partes de la células y proteger las fibras nerviosas.
- Su cuerpo produce todo el colesterol en sangre que necesita, por lo que los Expertos recomiendan que las personas consuman la menor cantidad posible de colesterol en la dieta mientras siguen un plan de alimentación saludable.
- El **colesterol dietético** se encuentra en los alimentos de origen animal, como la carne, el marisco, las aves de corral, los huevos y los productos lácteos.

¿Cuál es la diferencia entre el colesterol LDL y el colesterol HDL?

- El colesterol viaja a través de la sangre en proteínas llamadas "lipoproteínas". Hay 2 tipos:

LDL (lipoproteína de baja densidad): colesterol "malo"	HDL (lipoproteína de alta densidad): colesterol "bueno"
• El LDL constituye la mayor parte del colesterol de su cuerpo. • Los niveles altos de colesterol LDL pueden acumularse en las paredes de los vasos sanguíneos (llamado placa) y aumentarsu riesgo de enfermedad cardíaca y derrame cerebral.	• El HDL absorbe el colesterol y lo lleva de regreso al hígado.Luego, el hígado lo elimina del cuerpo. • Los niveles altos de colesterol HDL pueden reducir su riesgo deenfermedad cardíaca y derrame cerebral.

¿Cómo determino mis niveles de colesterol?

- Su equipo de atención médica puede realizar un análisis de sangre simple, llamado "perfil lipídico," para medir sus niveles de colesterol.
- Esta información ayuda a su equipo de atención médica a determinar su riesgo de enfermedad cardíaca o derrame cerebral.

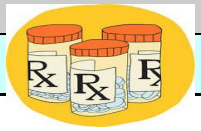
¿Cómo sé mi riesgo de colesterol alto?

- Ciertas afecciones de salud, su estilo de vida y sus antecedentes familiares pueden aumentar su riesgo de colesterol alto.
- Estos se denominan "factores de riesgo" y se enumeran a continuación en detalle.

Factores de riesgo para el colesterol alto	
Afecciones de salud	La diabetes tipo 2 y la obesidad pueden elevar los niveles de colesterol y grasas en sangre (triglicéridos).
Comportamientos	Una dieta alta en grasas saturadas y grasas trans, no hacer suficiente ejercicio, fumar, beber en exceso.
Antecedentes familiares	Algunas personas tienen una afección genética poco común llamada hipercolesterolemia familiar (familialhypercholesterolemia, FH). Esta afección causa niveles muy altos de colesterol de lipoproteínas de baja densidad (LDL o "malo") a partir deuna edad temprana que, si no se tratan, empeoran con la edad. Si alguien de su familia sufre un ataque cardíaco a una edad temprana, hable con su equipo de atención médicasobre su riesgo de sufrir de FH. Si tiene antecedentes familiares de colesterol alto, es más probable que tenga colesterol alto. Es posible quenecesite controlar sus niveles de colesterol con más frecuencia que las personas que no tienen antecedentesfamiliares de colesterol alto. El riesgo de colesterol alto puede aumentar aún más cuando los antecedentes familiares de colesterol alto secombinan con opciones de estilo de vida poco saludables, como una dieta poco saludable.
Edad y género	El riesgo de tener colesterol alto aumenta con la edad. Esto se debe a que, a medida que envejecemos, nuestros cuerpos no pueden eliminar el colesterol de la sangre tan bien como lo hacían cuando éramos másjóvenes. Esto resulta en niveles más altos de colesterol, lo que aumenta el riesgo de enfermedad cardíaca y derrame cerebral.

EDUCACIÓN PARA EL PACIENTE/ CONTROL PERSONAL DEL CASO

Medicamentos con estatinas



Lo que debe saber

¿Qué son las estatinas?

- Los medicamentos con estatinas generalmente son el primer tipo de medicamento que los médicos recetan para reducirlas LDL. También reducen los triglicéridos, que son otro tipo de grasa en la sangre y pueden elevar levemente su nivel decolesterol “bueno” (HDL).

¿Qué ocurre cuando tomo este medicamento?

- Debe hacerse un análisis de sangre antes de comenzar el tratamiento. Esto verifica el nivel de colesterol. Tambiénverifica si su hígado está funcionando de forma adecuada.
- Después de comenzar el tratamiento, debe realizarse un análisis de sangre dentro de 1 a 3 meses y de nuevo a los 12meses.
- Se revisa la sangre para medir el nivel de colesterol y ver qué tan bien está funcionando la estatina.

¿Cuáles son los efectos secundarios de este medicamento?

- La mayoría de las personas que toman estatinas no presentan efectos secundarios o solo presentanefectos secundarios menores.
- Los posibles efectos secundarios incluyen:
  - Dolor de cabeza
  - Dolores musculares
  - Dolor estomacal
  - Inflamación
  - Diarrea
  - Sensación de mareo (náuseas)
  - Sarpullido
  - Hormigueo
- Informe a su médico si tiene alguno de estos, especialmente dolores musculares, sensibilidad, calambres o debilidad. Estopuede ocurrir como un efecto secundario poco común de las estatinas y es una forma grave de inflamación muscular.
- Los dolores musculares pueden ser más probables si también está tomando un medicamento llamado amlodipino odiltiazem.
- Es posible que su médico tenga que ajustar su dosis de estatina para reducir el riesgo de daño muscular.

¿Cómo puedo saber si no debo tomar este medicamento?

- No debe tomar estatina si tiene una enfermedad hepática activa, si está embarazada otiene la intención de quedar embarazada, o si está amamantando.
- Debe dejar de tomar estatina si desarrolla una enfermedad hepática.

¿Qué debo evitar cuando tomo este medicamento?

- No coma toronja ni beba jugo de toronja si está tomando estatinas.
- Una sustancia química en la toronja puede aumentar el nivel de estatinas en el torrente sanguíneo, lo que aumenta la probabilidad de sufrir los efectos secundarios de la estatina. Este problema solo sucede con simvastatina, atorvastatina y lovastatina. Otras estatinas, comopravastatina, no interfieren con la toronja.
- En ocasiones, los medicamentos pueden interferir con las estatinas. Por ejemplo, algunos antibióticos y la ciclosporina. Es posible que se tengan que ajustar las dosis de la estatina o del otro medicamento que interactúa.
- Si le recetan (o compra) otro medicamento, recuérdele al médico o farmacéutico que está tomando una estatina en casode que puedan interferir.