

Foundations of Cardiometabolic Health Certification Course

Certified Cardiometabolic Health Professional (CCHP)



Lipid Tests and Risk Assessment for Patients with Dyslipidemia

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**CVD *risk* is the guide to the *intensity* of
lipid-lowering therapy**

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ASCVD Risk Assessment – Patient Case #1

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Case 1

47-year-old African American male is seen in follow-up 2 months after recent NSTMI. Risk factors are significant for HTN for 10 years and lifelong elevated cholesterol along with a family history of premature CHD and high cholesterol. He has taken numerous statins and was on 80 mg of atorvastatin and 10 mg of ezetimibe until he developed terrible muscle pain and weakness 4 months ago and stopped his statin. He was discharged on rosuvastatin 10 mg plus ezetimibe 10 mg and complains of sore muscles but denies any weakness.

PE: 135/85 mm Hg, height 6' 2", weight 180 lb, BMI 23.1 kg/m²

Corneal arcus present, no xanthomas or xanthelasma, normal cardiac and vascular exam

Case 1

Current meds:

- Chlorthalidone 25 mg/d, amlodipine 5 mg/ day, carvedilol 12.5 mg bid
- Rosuvastatin 10 mg, ezetimibe 10 mg
- ASA 81 mg daily

Lipid profile: Total cholesterol 198 mg/dL, triglycerides 100 mg/dL, HDL-C 60 mg/dL, LDL-C 118 mg/dL

Glucose 98 mg/dL. Normal renal function and LFTs, CK 320 U/L (ULN 170 U/L), hs-CRP 2.2 mg/L

What kind of lipid disorder does he have?
Do you think he has a genetic disorder?
If so, how do you make the diagnosis?

F

+

H

=

FH

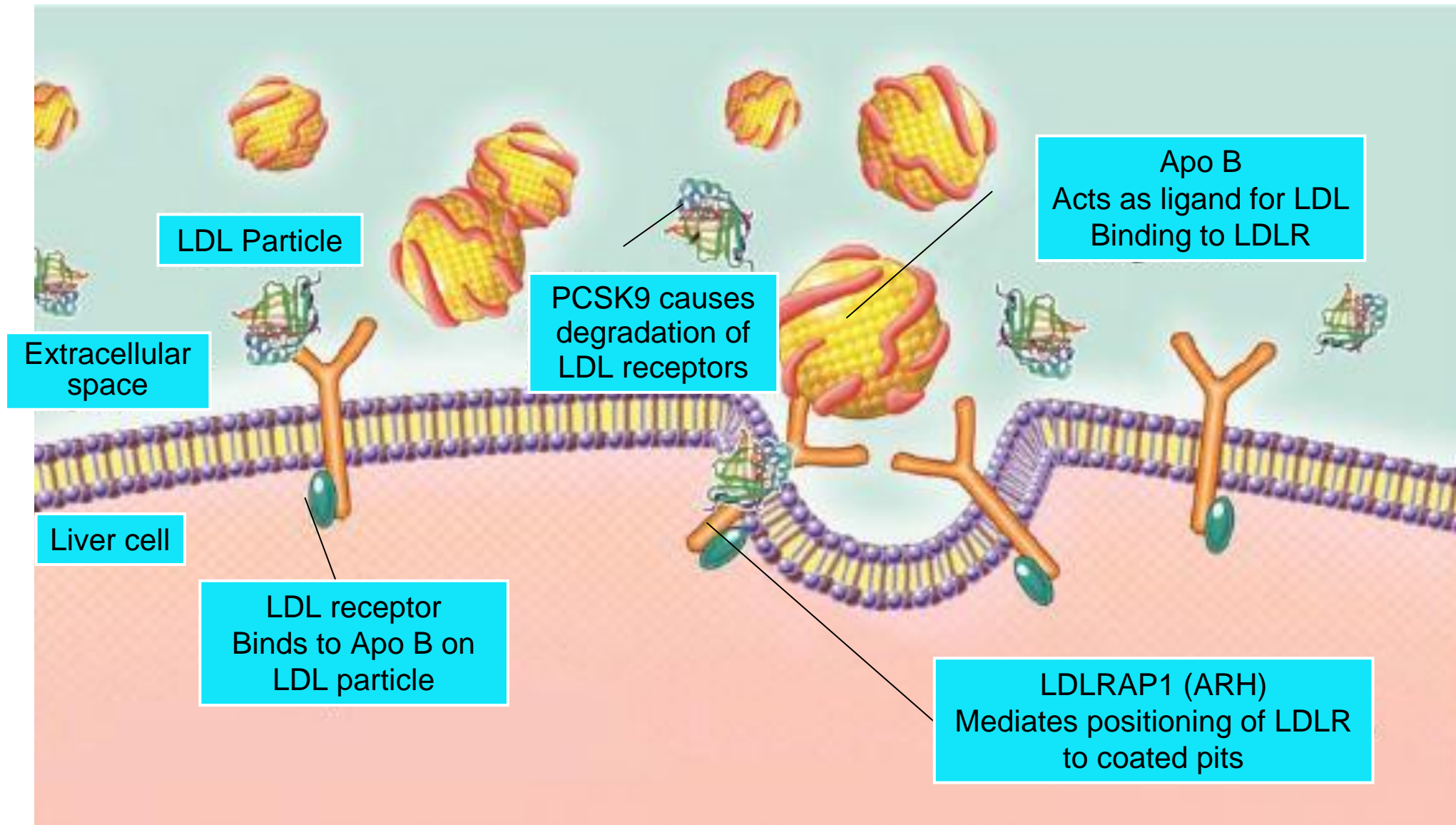


Family History
of early cardiovascular
events

High LDL Cholesterol
Above 190 mg/dL in adults
and 160 mg/dL in children

FH Familial
Hypercholesterolemia

4 Known FH Genes: LDLR, APOB, PCSK9, LDLRAP1



The rationale for genetic testing

Provides a definitive molecular diagnosis of FH

Provides prognostic and risk stratification information

Facilitates family-based cascade testing

Allows for precision during genetic counseling

Personal utility

Minimal psychological impact

Affordable and accessible

THE PRESENT AND FUTURE

JACC SCIENTIFIC EXPERT PANEL

Clinical Genetic Testing for Familial Hypercholesterolemia

JACC Scientific Expert Panel

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Convened by the Familial Hypercholesterolemia Foundation



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Genetic testing for gene variants associated with familial hypercholesterolemia (FH) can provide important medical information for individuals as well as their family members who may be at risk for FH.

Some benefits of genetic testing for FH:

- Confirmation of a clinical diagnosis of FH, especially in cases where it is not clear whether the person has FH or not.
- Provides more information about one's risk or diagnosis, since not all individuals with FH present the same.
- Often results in initiation and intensification of therapy by a healthcare provider. Studies have also shown that individuals with FH are more willing to start, intensify or continue taking prescribed medications when given genetic confirmation.
- Provides information regarding why a healthy lifestyle and diet have not been able to control cholesterol levels on their own.
- Helps other family members to be screened.
- Determines whether or not FH has been passed down to a child, since everyone with FH has a 50% chance of doing so.



Raising Awareness. Saving Lives.

Genetic Testing Can Help Clinicians and Patients Understand the Risk of Familial Hypercholesterolemia

Genetic testing is not right for everyone, and the test itself has limitations including:

- It does not always provide a simple "yes" or "no" answer about FH.
- A negative test result does not always mean someone does not have FH—it simply means that their genetic cause(s) were not identified with current knowledge and genetic testing technologies. About 30-40% of people with clinically diagnosed FH may test negative. These results may be "false negatives" or the person might have a gene variant

Potential Benefits of Genetic testing: NLA Scientific Statement on Genetic Testing

Positive genetic testing results can...

- Aid in clinical diagnosis and targeted treatment for specific disorders
- Guide optimal management and prevention strategies
- Increase patient adherence and motivation
- Provide early identification of affected family members through cascade screening

Genetic Testing Considerations: NLA Scientific Statement on Genetic Testing

- Not all genetic tests are created equal
- Review analytic and clinical validity of a test prior to medical management decisions
- Attributing pathogenicity to a DNA variant can differ between laboratories
- Direct-to-consumer genetic testing may provide both false positive and false negative results; confirmation using a clinical genetic test is recommended
- Currently polygenic scores for dyslipidemia are not standardized and not recommended or appropriate for clinical use in dyslipidemia
- Intensity of treatment for familial hypercholesterolemia should be guided by LDL-C elevation rather than the underlying genotype

Brown EE et al. *J Clin Lipidol* 2020;15:629-648).

Case

Current meds:

- Chlorthalidone 25 mg/d, amlodipine 5 mg/ day, carvedilol 12.5 mg bid
- Rosuvastatin 10 mg, ezetimibe 10 mg
- ASA 81 mg daily

Lipid profile: Total cholesterol 198 mg/dL, triglycerides 100 mg/dL, HDL-C 60 mg/dL, LDL-C 118 mg/dL

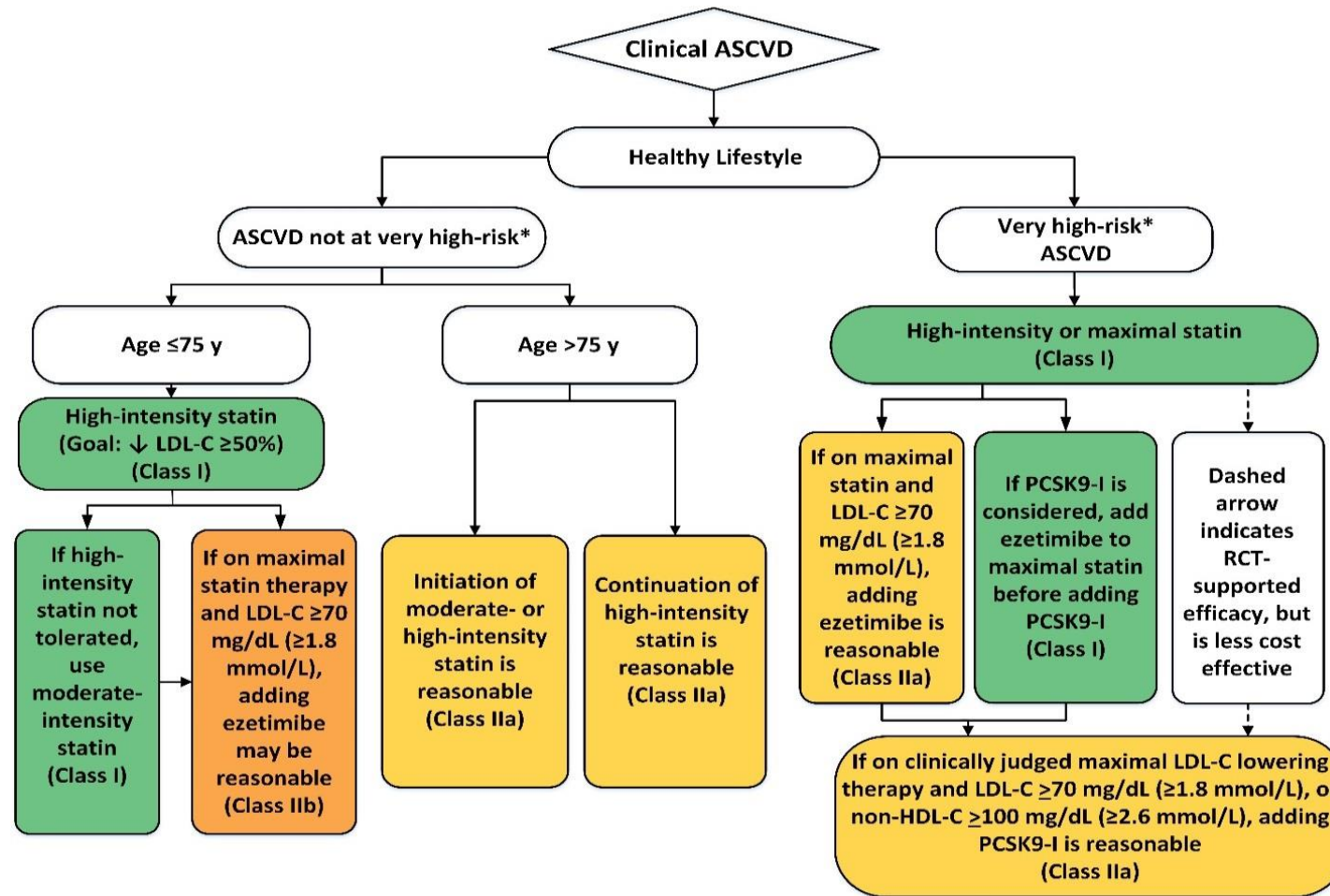
Glucose 98 mg/dL. Normal renal function and LFTs, CK 320 U/L (ULN 170 U/L), hs-CRP 2.2 mg/L

Case

Which of the following answers is most consistent with your views about his LDL-C?

1. The LDL-C target should be less than 70 mg/dL
2. The LDL-C target should be less than 100 mg/dL
3. There is no LDL-C target and he should be placed back on 40 mg of rosuvastatin with discontinuation of ezetimibe
4. His LDL-C is not an important issue and would focus on lowering his hs-CRP with higher dose aspirin therapy
5. His LDL-C is above the threshold to consider additional LDL-C–lowering therapy for a high-risk individual with ASCVD

Secondary Prevention: 2018 AHA/ACC Cholesterol Guidelines



Grundy SM et al. *J Am Coll Cardiol* 2019;73:e285–e350.

Very High-Risk ASCVD Patients

Major ASCVD Events

Recent ACS (within the past 12 mo)

History of MI (other than recent ACS event listed above)

History of ischemic stroke

Symptomatic peripheral arterial disease (history of claudication with ABI <0.85, or previous revascularization or amputation)

High-Risk Conditions

Age ≥ 65 y

Heterozygous familial hypercholesterolemia

History of prior coronary artery bypass surgery or percutaneous coronary intervention outside of the major ASCVD event(s)

Diabetes mellitus

Hypertension

CKD (eGFR 15-59 mL/min/1.73 m²)

Current smoking

Persistently elevated LDL-C (LDL-C ≥ 100 mg/dL [≥ 2.6 mmol/L]) despite maximally tolerated statin therapy and ezetimibe

History of congestive HF

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

According to the 2018 AHA/ACC guidelines:

What should be the next step in the treatment of his lipids?

Risk-based Treatment Goals/Thresholds for LDL-C, non-HDL-C, and apoB

Cardiovascular Risk	LDL-C, mg/dL	Non-HDL-C, mg/dL	ApoB, mg/dL
<i>American Heart Association/American College of Cardiology (2018)</i>			
Secondary prevention	≥70 threshold for nonstatins	≥100 threshold for nonstatins	Not recommended
Primary prevention	≥100 threshold for nonstatins	190–219 risk-enhancing factor	≥130 risk-enhancing factor
<i>European Society of Cardiology/European Atherosclerosis Society (2019)</i>			
Very high	<55 ≥50% reduction	<85	<65
High	<70 ≥50% reduction	<100	<80
Moderate	<100	<130	<100
Low	<116		

Grundy SM et al. *J Am Coll Cardiol* 2019;73:e285–e350.

Task Force for the management of dyslipidaemias of the ESC and EAS. *Eur Heart J* 2020;41:111-188.

**The patient was started on a PCSK9 inhibitor.
When should you obtain follow-up lipid labs?**

Lipid Measurements in the Management of Cardiovascular Diseases: Practical Recommendations

NLA Scientific Statement

CLINICAL CARE CONSIDERATIONS:

- Obtain lipid measurements 4–12 weeks after a change in lipid treatment
- Acutely ill patients may have lower lipid levels; inpatient lipid testing may require follow-up outpatient lipid testing
- For persons on lipid apheresis, measure lipids immediately before and after the apheresis procedure
- For persons using injectable lipid-lowering medications such as PCSK9 inhibitors, consider the number of days since the last injection and the time interval between injections when evaluating response to therapy

Wilson PFWF et al. *J Clin Lipidol* 2021;15:629-648).

What Test Should You Order to Evaluate the Response to Therapy?

1. Lipid panel with calculated LDL-C
2. Direct LDL-C
3. Apo B
4. LDL-P by NMR
5. LDL-P by ion mobility

Lipid Measurements in the Management of Cardiovascular Diseases: Practical Recommendations

NLA Scientific Statement

CLINICAL CARE CONSIDERATIONS:

- In the absence of a cost-effective, accurate, and widely available direct method of measuring LDL-C, the assessment of LDL-C in clinical trials and in clinical practice has largely relied on calculation of LDL-C, recognizing problematic underestimation particularly in patients with high TG
- Non-HDL-C is measured reliably in either the fasting or the nonfasting state and can effectively guide ASCVD prevention
- LDL-C can be estimated from total cholesterol, HDL-C, and TG measurements:
 - *Friedewald equation in patients with LDL-C >100 mg/dL and TG ≤150 mg/dL*
 - *Martin/Hopkins method throughout the range of LDL-C levels and TG ≤399 mg/dL*
 - *Current LDL-C–estimating equations are not recommended for TG ≥400 mg/dL*
- When LDL-C or TG screening results are abnormal (especially TG >175 mg/dL), consider obtaining fasting lipids

Lipid Measurements in the Management of Cardiovascular Diseases: Practical Recommendations

NLA Scientific Statement

SAMPLE LIPID LAB REPORT:

Measurement	Desirable Values*	Results	High Alert Values* (Refer to Lipid Specialist)
Patient Name			
Fasting Yes () No ()			
Total cholesterol	<200 mg/dL		
HDL-C	>40 mg/dL for men >50 mg/dL for women		≤ 20 mg/dL
Non-HDL-C	<130 mg/dL <100 mg/dL for ASCVD* or high risk pts		≥220 mg/dL Consider inherited hyperlipidemia
LDL-C	<100 mg/dL <70 mg/dL for ASCVD or high risk pts		<50 untreated ≥190 mg/dL Consider Familial Hypercholesterolemia
TG	<150 mg/dL fasting <175 mg/dL nonfasting ¹		500-999 mg/dL – severe ≥1000 mg/dL – critical value

Wilson PFWW et al. *J Clin Lipidol* 2021;15:629-648).

Lipid Measurements in the Management of Cardiovascular Diseases: Practical Recommendations

NLA Scientific Statement

ADVANCED LIPOPROTEIN TESTING CONSIDERATIONS IN SELECT PATIENTS

- Measurement of apoB may help to guide therapy as part of the initial lipid evaluation in selected patients and in persons on lipid therapy
- Measurement of LDL-P may help to guide therapy in persons after initial lipid evaluation in selected patients, but LDL-P assays are not standardized
- Measurement of Lp(a) can help to guide therapy in persons with primary hypercholesterolemia or those at very high risk to develop ASCVD events
- For patients with high ASCVD risk and LDL-C <70 mg/dL, residual atherogenic burden may be ascertained by assessing lipid measures beyond LDL-C; the most widely used assays are non-HDL-C, apoB and LDL-P

Wilson PFWW et al. *J Clin Lipidol* 2021;15:629-648).

Lipid Measurements in the Management of Cardiovascular Diseases: Practical Recommendations

NLA Scientific Statement

OTHER ATHEROGENIC LIPID/LIPOPROTEIN MEASURES (I):

Measure	Method	Analytes	Sources of Variability	Bias (%) * Precision; CV; Total Error (%)
Revised Calc-LDL-C	estimate cholesterol contents in TGRL based on TG level	TC, HDL-C, TG	same as of traditional lipid profile	NA
Remnant lipoprotein cholesterol (REM-C)	precipitation using a mixture of specific antibodies; REM-C = TC – LDL-C – HDL-C	TC, LDL-C, HDL-C	same as of traditional lipid profile	NA
Non-HDL-C	calculation: TC – HDL-C	TC, HDL-C	same as for TC and HDL-C	1.2; 3.3%; 6.7
apoB	immunospectrometry, nephelometry, ELISA	apoB	immunoassay	-27% to + 11% vs IN; 14.1%; N/A

Wilson PFW et al. *J Clin Lipidol* 2021;15:629-648).

Lipid Measurements in the Management of Cardiovascular Diseases: Practical Recommendations

NLA Scientific Statement

OTHER ATHEROGENIC LIPID/LIPOPROTEIN MEASURES (II):

Measure	Method	Analytes	Sources of Variability	Bias (%) * Precision; CV; Total Error (%)
HDL-P subclasses; VLDL-P, LDL-P, HDL-P, sizes	NMR	count of lipoprotein particles	count can change with software version	N/A; <5%; NA
LDL-P and HDL-P	electrospray differential mobility analysis	particle count of different size analytes	operational parameters and calibration	N/A; 4%; N/A
Small dense LDL-C	gel electrophoresis, selective precipitation	cholesterol in small dense LDL		N/A; 8%; N/A
Lp(a)	immunoturbidometry, nephelometry, ELISA	apo(a)	genetic heterogeneity	N/A CV = 11% N/A

Wilson PFWW et al. *J Clin Lipidol* 2021;15:629-648).

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ASCVD Risk Assessment – Patient Case #2

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Case 2

53-year-old white male with a family history of CVD, diabetes and hyperlipidemia presents complaining of a rash on his abdomen and back. He has a history of hypertension, high cholesterol and triglycerides which has been treated for 10 years. He has not been exercising, says he has had long hours and lots of stress and has gained 15 lbs since his last visit 2 years ago.

Current meds: Atenolol 100 mg, HCTZ 25 mg, ASA 81 mg, atorvastatin 10 mg

Question 1

According to the ACC/AHA guidelines, should you check his lipid profile?

1. Yes
2. No – primary prevention on appropriate therapy of atorvastatin 10 mg
3. Unsure

Question 1

According to the ACC/AHA guidelines, should you check his lipid profile?

- 1. Yes**
2. No – primary prevention on appropriate therapy of atorvastatin 10 mg
3. Unsure

2013 ACC/AHA Cholesterol Guidelines: Monitoring Statin Therapy

- Adherence to medication and lifestyle, therapeutic response to statin therapy, and safety should be regularly assessed [I/A]
 - *This should also include a fasting lipid panel performed within 4–12 weeks after initiation or dose adjustment, and every 3–12 months thereafter*
 - *Other safety measurements should be measured as clinically indicated*

Physical Exam

Initial exam

- Obese
 - *BP 145/95 mm Hg*
 - *height 5'10"*
 - *weight 220 lb, BMI 31.6*
 - *waist 42 inches (107 cm)*
 - *normal cardiac and vascular exam*
 - *liver is palpable 4 cm below costal margin*



Lab Values

- TG: 1150 mg/dL
- HDL-C: 30 mg/dL
- TC 310 mg/dL
- Glucose: 165 mg/dL
- AST: 60 U/L (ULN 42), ALT: 60 U/L (ULN 55)
- Bilirubin 1.0 mg/dL, alkaline phosphatase 77 U/L
- BUN 23 mg/dL, creatinine 1.4 mg/dL
- TSH 2.37 mIU/L (ULN 5.50)

Question 2

What is the most likely lipid diagnosis?

- a. Type I hyperlipidemia
- b. Type II hyperlipidemia
- c. Type III hyperlipidemia
- d. Type IV hyperlipidemia
- e. Type V hyperlipidemia

Question 2

What is the most likely lipid diagnosis?

- a. Type I hyperlipidemia
- b. Type II hyperlipidemia
- c. Type III hyperlipidemia
- d. Type IV hyperlipidemia
- e. **Type V hyperlipidemia**

Question 3

Which of the following may be an important secondary cause of his hyperlipidemia?

- a. Alcohol intake
- b. Diabetes
- c. Beta-blocker
- d. Diuretic
- e. All of the above

Question

3

Which of the following may be an important secondary cause of his hyperlipidemia?

- a. Alcohol intake
- b. Diabetes
- c. Beta-blocker
- d. Diuretic
- e. All of the above**

Question 4

Which agent would not be effective for reducing his TG?

- a. Statin
- b. Fibrate
- c. Niacin
- d. Bile acid-binding resin
- e. Omega-3 fatty acids

Question 4

Which agent would not be effective for reducing his TG?

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- d. Bile acid-binding resin**
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Lab Values

- TG: 1150 mg/dL
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- TSH 2.37 mIU/L (ULN 5.50)

Approach to the Patient with Hypertriglyceridemia

- Evaluation of secondary causes of hypertriglyceridemia.
- Attention should be given to alcohol intake, medication use, signs and symptoms of diabetes, weight gain, and dietary habits.
- Laboratory tests should include glucose, HbA1C, BUN/creatinine, urinalysis, thyroid-stimulating hormone (TSH), alanine aminotransferase (ALT), and aspartate aminotransferase (AST).

Question 5

What is the initial goal of therapy recommended by the lipid guidelines for this patient with severe hypertriglyceridemia?

- a. TG <150 mg/dL
- b. TG <500 mg/dL
- c. Non-HDL-C <160 mg/dL
- d. HDL-C >50 mg/dL
- e. All of the above

Question 5

What is the initial goal of therapy recommended by the lipid guidelines for this patient with severe hypertriglyceridemia?

- a. TG <150 mg/dL
- b. TG <500 mg/dL**
- c. Non-HDL-C <160 mg/dL
- d. HDL-C >50 mg/dL
- e. All of the above

Treatment Plan

- He was instructed to stop all alcohol, scheduled for a dietary consult for a diet with a reduction in fat and simple carbohydrates, and instructed to begin walking 30 minutes daily
- Medications
 - *Antihypertensive regimen was changed to amlodipine 10 mg plus lisinopril 20 mg once daily*
 - *Metformin 500 mg BID was initiated; check A1C at baseline*
 - *Prescription omega-3 fatty acids 2 gm po BID was initiated*

Laboratory Assessment

<u>Measurement</u>	<u>Baseline</u>	<u>6 weeks</u>
Weight (lbs)	220	214
BMI	31.6	30.7
Blood pressure (mmHg)	145/95	135/85
Waist (inches)	42	41
TG (mg/dL)	1650	350
TC (mg/dL)	310	202
HDL-C (mg/dL)	35	42
LDL-C (mg/dL)	-	90
Non HDL-C	280	162
Glucose (mg/dL); A1C	175 ND	125 6.4%

Question 6

What would you recommend at this time?

- a. Increase atorvastatin to 80 mg
- b. Increase exercise and intensify diet to achieve further weight loss
- c. Increase metformin to 1000 mg bid
- d. All of the above

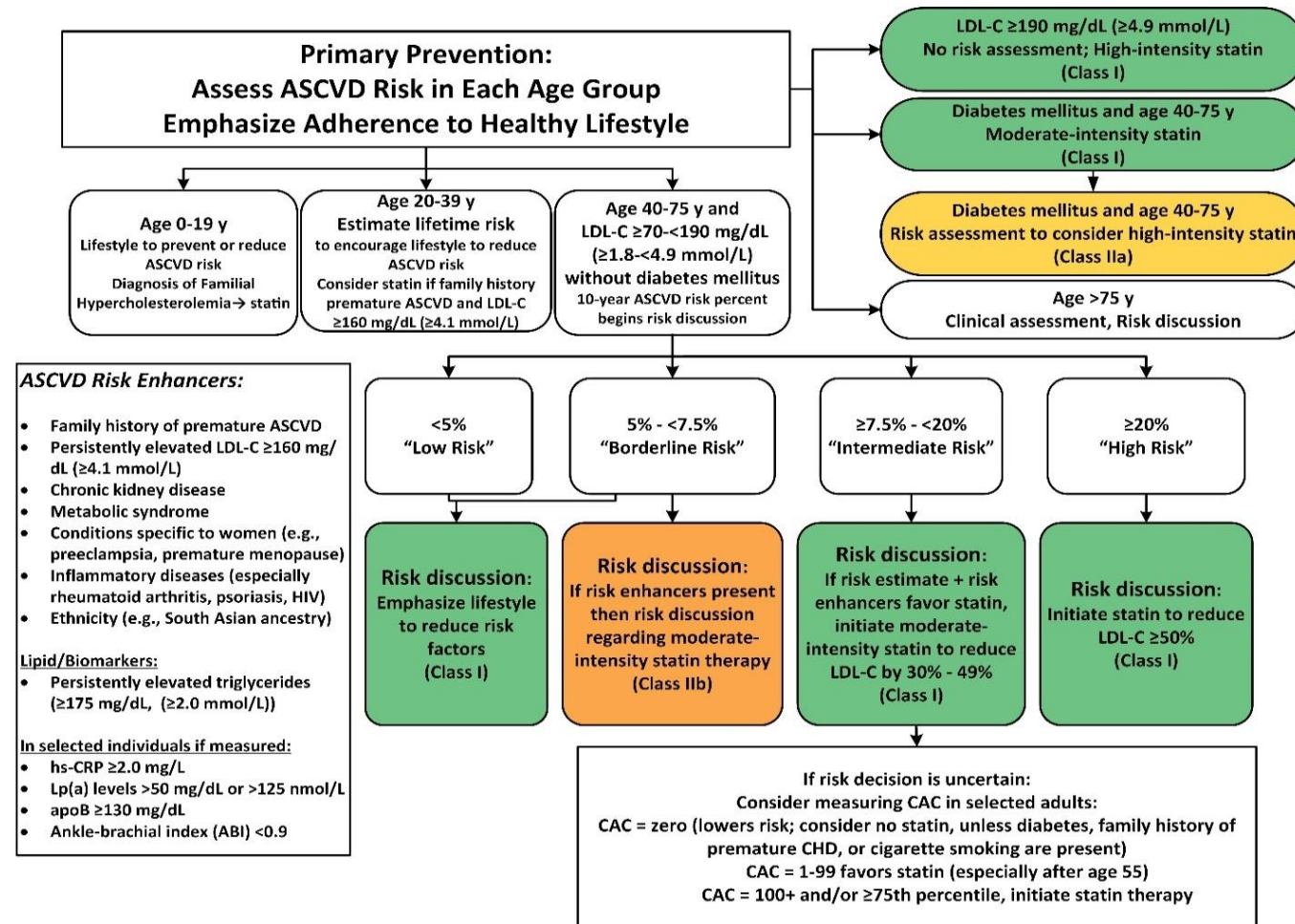
In patients aged 40–75 years with diabetes and LDL-C \geq 70 mg/dL (\geq 1.8 mmol/L), start moderate-intensity statin therapy without calculating 10-year ASCVD risk

- In patients with diabetes mellitus at higher risk, especially those with multiple risk factors or those aged 50–75 years, it is reasonable to use high-intensity statin to reduce LDL-C by \geq 50%

Recommendations for Patients with Diabetes Mellitus

- In adults aged 40–75 years and LDL-C 70–189 mg/dL, it is reasonable to assess 10-year risk of first ASCVD event by using race- and sex-specific PCE to help stratify ASCVD risk (IIa B-NR)
- In adults with multiple ASCVD risk factors, it is reasonable to prescribe high-intensity statin therapy to reduce LDL-C by $\geq 50\%$ (IIa B-R)
- In adults aged ≥ 75 years and already on statin therapy, it is reasonable to continue statin therapy (IIa B-NR)
- **In adults with 10-year ASCVD risk $\geq 20\%$, it may be reasonable to add ezetimibe to maximally tolerated statin to reduce LDL-C by $\geq 50\%$ (IIb C-LD)**
- In adults aged ≥ 75 years, it may be reasonable to initiate statin therapy after a clinician–patient discussion of potential benefits and risks (IIb C-LD)
- In adults aged 20–39 years with DM of long duration (≥ 10 y T2DM, ≥ 20 y T1DM), albuminuria (≥ 30 μg albumin/mg creatinine), eGFR < 60 mL/min/1.73 m², retinopathy, neuropathy, or ABI < 0.9 , it may be reasonable to initiate statin therapy (IIb C-LD)

Primary Prevention: 2018 AHA/ACC Cholesterol Guidelines



Grundy SM et al. *J Am Coll Cardiol* 2019;73:e285–e350.

Risk-Enhancing Factors (I)

Risk-Enhancing Factors

- **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 [>175 mg/dL], elevated blood pressure, elevated glucose, and low HDL-C [<40 mg/dL in men; <50 in women mg/dL] 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 psoriasis, RA, or HIV/AIDS
- **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)

Grundy SM et al. *J Am Coll Cardiol* 2019;73:e285–e350.

Risk-Enhancing Factors (II)

Risk-Enhancing Factors

- **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 Lp(a):** A relative indication for its measurement is 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:** A relative indication for its measurement would be triglyceride ≥ 200 mg/dL. A level ≥ 130 mg/dL corresponds to an LDL-C > 160 mg/dL and constitutes a risk-enhancing factor
 - **ABI < 0.9**

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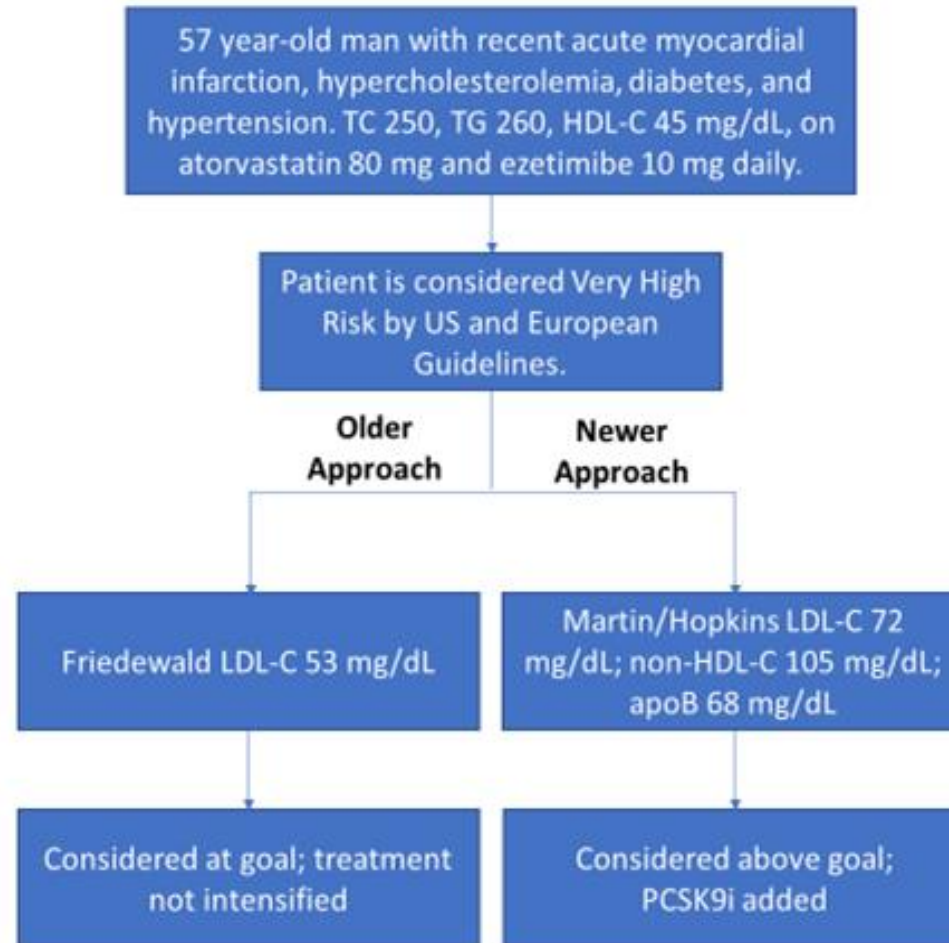
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ASCVD Risk Assessment – Patient Case #3

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Case #3



Mora S and Martin SS. *Clinical Lipidology*. In press.

Methods to Calculate LDL-C

1. Friedewald Equation

- $LDL-C = TC - (HDL-C + TG/5)$
- can be used if $TG < 400$ mg/dL

2. Martin–Hopkins equation: better performance for after intensive LDL-C reductions (Friedewald gives falsely low levels) and also when TG are higher.

3. NIH equation: Better performance than Friedewald equation across the range of TGs

Fukuyama N et al. *J Clin Biochem Nutr* 2007;43:1-5.

Martin SS et al. *JAMA* 2013;310:2061-2068.

Sampson M et al. *JAMA Cardiol* 2020;5:540-548.

Recommendations for Clinical Use of apoB or LDL-P

National Lipid Association Dyslipidemia Statement 2015

- ApoB is optional secondary target for treatment. Consider LDL-P as an alternative to apoB.

American Heart Association / American College of Cardiology Multi-society 2018/2019 Guidelines

- ApoB ≥ 130 mg/dL is a risk enhancing factor (if measured) for personalizing risk assessment in patients with borderline to intermediate risk (10-year ASCVD risk of 5-20%). Consider testing when triglycerides ≥ 200 mg/dL.

European Society of Cardiology/European Atherosclerosis Society 2019 Guidelines

- ApoB is recommended for risk assessment, particularly in people with high triglycerides, diabetes, obesity, metabolic syndrome, or very low LDL-C.
- ApoB is an alternative to LDL-C, if available, as the primary measurement for screening and management, and may be preferred over non-HDL-C in people with high triglycerides, diabetes, obesity, or very low LDL-C.

Grundy SM et al. *J Am Coll Cardiol* 2019;73:e285–e350.

Task Force for the Management of Dyslipidaemias of the ESC and EAS. *Eur Heart J* 2020;41:111-188.

Jacobson TA et al. *J Clin Lipidol* 2015;9:129-69.

Summary

1. CVD risk assessment is the guide to the intensity of lipid lowering therapy
2. Follow up assessment of lipids is necessary in all patients to assess efficacy and adherence to lifestyle and drug therapy
3. Atherogenic lipoproteins are the target of therapy
4. Levels of LDL-C, non HDL-C, apo B, LDL-p, Lp(a) and triglycerides may all be useful to assess lipoprotein related risk
5. Follow up testing with calculated LDL-C, non HDL-C, apo B and LDL-P **may** be useful to assess response to therapy and need to intensify therapy.
6. Important to understand limitations of calculated LDL-C and differences in methodologies re measurement of LDL-P