Optum Health Education™

Contemporary Evaluation of Stable Chest Pain and Coronary Calcium Scoring in Primary Prevention

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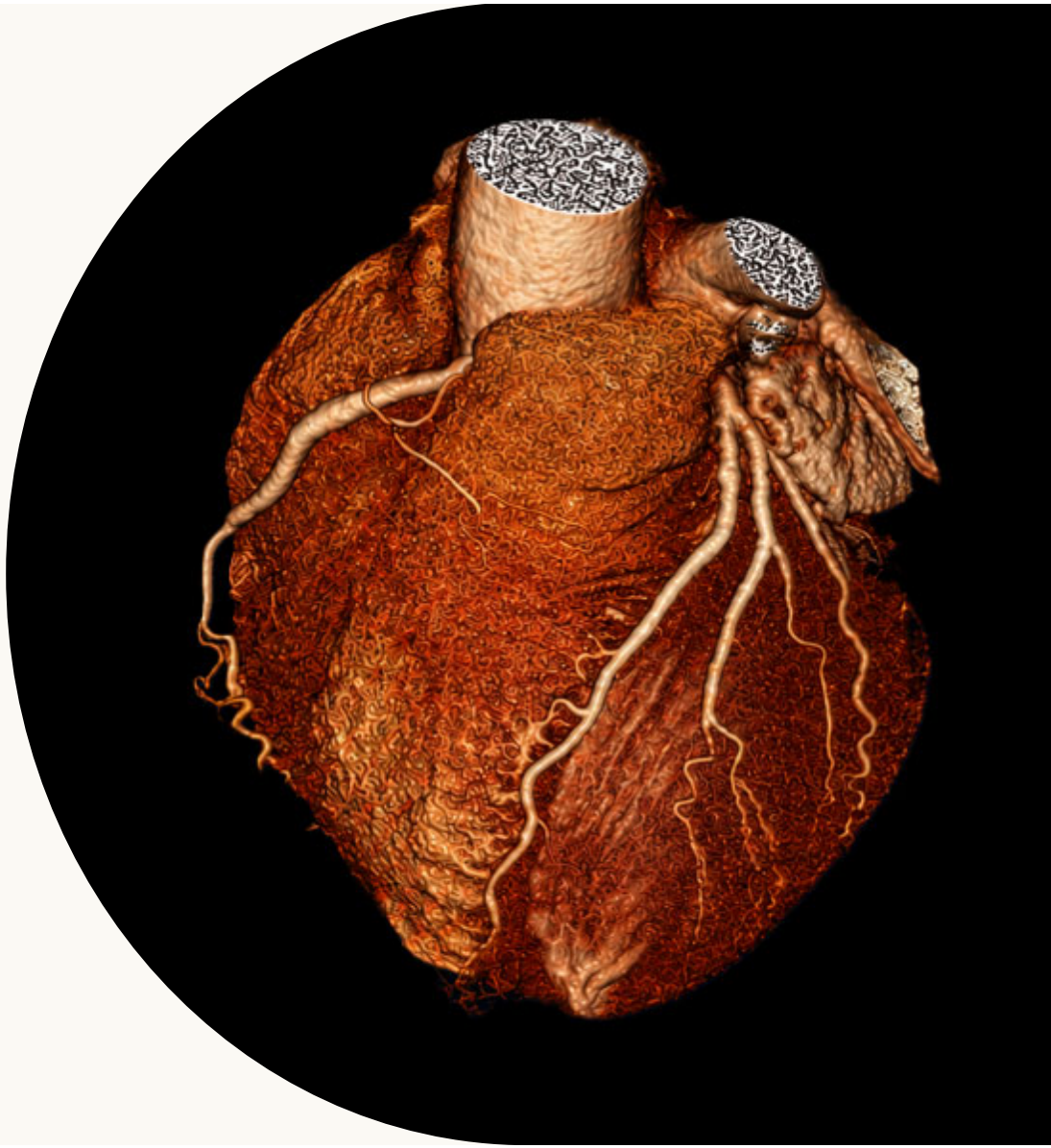

Learning objectives

At the end of this educational activity, participants should be able to:

- Review contemporary evaluation strategies for stable chest pain without known CAD.
- Select appropriate noninvasive testing strategies based on individual patient risk profiles.
- Assess the clinical role and limitations of CAC scoring in cardiovascular risk stratification.
- Apply current lipid guideline recommendations to support evidence-based management in primary prevention.

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CLINICAL SCENARIO

65-year-old female presents with 3 months history of chest pain with walking uphill, resolves at rest

Past medical history: diabetes, hypertension, hyperlipidemia

On exam: BP 130/80, heart rate 65, no murmur

EKG: NSR 66 BPM

Blood tests: normal hemoglobin, creatinine, LDL 100

What to do?

Pretest Probabilities of Obstructive CAD in Symptomatic Patients
(A) according to age, sex, and symptoms;
(B) according to age, sex, symptoms, and CAC

Age, y	Chest Pain		Dyspnea	
	Men	Women	Men	Women
30–39	≤4	≤5	0	3
40–49	≤22	≤10	12	3
50–59	≤32	≤13	20	9
60–69	≤44	≤16	27	14
70+	≤52	≤27	32	12

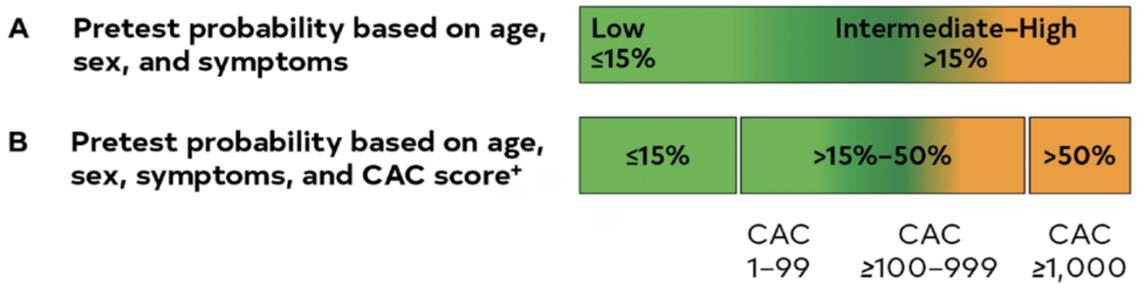

Figure 11. Pretest Probabilities of Obstructive CAD in Symptomatic Patients According to Age, Sex, and Symptoms
Modified from Juarez-Orozco et al[1] and Winther et al.[2] 1) The pretest probability shown is for patients with anginal symptoms. Patients with lower-risk symptoms would be expected to have lower pretest probability. 2) The darker green– and orange-shaded regions denote the groups in which noninvasive testing is most beneficial (pretest probability >15%). The light green–shaded regions denote the groups with pretest probability of CAD ≤15% in which the testing for diagnosis may be considered based on clinical judgment.[1] 3) If CAC is available, it can also be used to estimate the pretest probability based on CAC score.[2] CAC indicates coronary artery calcium; and CAD, coronary artery disease.

COR	LOE	Recommendations
Index Diagnostic Testing		
Anatomic Testing		
1	A	1. For intermediate-high risk patients with stable chest pain and no known CAD, CCTA is effective for diagnosis of CAD, for risk stratification, and for guiding treatment decisions.[1-12]
Stress Testing		
1	B-R	2. For intermediate-high risk patients with stable chest pain and no known CAD, stress imaging (stress echocardiography, PET/SPECT MPI or CMR) is effective for diagnosis of myocardial ischemia and for estimating risk of MACE.[8,13-35]

LEVEL (QUALITY) OF EVIDENCE‡

LEVEL A

- High-quality evidence‡ from more than 1 RCT
- Meta-analyses of high-quality RCTs
- One or more RCTs corroborated by high-quality registry studies

LEVEL B-R **(Randomized)**

- Moderate-quality evidence‡ from 1 or more RCTs
- Meta-analyses of moderate-quality RCTs

OUTCOMES: FUNCTIONAL STRESS TESTS VS. CCTA

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Coronary CT Angiography and 5-Year Risk of Myocardial Infarction

The SCOT-HEART Investigators*

Newby et al, NEJM 2018

4,146 patients across the UK randomized between CCTA or standard care

Key findings: **41% lower death & MI rate in CCTA group** than in standard care group at 5 years despite similar rates of cath, CABG and PCI in both groups; 40% higher preventive therapies in CT arm

CCTA 1st strategy - outcomes:

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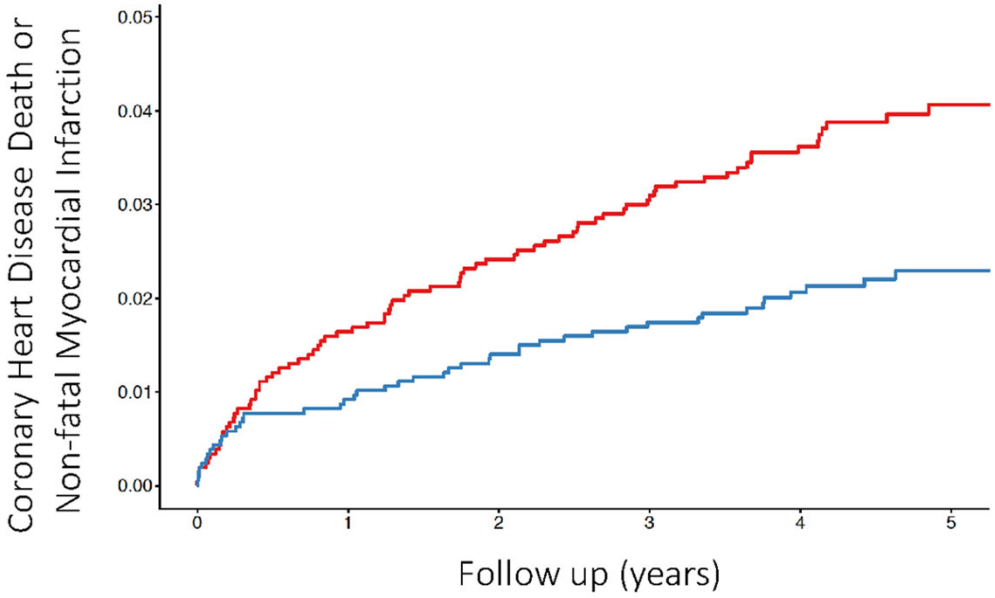
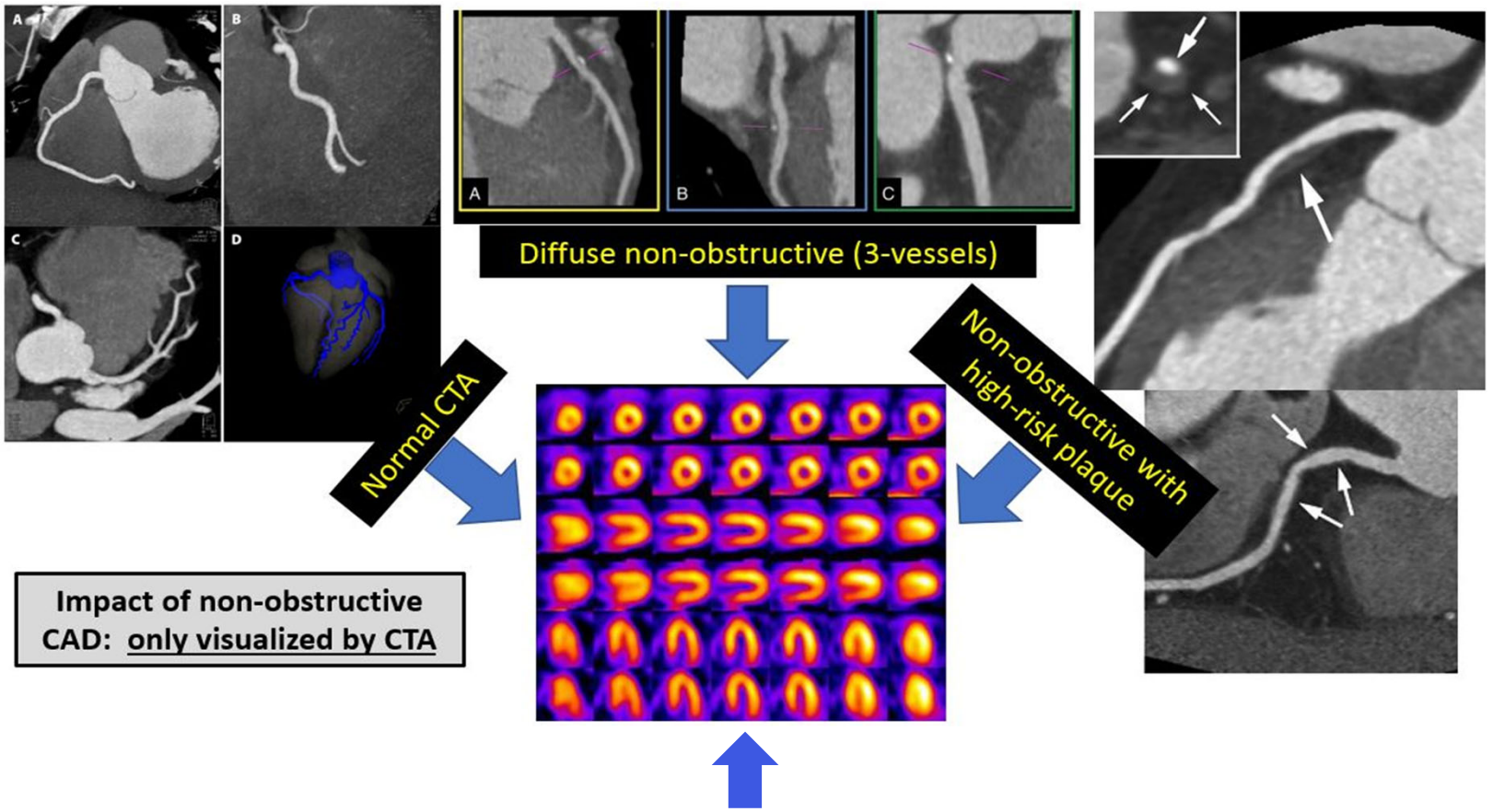


*Changes in diagnosis occurred in 25% of patients receiving CCTA and only 1% of pts receiving standard protocol ($P < .001$).

Nuclear Stress Test vs CCTA

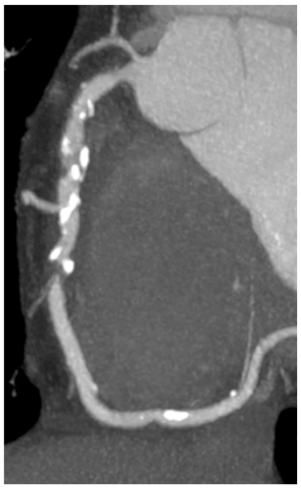


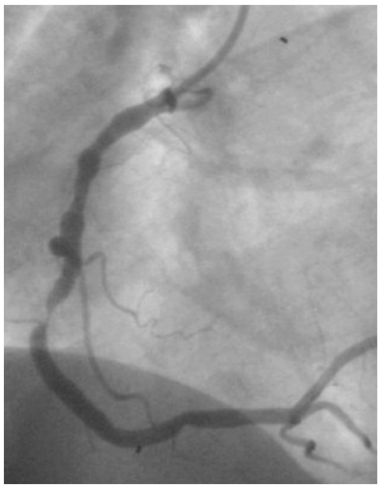
Testing Diagnostic Performance

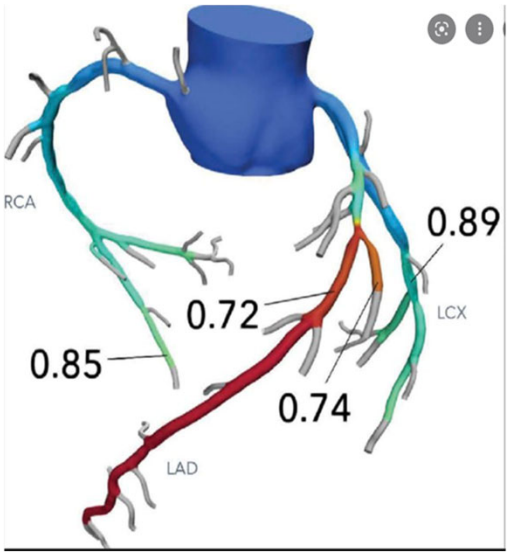
Test	Sensitivity	Specificity
Exercise ECG treadmill	68%	77%
Exercise Echo treadmill	86%	81%
Dobutamine Echo	~85%	~85%
Exercise nuclear treadmill	87%	73%
Pharmacologic nuclear	89%	75%
PET stress test	91%	87%
Coronary CTA	**95%**	**83%**

CT angiography vs invasive angiography

- **Atherosclerosis/anatomic detail/stenosis**
- **Ischemia : FFRct**







- From typically acquired CCTA
- Applies computational fluid dynamics to CCTA

- **Lower Radiation Dose (2-7 mSv)**
- **Diagnostic cath 7mSv**
- **Rest-stress Tc nuclear stress test 11 mSv**

Source: Min JK et al. J Cardiovasc Comput Tomogr 2011, Min JK et al. Am J Card 2012; Min JK et al. J Cardiovasc Comput Tomogr. 2012; Grunau GL et al. Curr Cardiol Report; Min JK et al. JAMA 2012; Koo et al. J Am Coll Cardiol 2012

CTA Contraindications/limitations

- **Stage 4 CKD.** GFR less than 30 ml/min
- **Allergy** to contrast
- **Inability to lay flat**, follow command such as breath hold
- **Inability to tolerate beta-blockers** (bronchospasm ect)
- **Overestimation of stenosis** with severe coronary calcifications
- **Suboptimal imaging:**
  - Tachycardia
  - Morbid obesity: BMI >40 kg/m2
  - Unable to receive beta-blockers
  - CABG
  - Previous stents

Recommendations for patients with stable chest pain

1) Low risk <15% group: no testing or CAC or exercise stress test (CCTA if abnormal)

2) Intermediate and high risk >15%: CCTA

3) Patients who have contraindication to CCTA (CKD, AFib, contrast allergy) or patients with established CAD: Cardiology referral

Coronary Artery Calcium Score (CAC)
in Primary Prevention
(Screening of Asymptomatic Patients)

American Heart Association.

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Why you should use CAC score in your practice:

- Simple, easy test, inexpensive, **no IV, no contrast**
- Radiation **similar** to a transatlantic flight or a mammogram
- Identifies **actual** disease
- Positive influence on patient **lifestyle** and risk factor choices
- **Superior** and additive to ASCVD risk scores; offers more accurate risk classification, with % of reclassification as follows:

Low risk	Intermediate risk	High risk
up to **15%**	up to **66%**	up to **36%**

When is CAC score not useful?

1. In younger populations (men < 40 yr or women < 45 yr)
2. When initial score is above zero, there is no consensus on repeating CAC score
3. When checking the effectiveness of statin therapy
4. In very low or very high risk of ASCVD
5. If other imaging tests showed vascular calcifications

When to Consider Coronary Artery Calcium (CAC) Score

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Statin-naive patients with high ASCVD risk who are hesitant to initiate statin therapy

Adults with intermediate ASCVD risk with factors that increase their ASCVD risk

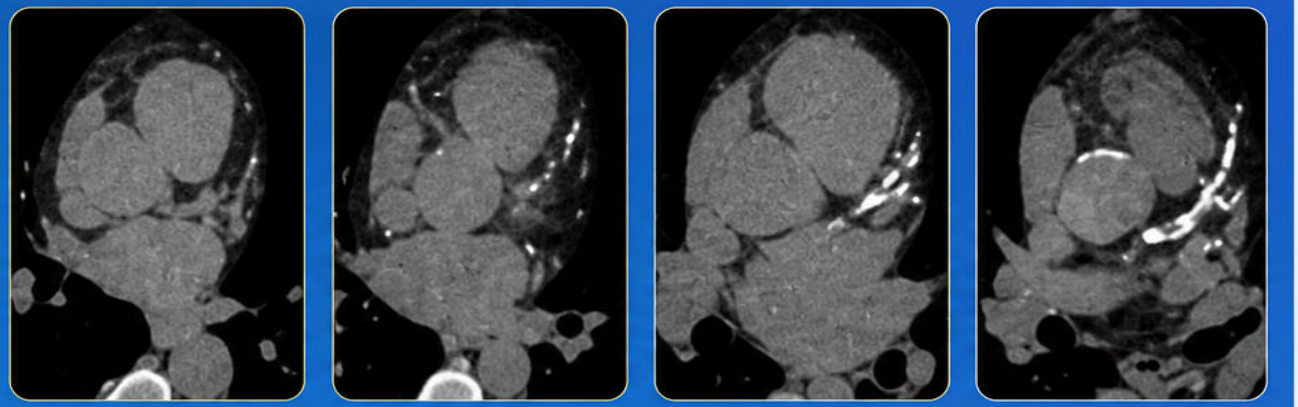
Warranty Period of a CAC = 0 (recommended rescan interval):

- **Low risk:** 6–7 years
- **Borderline to intermediate risk:** 3–5 years
- **High risk:** 3 years

What to do with CAC score:

- **CAC = 0.** Consider NO statin, unless DM and other **high risk** conditions, LDL-C >160 mg/dl, FH of premature CHD, or cigarette smoking
- **CAC = 1–99.** Favors statin
- **CAC score = 100+.** Low dose Aspirin and initiate statin
- **CAC score > 300.** Treat as established ASCVD or refer to cardiology

Coronary Calcium Test Scores



0	1–100	101–299	300+
No calcium is detected	Mildly increased risk of heart disease	Moderately increased risk of heart disease	Moderately to severely increased risk of heart disease

Higher CAC score = Higher risk. Use in clinical context to guide preventive therapy.

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When CAC score test is "too early":

- Males <40 yr, females <45 yr without risk factors
- Low ASCVD risk unless family history of premature CAD or current smokers

When CAC score test is "too late" (to change clinical management):

- Already on statin therapy/ to measure statin efficacy against reducing plaque volume
- Age > 65 yr with many ASCVD risk factors
- Age > 80 yr

When CAC score test is "too often":

- Repeating CAC if CAC >300 for progression of disease
- Repeating CAC score if CAC >100 and on risk-reduction therapy
- Repeating CAC in older patients >75 yr if CAC = 0

Circulation
Volume 153, Issue 17, 28 April 2026; Pages e1154–e1276
https://doi.org/10.1161/CIR.0000000000001423

American Heart Association.

CLINICAL PRACTICE GUIDELINES

2026

ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/ Guideline on the Management of Dyslipidemia: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

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1 Start Earlier: Reduce Lifetime Exposure to LDL-C

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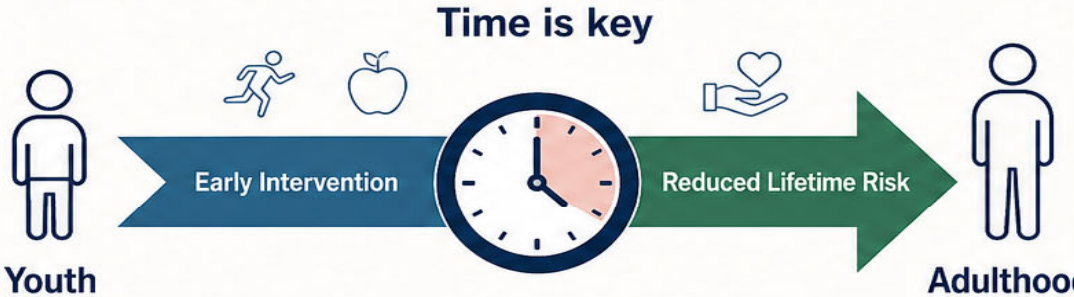
Start Statin Therapy for:

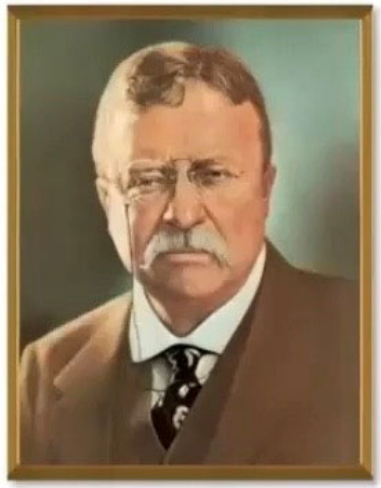
- All Adults with HeFH
- Age ≥30:
  - LDL-C ≥160 mg/dL **OR**
  - Strong family history of premature ASCVD **OR**
  - High 30-year risk of ASCVD



Treat these groups **early** even if 10-year risk is low to prevent long-term exposure to high LDL-C.

1	B-NR	1. In adults, screening with a lipid profile is recommended beginning at age 19 y and at least every 5 y thereafter to identify treatable ASCVD risk, with frequent screening recommended for individuals with additional ASCVD risk factors.[1,2]
1	B-NR	2. In children 9 to 11 y of age not previously tested, it is recommended to screen with a lipid profile to identify familial hypercholesterolemia (FH) and other significant lipid disorders.[3–6]
2a	B-NR	3. In individuals with first- or second-degree relatives with premature ASCVD, severe hypercholesterolemia, or FH, it is reasonable to perform screening with a single lipid profile (eg, cascade screening) starting at ≥2 y of age to identify FH.[7–10]

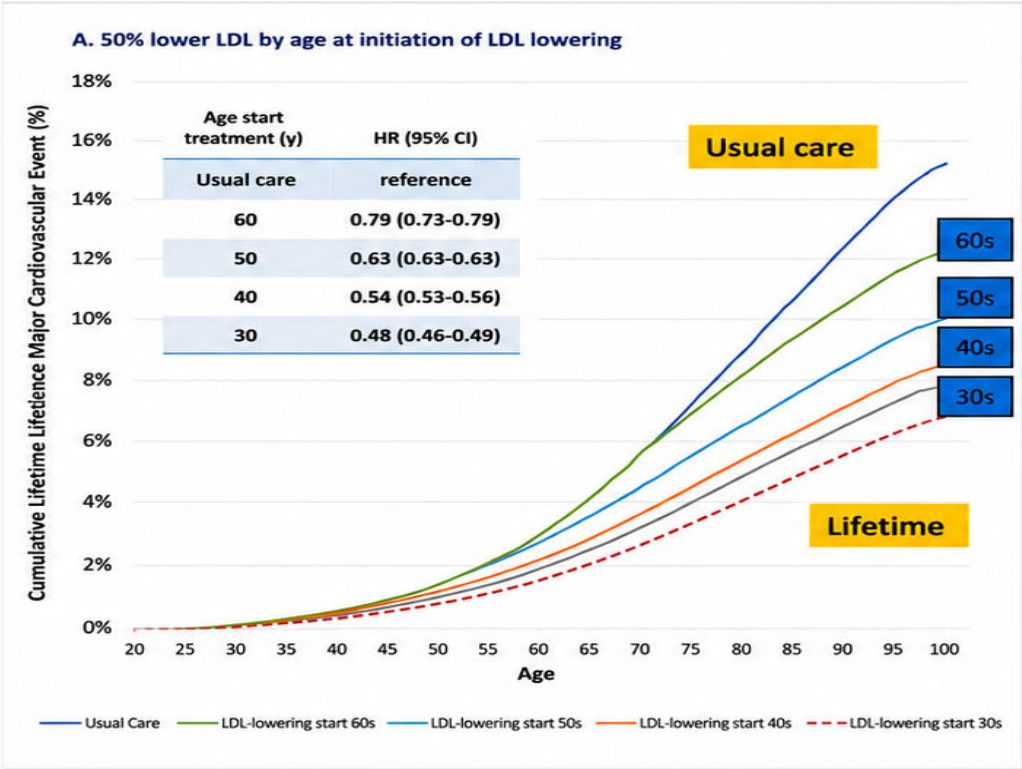
In the United States, approximately 25% of adults have an LDL-C ≥130 mg/dL (3.4 mmol/L),[2,11] and severe lipid abnormalities such as the heterozygous familial hypercholesterolemia (HeFH) phenotype affect 1 in 250 to 300 individuals.[18–20]

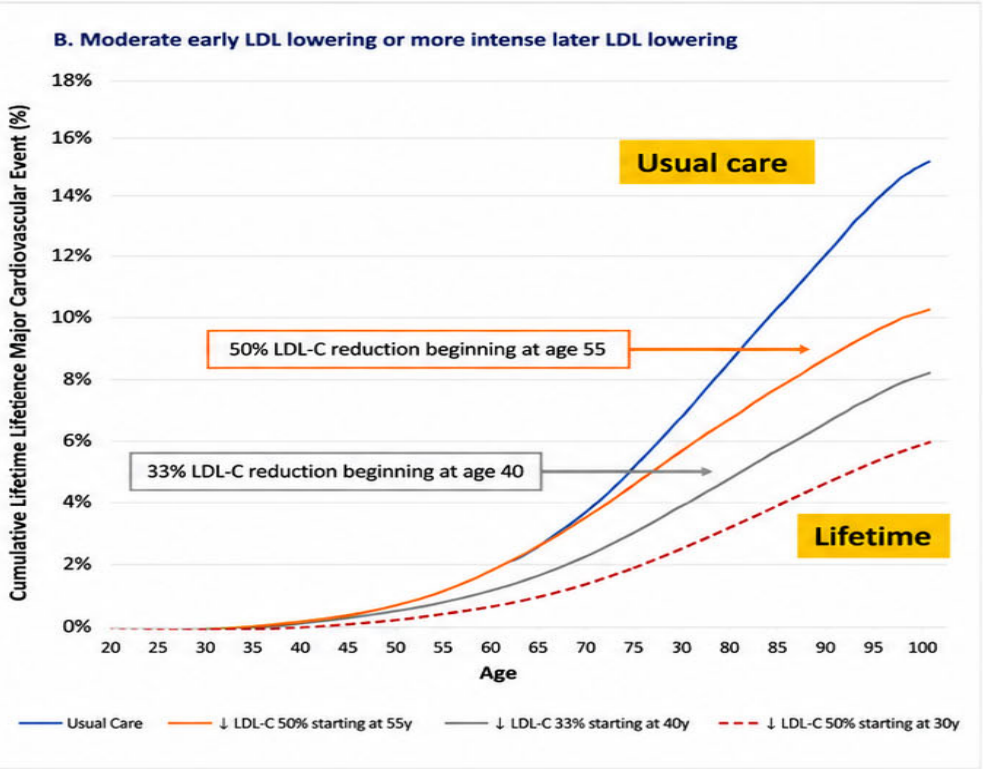


As Theodore Roosevelt said,

"Old age is like everything else. To make a success of it, you've got to start young."

Benefit of reducing cumulative exposure to LDL-C on the lifetime risk of ASCVD–Starting just a decade early!





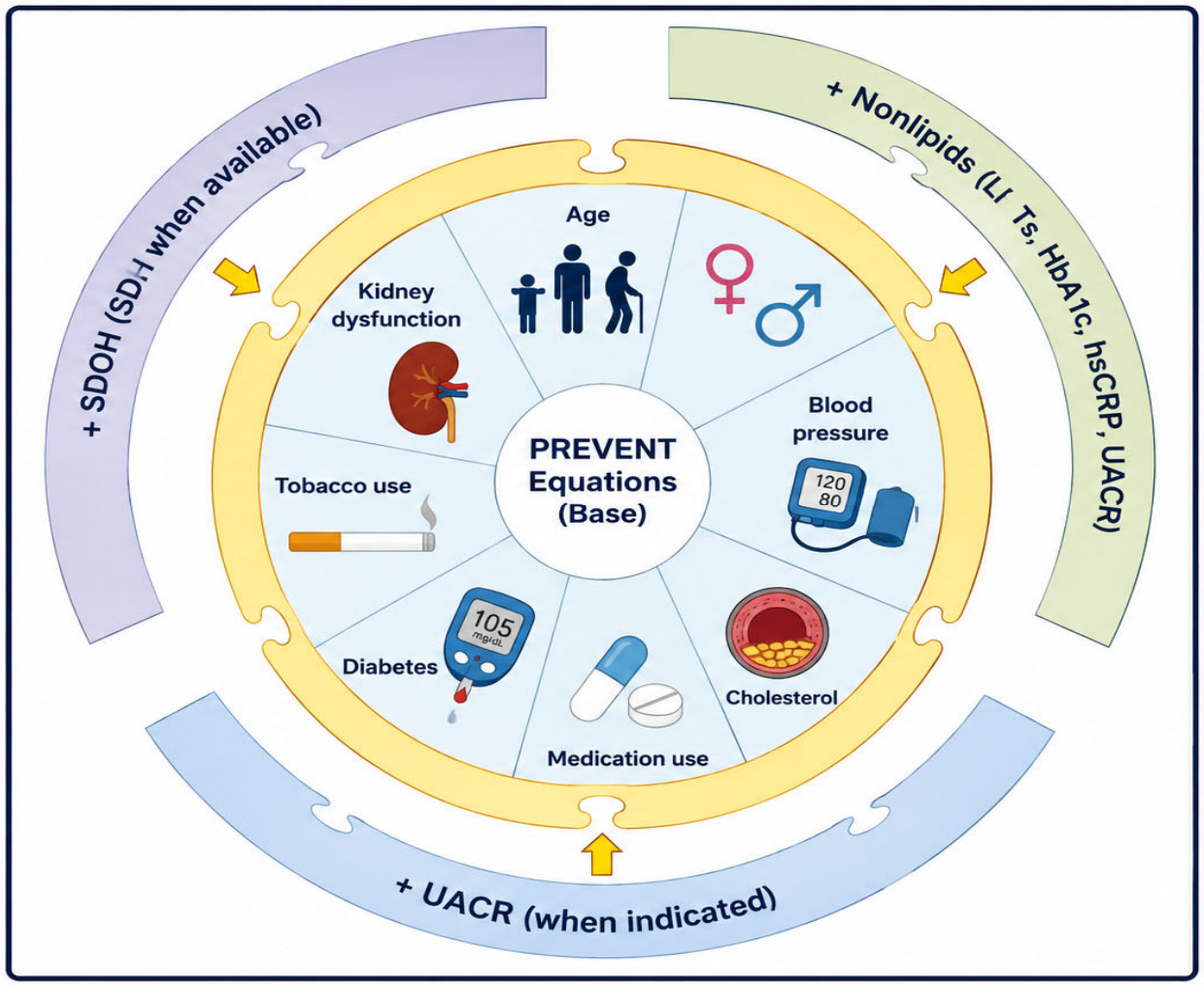
Use PREVENT-ASCVD for Risk Assessment

2a

- **C**alculate Risk (if no ASCVD or FH)
  - 10-yr ASCVD risk if age 30-79
  - 30-yr ASCVD risk if age 30-59
- **P**ersonalize risk assessment
  - Evaluate Risk Enhancers
  - CAC if LLT decision uncertain
- **R**eassess the need for lipid-lowering therapy

PCE risk estimates were known to over-estimate 10-year risk. PREVENT-ASCVD 10-y risk estimates are generally lower than PCE results. Approximate equivalent ranges for PCE and PREVENT ASCVD are shown here:

10-yr risk Category	PCE	PREVENT ASCVD
Low	<5%	<3%
Borderline	5-<7.5%	3-<5%
Intermediate	7.5-<20%	5-<10%
High	≥20%	>-10%


+ SDOH (SDH when available)
+ Nonlipids (LDL-C, TG, HbA1c, hsCRP, UACR)
+ UACR (when indicated)
Age
Kidney dysfunction
Blood pressure
Tobacco use
PREVENT Equations (Base)
Diabetes
Cholesterol
Medication use
120
80
105
mg/dL

2b ASCVD Risk Enhancers to Guide Primary Prevention Risk Assessment

ASCVD Risk Enhancers	
Demographic, Genetic and Family History	**Family history of premature ASCVD** *Sibling or parent <55y if M, <65y if F* **Higher risk ancestry** *South Asian, Filipino* **High polygenic risk**
Clinical Conditions that Increase ASCVD Risk	**Chronic inflammatory diseases** *Systemic lupus, inflammatory arthritis, psoriasis* **CKM Syndrome** **Reproductive risk markers** *Early menopause (<45 y), preeclampsia, gestational DM*
Biomarkers	**hsCRP ≥2 mg/L** *on >1 measurement* **Elevated TGs** *≥150 mg/dL fasting, ≥175 mg/dL nonfasting* **Persistently elevated LDL-C (≥160 mg/dL) , non-HDL-C (≥190 mg/dL) or ApoB (≥120 mg/dL)**

If present, risk enhancers favor statin therapy in borderline to intermediate risk individuals (10-year ASCVD risk 3-<10%)

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3

LDL-Lowering in Primary Prevention: Risk-Guided Decision Making

Based on 10-Year PREVENT-ASCVD Risk Estimates

Low Risk (<5%)

10-Year ASCVD Risk

High Risk (≥10%)

BORDERLINE RISK: 3% to <5%

LDL-lowering therapy can be considered based on clinical judgment.

INTERMEDIATE RISK: 5% to <10%

LDL-lowering therapy should be considered following a thorough clinician–patient discussion weighing benefits, risks, and patient preferences.

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4/5

LDL-C & Non-HDL-C Goals are Back + apoB Goals are Included

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TC - HDL-C

	Patient Examples	LDL-C mg/dL	Non-HDL-C mg/dL	ApoB mg/dL
Borderline to Intermediate Risk	• 3-<5% or 5-<10% 10y ASCVD Risk • <3%10y ASCVD risk + (LDL-C ≥160 mg/dL or Fam Hx or 30y risk ≥10%) • T2DM without ASCVD or Risk Factors • CAC 1-99 or mild incidental CAC	<100	<130	<90
High Risk	• >10% 10-yr ASCVD Risk • T2DM + Risk Factors • HeFH without ASCVD • CAC ≥100 – 999 AU • Moderate to severe incidental CAC	<70	<100	<70
Very High Risk	• ASCVD (optional if not very-high risk) • CAC ≥1000 AU (optional if ≥300 AU)	<55	<85	<55

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5

Use ApoB to Assess Residual Lipid-Related Risk

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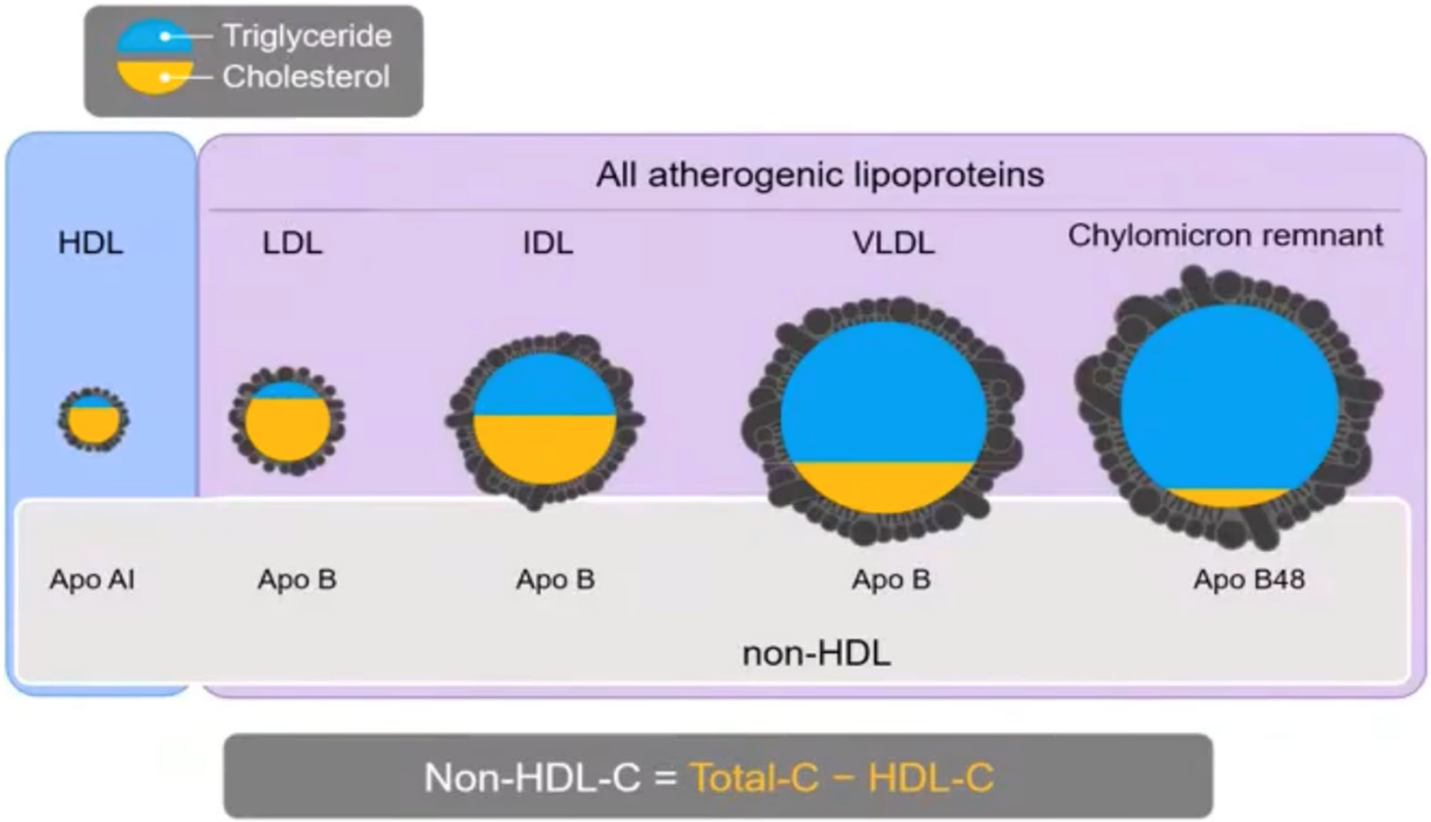
Consider ApoB when

- LDL-C and non-HDL-C goals are met, especially in patients with:
  - Cardiovascular-Kidney-Metabolic syndrome
  - Diabetes
  - TG ≥200 mg/dL
- If ApoB above goal, treatment should be intensified

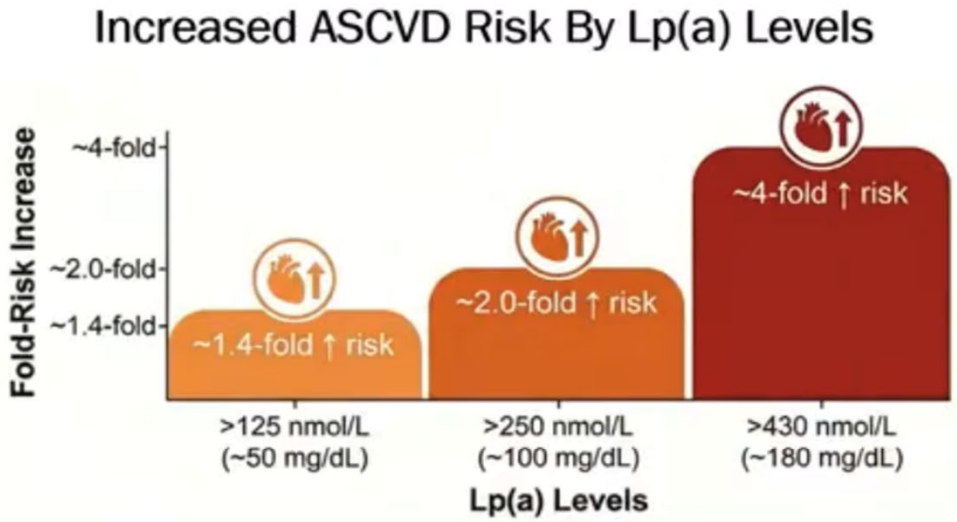
About ApoB:
All atherogenic lipoproteins have a single apoB100 protein. ApoB reflects the total number of atherogenic lipoproteins. ApoB is not affected by fasting.

Understanding Non-HDL-C



Non-HDL-C = Total-C − HDL-C

6 Measure Lp(a) Once in All Adults



About Lp(a):
Lp(a) particles consist of an LDL particle bound to an apo(a) protein. Lp(a) levels largely genetically determined and mostly stable over a lifetime. Repeat testing is generally not needed.

If Lp(a) is elevated, LDL-C lowering should be intensified and other ASCVD risk factors aggressively controlled

CAC-Score Guided Treatment of Subclinical Atherosclerosis

7

CAC Scoring Recommended if Borderline (3-<5%) or intermediate (5-<10%) 10y ASCVD Risk in Men ≥40, Women ≥45 y.o.

The importance of %ile esp. in young e.g., CARDIA

- 0 AU
  - LDL-C goal <100 mg/dL
- 1-99 AU or <75th %ile
  - LDL-C goal <70 mg/dL
- 100-299 AU Or ≥75th%ile
  - LDL-C goal <70 mg/dL, optional <55 mg/dL
- 300-999 AU
  - LDL-C goal <55 mg/dL
- 1000 AU

Incidental CAC on a nongated CT scan can also be used to guide therapy. If mild, LDL-C goal <100 mg/dL. If moderate or severe, LDL-C goal is <70 mg/dL. Absence of incidental CAC should not be used to defer therapy given the low negative predictive value of nongated scans (FNR 24%)

8 Special Considerations in Therapy

CKD
Stage 3-4
All Ages

HIV
Age ≥40

Diabetes
Age ≥40

Statin Therapy Recommended if LDL-C ≥70 mg/dL

Cancer

Continue lipid-lowering therapy unless contraindicated

Pregnancy

Consult a dietitian and defer most LLT

If TG are ≥500 mg/dL (after 1st trimester), consider fibrate or omega-3 FA

9

Lipid-Lowering Therapy Beyond Statins

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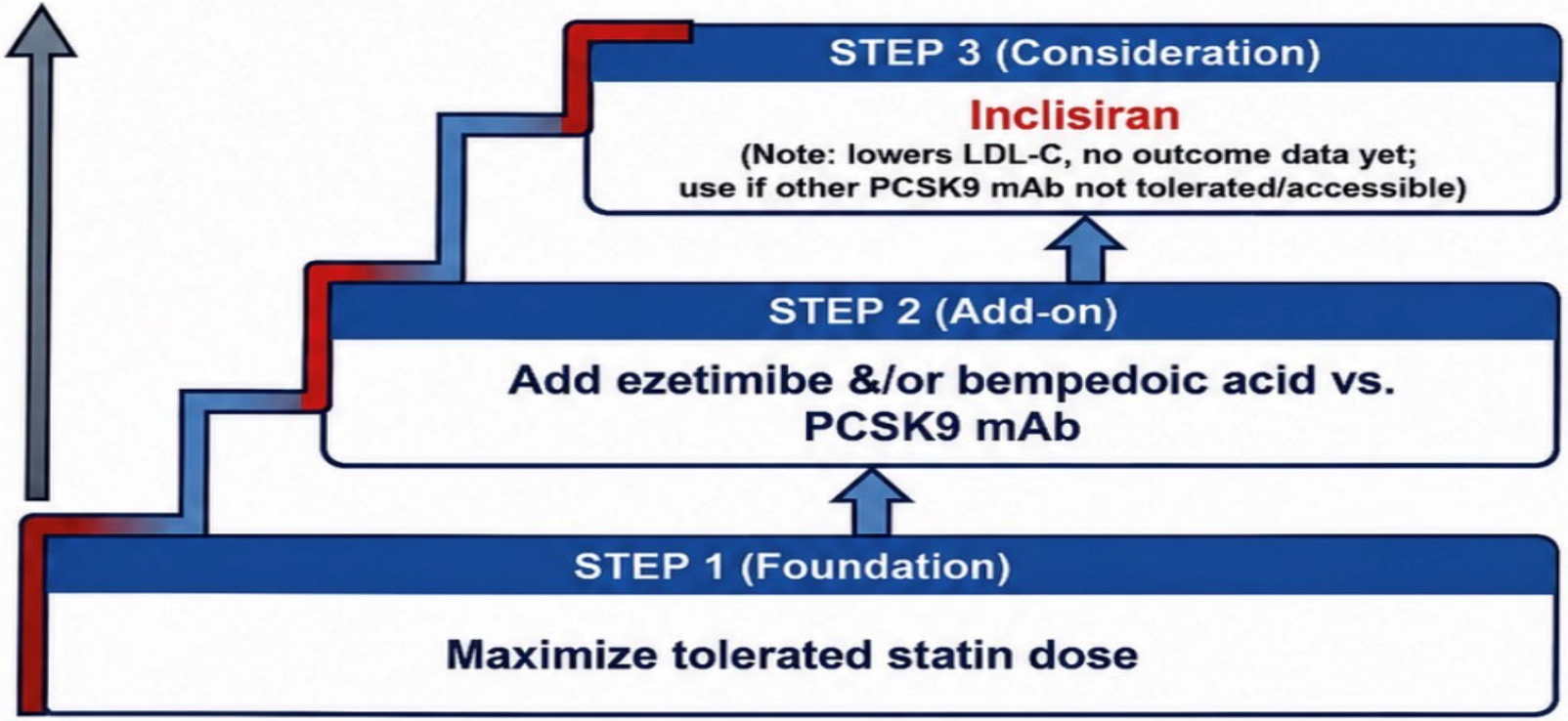

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	Mechanism	Evidence / Key Benefit	Dose	Frequency	Route	Key Takeaway
Cholesterol absorption inhibitor 	Blocks the sterol transporter (NPC1L1) in intestine and liver → ↓ LDL receptor expression on hepatocytes	**Ezetimibe**	10 mg	Once daily	Oral	• CVOT evidence shows reduced cardiovascular events in very high-risk secondary prevention when added to statin therapy • Expected LDL-C reduction: ~18% (mono); ~25% (add-on) • Drug of choice in statin intolerance
PCSK9 inhibitor – monoclonal antibodies 	Binds to PCSK9 in the circulation → ↑ LDL receptor recycling	**Alirocumab**	Alirocumab: 75–150 mg or 300 mg	Alirocumab: Every 2 wk or 4 wk	Subcutaneous (SC)	• Reduces CV events in very high-risk patients on statin ± ezetimibe • Expected LDL-C reduction: ~45–64% • Lower mean LDL-C reduction (21–31%) in homozygous FH due to LDL*R* gene variants*
		Evolocumab	Evolocumab: 140 mg	Evolocumab: Every 2 wk		
ATP citrate lyase inhibitor 	Inhibits ATP citrate lyase in the liver → ↓ cholesterol and fatty acid synthesis, ↓ LDL receptor expression on hepatocytes	**Bempedoic acid**	180 mg	Once daily	Oral	• CVOT evidence shows reduced CV events in statin-intolerant patients at high risk (primary & secondary prevention) • Prodrug activated in liver → lower muscle-related side effects • Expected LDL-C reduction: ~17–18%
PCSK9 inhibitor – small interfering RNA (siRNA) 	Silences PCSK9 synthesis in liver via RNA interference → ↓ PCSK9 and ↑ LDL receptor	**Inclisiran**	284 mg	Initial dose, again at 3 mo; then every 6 mo	Subcutaneous (SC)	• Administered by healthcare professional • Expected LDL-C reduction: ~48–52% • CVOT in progress

* LDL-C lowering therapy should be individualized based on patient factors and clinical judgment.

† Percent reduction in LDL-C when compared with baseline.

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10a

Dietary Interventions for Hypertriglyceridemia

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Lifestyle is First Line for All With Elevated TGs:

≥5% weight loss, ≥150 min/week moderate intensity activity

Implemented shared decision-making intervention	TG ≥150 to 499 mg/dL* (1.7 to <5.7 mmol/L)	TG ≥500 to 999 mg/dL* (5.7 to <11.3 mmol/L)	TG ≥1000 mg/dL† (≥11.3 mmol/L)
Added sugars (percent calories)	<6%	<5%	Eliminate
Total fat (percent calories)	30%–35%	20%–25%‡	10%–15%§¶
Alcohol	Avoid	Abstain completely	Abstain completely
Physical activity	At least 150 minutes/week of accumulated moderate-intensity or 75 minutes/week of vigorous intensity aerobic activity (or equivalent combination of both) and 2 days/week of upper and lower body resistance exercise‖		
Weight loss (percent body weight)	Recommended weight loss goal of 5%–10% for patients who are overweight or obese with elevated TG		

Virani SS et al. *J Am Coll Cardiol*. 2021 Aug 31;78(9):960-993

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10b

Pharmacologic Approach to Hypertriglyceridemia

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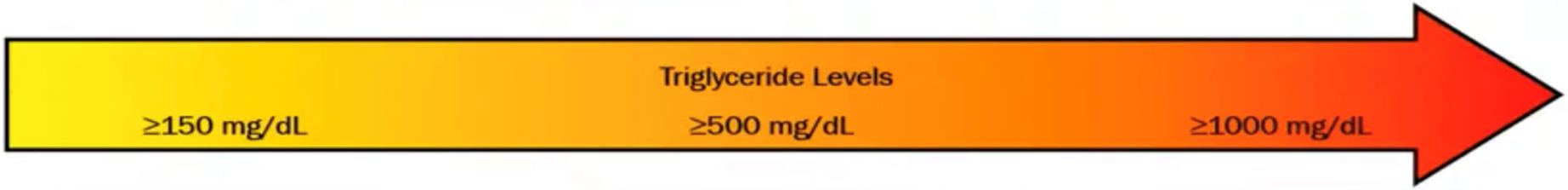
Treatment of Hypertriglyceridemia based on ASCVD and Pancreatitis Risk



Increased ASCVD Risk

Statins first-line to reduce ASCVD Risk.

Use non-HDL-C and ApoB to guide lipid lowering therapy.

Increased Risk of Pancreatitis

Add Omega-3 FA (EPA) and/or fibrate after statin to lower pancreatitis risk.

Possible Familial Chylomicronemia

Very high pancreatitis risk. Refer to lipid specialist. Consider olezarsen (GalNac Conjugated Apo C-III ASO) if TGs persistently elevated after other Rx.

Key Take-aways

- **Start Early!**
- Use **PREVENT** Risk Assessment tool to guide preventive strategies.
- **ASCVD Risk** guided statin therapy (3,5,10) in the context of risk enhancers – Use CAC if uncertainty. Remember CPR!
- **LDL-C** and **non-HDL-C** goals are back. Use **ApoB** once LDL-C/non-HDL-C goals are met in select individuals.
- Measure **Lp(a)** once in all adults.
- Remember **LDL-C/non-HDL-C** goals in patients with established **ASCVD**.
- Maximally tolerated statin therapy → **ezetimibe-bempedoic acid-PCSK9mAb** (Inclisiran if PCSK9 mAbs not tolerated or patient preference).
- Remember increased **ASCVD** risk in those with elevated **TGs**. Diet, statins, IPE, fibrates, Apo C3i in FCS.