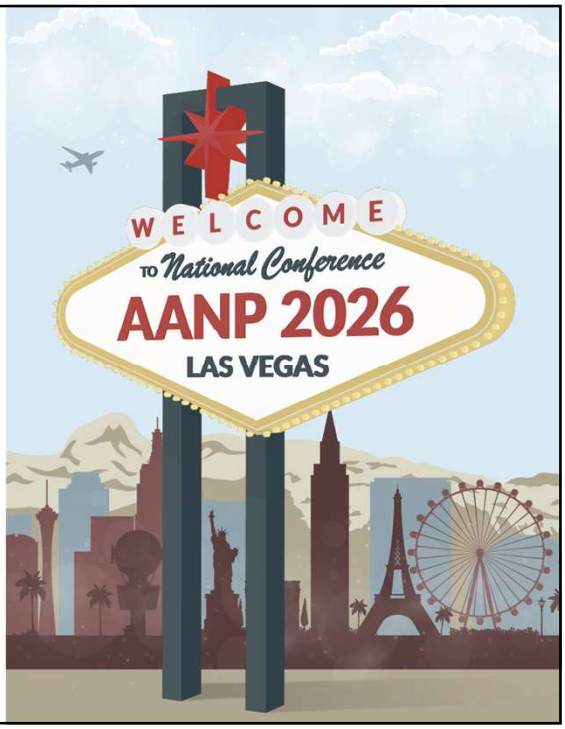
EVOLVING LIPID GUIDELINES: PRACTICAL APPLICATIONS

Carrie Palmer, DNP, RN, ANP-BC
The University of North Carolina at Chapel Hill
Schools of Nursing and Medicine



1

DISCLOSURES

- **Artificial Intelligence Use Disclosure**
  Artificial intelligence tools were used to assist with literature synthesis and presentation development. All content has been independently reviewed, edited, and validated by the presenter to ensure accuracy, appropriate citation of sources, and alignment with current clinical guidelines.

2

OBJECTIVES

- Summarize key updates in contemporary lipid guidelines relevant to primary and secondary cardiovascular prevention.
- Apply ASCVD risk assessment tools, risk enhancers, lipid-related laboratory markers, and coronary artery calcium (CAC) scoring to guide individualized lipid management decisions.
- Differentiate statin and nonstatin lipid-lowering therapies based on mechanism of action, patient risk profile, and guideline-directed treatment goals.
- Develop evidence-based lipid management strategies using lifestyle interventions, pharmacotherapy, shared decision-making, and stepwise treatment intensification.



3

HOT OFF THE PRESS!

CLINICAL PRACTICE GUIDELINES

2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/I Guideline on the Management of Dyslipidemia: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

2026 ACC/AHA Multisociety Guideline on the Management of Dyslipidemia. *Circulation.* 2026. doi:10.1161/CIR.0000000000001423



4

CORE PRINCIPLES THAT REMAIN UNCHANGED

- Lifestyle management remains foundational
- LDL-C remains the primary diagnostic and treatment target
- ASCVD risk drives treatment decisions
- Primary and secondary prevention targets
- Risk enhancers refine treatment decisions
- Statins remain first-line medications
- Stepwise intensification of therapy based on LDL-C goals
- Shared decision-making remains essential

5

KEY ADDITIONS IN THE 2026 DYSLIPIDEMIA GUIDELINE

Stronger recommendations for non-statin therapy
Look beyond LDL-C: non-HDL-C, apoB, Lp(A)
Risk-based LDL-C targets
No recommended dietary supplements for lipid management
PREVENT™ ASCVD risk equations
Referral recommendations

6

KEY ADDITIONS IN THE 2026 DYSLIPIDEMIA GUIDELINE

- Reproductive risk markers
- Coronary artery calcium (CAC) scoring
- Risk refinement in primary prevention
- Earlier assessment of response to therapy
- Earlier escalation of therapy
- Genetic testing for familial hypercholesterolemia
- Special populations

7

WELCOME TO National Conference AANP 2026 LAS VEGAS

IDENTIFY AND TREAT DYSLIPIDEMIA EARLY TO REDUCE LIFETIME RISK

8

RATIONALE FOR EARLY TREATMENT

- Atherosclerosis develops over decades
- Early lipid screening identifies patients at risk for ASCVD
- Earlier intervention lowers cumulative LDL exposure
- Lower lifetime LDL burden → lower ASCVD risk



9

CASE STUDY - VIRGINIA

Presentation

- Virginia is a 54-year-old female presenting for her annual exam
- Past medical history: HTN, prediabetes, class I obesity, former smoker (quit in 2018)
- Family history: Premature ASCVD
  - Father had MI at age 52
- Medications: Olmesartan-HCT 40/25 mg daily
- BP: 124/82

Lab Assessment

- TC: 242
- LDL-C: 158
- HDL-C: 42
- TG: 210
- Non-HDL-C: 200
- A1c: 6.1%



10

TREATING EARLY BEGINS WITH SCREENING

- Adults: Standard lipid profile beginning at 19 years, repeat every 5 years
- Children 9-11: Standard lipid profile once to identify genetic or severe dyslipidemias
- Individuals with first- or second-degree relatives with premature ASCVD, severe hypercholesterolemia, familial hypercholesterolemia – Standard lipid profile beginning at age 2
- Fasting lipid panel to measure residual risk in those treated for dyslipidemia

11

PRIMARY PREVENTION GROUPS

Adults age 30-79 with LDL-C 70-189

- PREVENT Score and risk refinement

LDL-C ≥190

- High-intensity statin therapy

Adults with diabetes, CKD, HIV

- At least moderate-intensity statin therapy

12

CPR MODEL IN PRIMARY PREVENTION

- C – Calculate
  - PREVENT-ASCVD™ equation
- P – Personalize risk
  - Risk enhancers
  - CKM syndrome
  - Lp(a)
  - ApoB
- R – Reclassify risk
  - CAC score when risk/benefit is uncertain



13

POOLED COHORT EQUATIONS VS PREVENT™ RISK EQUATIONS

Pooled Cohort Equations (PCE)	PREVENT™ Equations
Estimate 10-year risk of ASCVD	Estimate 10-year and 30-year cardiovascular risk
40–79 years	30–79 years
ASCVD events (MI, stroke, ASCVD death)	Cardiovascular outcomes including ASCVD, heart failure, and cardiovascular death
Age, sex, race, total cholesterol, HDL, systolic BP, HTN treatment, diabetes, smoking	Traditional factors plus additional cardiometabolic variables (e.g., kidney function, BMI)
Includes race-specific equations	Removes race as a variable



14

RISK ENHANCERS

- History of premature ASCVD in parent or sibling
- Higher risk ancestry – South Asian, African American/Black, Hispanic/Latino, Native American
- High polygenic risk
- Chronic inflammatory diseases
- Lp(a) ≥125 nmol/L or 50 mg/dL
- hsCRP ≥2 mg/L on >1 occasion without explanation
- TG persistently ≥175 mg/dL nonfasting or ≥150 mg/dL fasting
- CKM syndrome
- LDL-C persistently ≥160–189 mg/dL, non–HDL-C ≥190–219 mg/dL, or apoB ≥120 mg/dL
- Reproductive risk markers

15

STATIN INTENSITY

High-Intensity Statins (LDL ↓ ≥50%)	Moderate-Intensity Statins (LDL ↓ 30–49%)
• Atorvastatin 40–80 mg • Rosuvastatin 20–40 mg	• Atorvastatin 10–20 mg • Rosuvastatin 5–10 mg • Simvastatin 20–40 mg • Pravastatin 40–80 mg • Lovastatin 40 mg • Fluvastatin XL 80 mg • Pitavastatin 2–4 mg

16

10-Year Risk (PREVENT™)	Risk Category	Recommended Approach
<3%	Low risk	Lifestyle optimization
3-5%	Borderline risk	Assess risk enhancers, consider CAC score; lifestyle optimization, consider statin based on risk assessment
5-<10%	Intermediate risk	Initiate moderate-intensity statin after clinician–patient discussion; consider CAC to refine risk
≥10%	High risk	Initiate high-intensity statin to achieve ≥50% LDL-C reduction and LDL-C goal <70
LDL-C ≥190 mg/dL	Severe hypercholesterolemia	High-intensity statin regardless of calculated risk
Diabetes age 40–75	High-risk condition	At least moderate-intensity statin; consider high-intensity if multiple risk factors

17

PRIMARY PREVENTION IN ADULTS WITH DIABETES

Adults age 40-75 with diabetes
- Moderate-intensity statin
- LDL-C goal of <100

Adults age 40-75 with diabetes and multiple ASCVD risk factors
- High-intensity statin
- LDL-C goal <70

Adults age 40-75 with diabetes and ASCVD risk estimate ≥10%
- Consider adding ezetimibe or PCSK9i

Adults age 20-39 with T2DM ≥10 years or T1DM ≥20 years or diabetes complications
- Moderate intensity statin

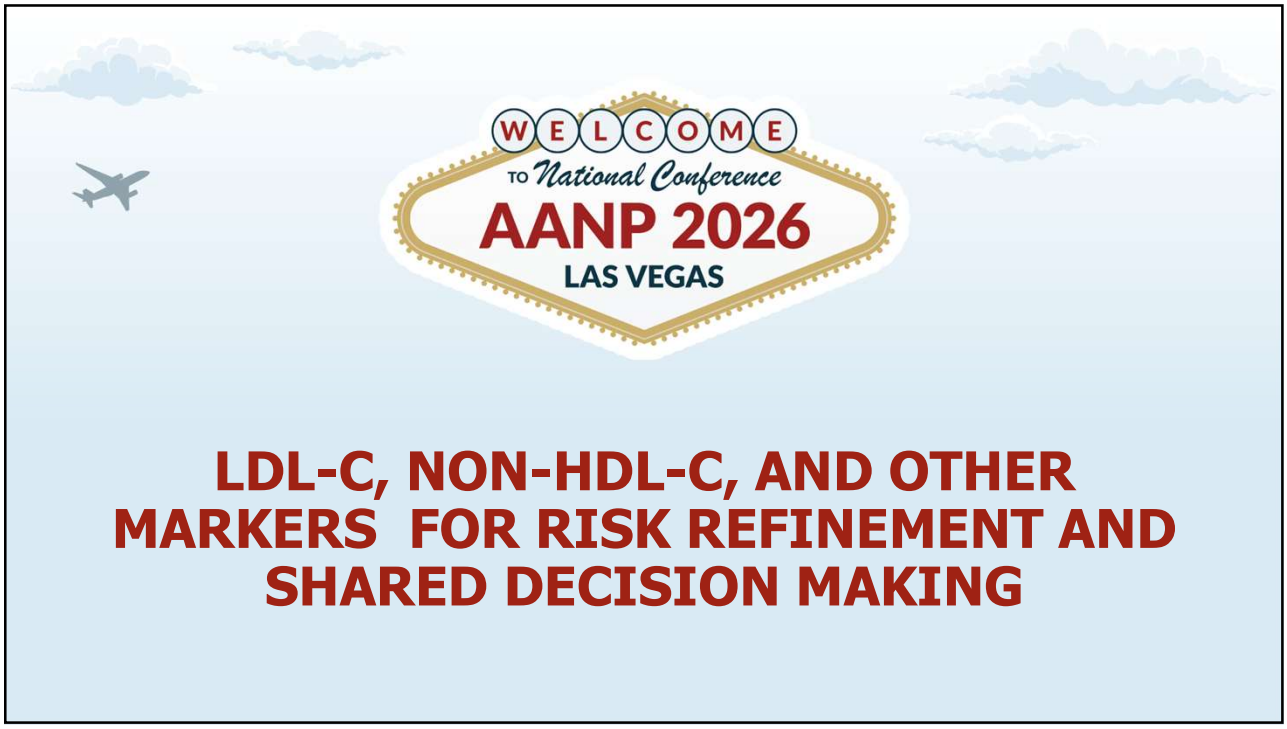
18

CASE STUDY: VIRGINIA

- PREVENT Estimate: Intermediate
  - 10-year: 6.7%
  - 30-year: 31.1%
- Risk Enhancer: Family history of premature ASCVD
- Treatment recommendation: Moderate-intensity statin
- "I really don't want to take a statin unless I absolutely need it."



19



LDL-C, NON-HDL-C, AND OTHER MARKERS FOR RISK REFINEMENT AND SHARED DECISION MAKING

20

LDL-C TARGETS FOR PRIMARY PREVENTION

Risk Category	ACC/AHA Decision Thresholds
Borderline Risk (3-5% 10-year risk)	Moderate-intensity statin to lower LDL-C ≥30% LDL-C <100 Non-HDL-C <130
Intermediate Risk (5-<10% 10-year risk)	Moderate-intensity statin to lower LDL-C ≥30% LDL-C <100 Non-HDL-C <130
High Risk (≥10% 10-year risk)	High-intensity statin to lower LDL-C ≥50% LDL-C <70 Non-HDL-C <100



21

NON-HDL-C

- All atherogenic lipoproteins:
  - LDL, VLDL, IDL, Lp(a), remnant lipoproteins
- Secondary target or alternative marker
- Used when:
  - Triglycerides are elevated
  - Atherogenic particles elevated despite LDL-C control – residual risk
- Non-HDL-C goal is 30 mg/dL higher than LDL-C goal



22

APOLIPOPROTEIN B

- Definition: Measurement of atherogenic lipoprotein particles
  - One apoB molecule per LDL, VLDL, and Lp(a) particle
  - Normal: <90 mg/dL
- Quantifies residual atherogenic risk after LDL-C treatment
- More accurate assessment of ASCVD risk when LDL-C and apoB are discordant
- Particularly useful in patients with:
  - ASCVD
  - CKM syndrome
  - Diabetes
  - Elevated triglycerides
- May help guide treatment decisions in patients with low or borderline ASCVD risk



23

LIPOPROTEIN(A) – LP(A)

- Definition – lipoprotein like LDL that carries a single apolipoprotein
- Improves identification of high-risk patients
- Concentrations are genetically determined and remain stable despite lifestyle modifications and statin therapy
- Elevated Lp(a) - ≥125 nmol/L (50 mg/dL) – increased ASCVD risk, aortic valve disease, and all cause mortality
- Cascade testing – measure in all people ≥2 years old with a first degree relative with elevated Lp(a)



24

CORONARY ARTERY CALCIUM (CAC) SCORE

- CAC – calcified plaques within coronary arteries
  - Indicates subclinical disease
  - Measured by noninvasive CT scan
- CAC Score – burden of CAC
- CAC Percentile – reference range
- Can reclassify ASCVD risk prediction in adults with borderline or intermediate 10-year risk
- Useful in shared decision-making



25

CAC Score	Interpretation	Clinical Implication
0	Absent - No detectable coronary calcium	Very low short-term ASCVD risk
1-9	Minimal	Lifestyle optimization
10–99	Mild plaque burden	Increased risk; consider preventive therapy
100–299	Moderate plaque burden	Higher risk; statin therapy usually recommended
300-999	High plaque burden	High ASCVD risk; aggressive risk reduction
>1000	Extensive plaque burden	High ASCVD risk; aggressive risk reduction

26

CAC PERCENTILE

- Compares CAC score with score distribution for same age, gender, race
  - Relative plaque burden compared to peers
  - Can identify premature atherosclerosis
- Used in conjunction with CAC score:
  - CAC = 30 in a 40-year-old → high percentile
  - CAC = 30 in a 75-year-old → lower percentile
- ≥ 75th percentile – consider lipid-lowering therapy
- ≥ 100th percentile – greatest benefit from statin therapy

27

WHEN TO OBTAIN CAC-SCORE

- To guide shared-decision making decisions about statins in borderline or intermediate risk patients without substantial risk enhancers
- Uncertain risk estimate or statin benefit
- Statin hesitancy
- NOT useful in:
  - Known ASCVD
  - LDL-C ≥190
  - High ASCVD risk
  - Very low-risk patients (<3% 10-year risk)
  - Adults <30

28

CASE STUDY: VIRGINIA

Initial recommendation:
- Intermediate 10-year risk → moderate intensity statin, 30% reduction in LDL-C

Risk refinement:
- CAC Score – 145
- CAC Percentile: 85th

Refined approach:
- High-intensity statin with LDL-C goal <70



29

CASE STUDY: VIRGINIA

Lifestyle modifications:	Pharmacotherapy:	Target:	Monitoring:
• Mediterranean diet • Weight reduction	• Rosuvastatin 20 mg	• LDL-C <70, non-HDL-C <100	• Lipid panel in 4-12 weeks • Assess reduction in LDL-C and augment therapy if not at goal



30

WELCOME TO National Conference AANP 2026 LAS VEGAS

IDENTIFY PATIENTS WHO NEED AGGRESSIVE LDL LOWERING

31

CASE STUDY: JOHN

Presentation

- 68-year-old male presenting for chronic disease follow up
- PMH: MI in 2024 with PCI, type 2 diabetes, chronic kidney disease Stage 3a, hypertension
- Medications: atorvastatin 80 mg, aspirin 81 mg, valsartan 160 mg, semaglutide 2 mg, empagliflozin 25 mg,

Labs

- TC: 156
- LDL-C: 82
- HDL-C: 38
- TG: 240
- Non-HDL-C: 118
- A1c: 7.8%
- eGFR: 52

32

INDICATIONS FOR AGGRESSIVE LIPID LOWERING

Secondary prevention

Severe hypercholesterolemia in the absence of ASCVD

High risk for ASCVD

High polygenic risk

Documented atherosclerosis

33

SECONDARY PREVENTION ESSENTIALS

- High-intensity statin therapy to achieve ≥50% reduction in LDL-C
- LDL-C goal with clinical ASCVD
  - Not at very high risk: LDL-C goal <70 mg/dL
  - At very high risk: LDL-C goal <55 mg/dL
- Reassess LDL-C 4–12 weeks after initiating or adjusting therapy
- Add nonstatin therapy if LDL-C goal is not achieved or if the patient cannot tolerate maximally tolerated statin therapy

34

VERY-HIGH RISK CONDITIONS WARRANTING LDL-C GOAL OF <55

History of major ASCVD events:	ASCVD + ≥1 high-risk conditions:
• ACS within the past 12 months • History of MI • History of ischemic stroke • Symptomatic PAD	• >65 years old • Coronary artery revascularization • Current smoker • Diabetes • History oF HF • Hypertension • LDL-C >100 despite appropriate treatment



35

CASE STUDY: JOHN

- LDL-C: 82
- TG: 240
- First-line therapy:
  - High-intensity statin
- Very-high risk conditions
  - ASCVD with MI and cardiac revascularization
  - Diabetes
  - HTN
  - CKD
- Next steps?



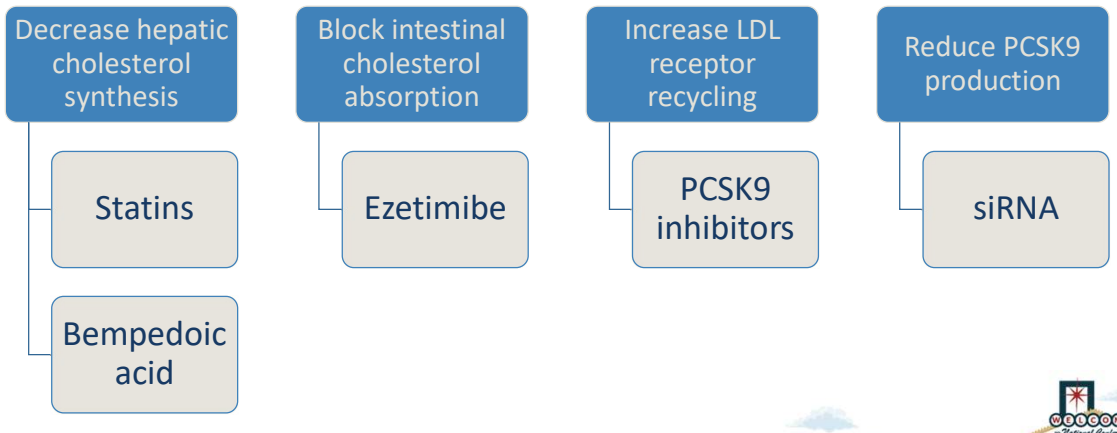
36

NON-STATIN THERAPIES

Medication	LDL-C Reduction	Indications
Ezetimibe	~15–25%	Added when LDL-C remains above goal despite statin therapy
PCSK9 inhibitors Alirocumab Evolocumab	~50–60%	High-risk patients needing additional LDL-C lowering after statin ± ezetimibe
Bempedoic acid	~15–25%	Option for patients needing additional LDL-C lowering or with statin intolerance
siRNA Inclisiran	~45–50%	Alternative/additional therapy in patients needing further LDL-C lowering

37

MECHANISMS OF LDL-C-LOWERING DRUGS



38

CASE STUDY: JOHN

- LDL-C not at goal
  - Initiate ezetimibe 10 mg
- TG elevated at 240
- Monitor lipid panel in 4-12 weeks
  - LDL-C in 2 months = 64
  - TG = 190
- Further intensification needed?
  - Consider PCSK9i to achieve LDL-C goal



39



PERSISTENTLY ELEVATED TRIGLYCERIDES WARRANT TREATMENT

40

CASE STUDY: TED

Presentation

- 46-year-old male presented after employer health insurance screening labs
- PMH: Class 2 obesity, type 2 diabetes, hypertension, MASLD
- Social history: 3-4 beers nightly, more on weekends
- Medications: Metformin 1000 mg BID, dulaglutide 3 mg, losartan 100 mg, HCTZ 25 mg daily

Labs

- TC: 278
- HDL-C: 32
- TG: 682
- LDL-C – unable to calculate
- A1c: 8.6%
- ALT: 68

41

ELEVATED TRIGLYCERIDES

Clinical concerns:	Causes:	Stepwise approach:
• ASCVD risk • Pancreatitis risk	• Primary – genetic or inherited • Secondary causes – nongenetic conditions, lifestyle, medications	• Treat secondary causes • Lifestyle therapy is first line • Statins as first-line therapy

42

TRIGLYCERIDES

Classification	Triglycerides (mg/dL)	Clinical Significance
Normal	<150 mg/dL	Desired level
Borderline high	150–199 mg/dL	Considered a risk-enhancing factor for ASCVD
High	200–499 mg/dL	Associated with increased cardiovascular risk
Very high	≥500 mg/dL	Increased risk of acute pancreatitis



43

SECONDARY CAUSES OF HYPERTRIGLYCERIDEMIA

Metabolic conditions	Lifestyle factors	Medications
• Metabolic syndrome • Type 2 diabetes • Hypothyroidism • CKD / nephrotic syndrome • MASLD	• Alcohol use • High refined carbohydrate intake	• Estrogens • Glucocorticoids • Thiazides • Beta blockers • Atypical antipsychotics • Protease inhibitors



44

CASE STUDY: TED

- Severe hypertriglyceridemia
  - Clinical concern – pancreatitis
- Secondary causes
  - Alcohol use
  - Poorly controlled diabetes
  - MASLD
  - Medications
  - Obesity



45

LIFESTYLE MODIFICATIONS FOR HYPERTRIGLYCERIDEMIA

- Weight loss (5–10%)
- Regular aerobic exercise
- Reduce sugars/refined carbohydrates
- Limit or avoid alcohol
- Follow a heart-healthy diet
- Very low-fat diet if TG ≥500 mg/dL



46

MECHANISMS OF TRIGLYCERIDE-LOWERING THERAPIES

Decrease hepatic triglyceride production	Increase clearance of triglycerides	Reduce triglyceride absorption and production and improve insulin sensitivity
• Statins • Omega-3 fatty acids	• Fibrates • Olezarsen	• Weight loss • Reduced refined carb intake • Reduced alcohol intake

47

TRIGLYCERIDE LOWERING AGENTS

Medication	TG Reduction	Indications
Fibrates Gemfibrozil Fenofibrate	~30-50%	Reduce pancreatitis risk
Omega-3 Fatty Acids Icosapent Omega-3-acid ethyl esters	~20-30%	Adjunct to statin to reduce ASCVD risk in adults with known ASCVD or diabetes + ≥2 risk factors with TG ≥150 **only Icosapent is recommended for TG treatment*
Olezarsen	~49-65%	Familial chylomicronemia syndrome

48

CASE STUDY: TED

Lifestyle modifications:	Medication changes:	Follow up in 3 months
• Stop alcohol • Reduce carbohydrates • Weight reduction	• Start high-intensity statin • Start fenofibrate • Consider stopping HCTZ and starting ARB or ACEi	• Assess lipids • Assess diabetes control • Assess lifestyle adherence



49

CASE STUDY: TED

Lab	Before	After
TG	682	248
A1c	8.6%	7.2%
BMI	36	32

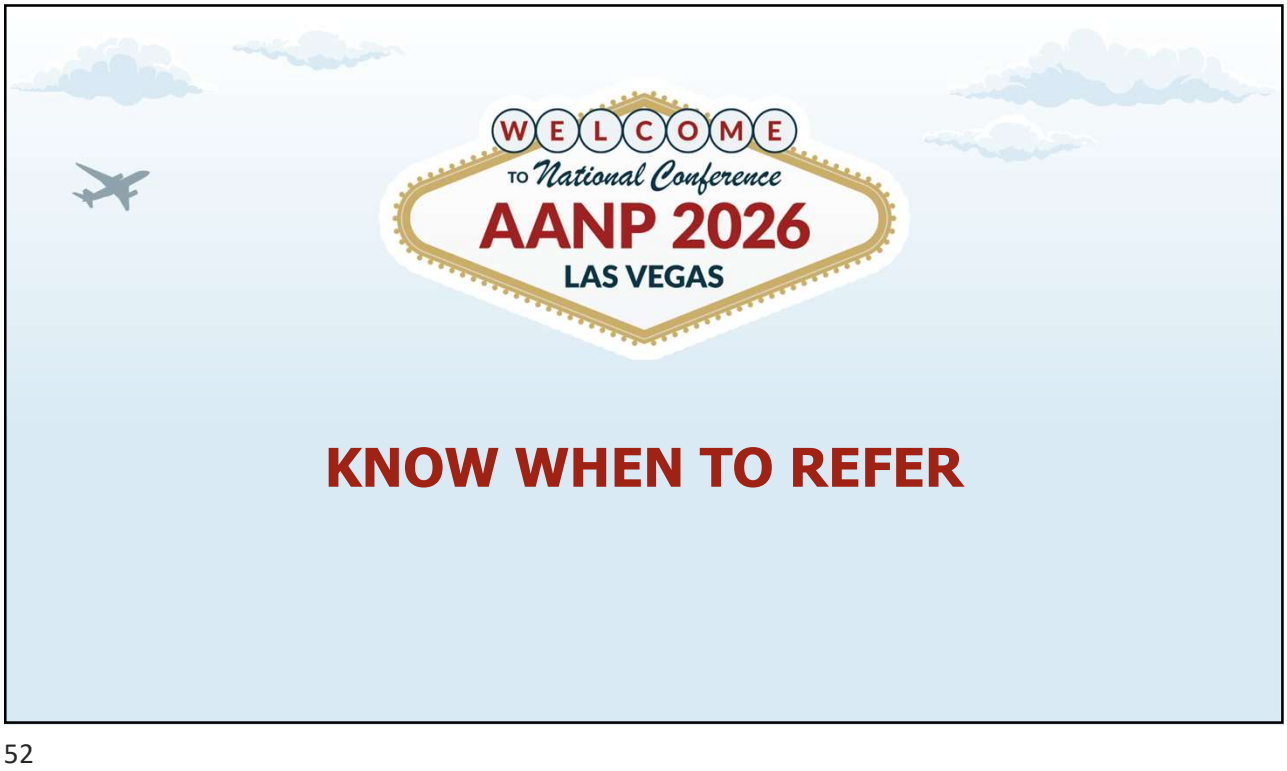


50

CASE STUDY: TED

High triglycerides	Secondary causes	Next steps:
• Clinical concern – ASCVD risk	• Alcohol use - minimized • Diabetes control improved • MASLD • Weight reduction	• Continue fenofibrate and high-intensity statin • Continue lifestyle modifications • Recheck in 3 months

51



KNOW WHEN TO REFER

52

WHEN TO REFER

- Familial lipid disorders
- Premature ASCVD (age <40 years)
- Severe or very high triglycerides
- *Intolerance to lipid-lowering therapies*
- Markedly elevated Lp(a)
- Complex medication regimens (e.g., HIV therapy, cancer treatment, transplant immunosuppression)
- High-risk patients requiring lipid management during pre-conception, pregnancy, or lactation



53

WHEN TO REFER

Registered Dietitian	Severe hypertriglyceridemia, obesity, diabetes, lifestyle counseling needs
Lipid Specialist	Statin intolerance, FH, refractory LDL elevation, complex medication regimens
Preventive Cardiology	Premature ASCVD, advanced risk refinement, persistent residual risk
Endocrinology	Severe mixed dyslipidemia with diabetes/metabolic disease
Genetics	Suspected inherited lipid disorders



54

STATIN INTOLERANCE

- Adverse effects or lab abnormalities that:
  - Occur within weeks to months of starting statin
  - Resolve with statin discontinuation
  - Recur with reintroduction of statin
- Common symptoms:
  - Statin-Associated Muscle Symptoms: Muscle pain, weakness, fatigue
  - Symmetric, proximal muscle groups
- Uncommon symptoms:
  - Elevated transaminases or CK
  - Rhabdomyolysis



55

APPROACH TO STATIN INTOLERANCE

1. Confirm symptoms and severity
2. Hold statin for 2-4 weeks
3. Rechallenge with lower dose or another statin
4. If symptoms recur, trial alternate dosing
5. Add non-statin therapy to achieve LDL-C goal



56

LABELING STATIN INTOLERANCE

Inability to tolerate at least 2 statins at lowest daily dose

Symptoms resolve with discontinuation and recur with rechallenge



57

TAKE-HOME POINTS

LDL-C is the primary target, non–HDL-C, apoB, and Lp(a) refine risk assessment
Individualize risk assessment with PREVENT™ equation, evaluation of risk enhancers, and CAC scoring
Earlier and more aggressive LDL-C lowering for high-risk and very high-risk patients
Statins remain first-line therapy, but nonstatin agents should be used to meet LDL-C goals
Severe hypertriglyceridemia requires evaluation for secondary causes and treatment to reduce pancreatitis and ASCVD risk
Statin intolerance should be confirmed, and maximally-tolerated dose should be provided if warranted
Shared decision-making, lifestyle counseling, and follow-up are essential components for lipid management



58

REFERENCES

- American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. (2026). 2026 guideline for the management of dyslipidemia. *Circulation*. https://doi.org/10.1161/CIR.0000000000001423
- Cheeley, M. K., Saseen, J. J., Agarwala, A., Ravilla, S., Ciffone, N., Jacobson, T. A., Dixon, D. L., & Maki, K. C. (2022). *NLA scientific statement on statin intolerance: A new definition and key considerations for ASCVD risk reduction in the statin intolerant patient*. *Journal of Clinical Lipidology, 16*(4), 361–375. https://doi.org/10.1016/j.jacl.2022.05.068
- Grundy, S. M., Stone, N. J., Bailey, A. L., Beam, C., Birtcher, K. K., Blumenthal, R. S., … Virani, S. S. (2021). 2021 ACC expert consensus decision pathway on the management of hypertriglyceridemia. *Journal of the American College of Cardiology, 78*(9), 960–993. https://doi.org/10.1016/j.jacc.2021.06.011
- Khan, S. S., Matsushita, K., Sang, Y., Ballew, S. H., Islam, S. S., Jia, X., … Lloyd-Jones, D. M. (2024). Development and validation of the American Heart Association PREVENT equations to estimate risk of cardiovascular disease. *Circulation, 149*(6), 430–449. https://doi.org/10.1161/CIRCULATIONAHA.123.067626
- Mach, F., Koskinas, K. C., Roeters van Lennep, J. E., Tokgözoğlu, L., Badimon, L., Baigent, C., … ESC/EAS Scientific Document Group. (2025). 2025 focused update of the 2019 ESC/EAS guidelines for the management of dyslipidaemias. *Eur Heart Journal, 46*(42), 4359–4378. https://doi.org/10.1093/eurheartj/ehaf190
- Patel, S. B., Wyne, K. L., Afreen, S., Belalcazar, L. M., Bird, M. D., Coles, S., Marrs, J. C., Peng, C. C.-H., Pulipati, V. P., Sultan, S., & Zilbermint, M. (2025). American Association of Clinical Endocrinology clinical practice guideline on pharmacologic management of adults with dyslipidemia. *Endocrine Practice, 31*(2), 236–262. https://doi.org/10.1016/j.eprac.2024.09.016
- Tsimikas, S., Karwatowska-Prokopczuk, E., Gouni-Berthold, I., Tardif, J. C., Baum, S. J., Steinhagen-Thiessen, E., … European Atherosclerosis Society Consensus Panel. (2022). Lipoprotein(a): A genetically determined, causal, and prevalent risk factor for atherosclerotic cardiovascular disease—A European Atherosclerosis Society consensus statement. *European Heart Journal, 43*(39), 3925–3946. https://doi.org/10.1093/eurheartj/ehac361



59