# MyCardioAdvocate™

## The Atherogenic Triad

***When 'normal LDL' hides high-risk particles, insulin resistance, and accelerated atherosclerosis.***

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## Why This Matters

You sit across from your cardiologist. The lipid panel shows LDL-C = 110 mg/dL. 'Your cholesterol is fine,' you are told. You leave reassured. Yet your triglycerides are 200 mg/dL, your HDL is 38 mg/dL, and your waist circumference is 42 inches. You have the atherogenic triad—a phenotype that markedly accelerates atherosclerosis despite 'normal' LDL-C. The triad is not rare. It is the dominant lipid pattern in metabolic syndrome, diabetes, and obesity, affecting millions of people globally. And it is profoundly underrecognized because standard lipid panels and traditional risk assessment tools miss it.

The atherogenic triad is: elevated triglycerides + low HDL + small dense LDL particles. The 'small dense LDL' component is invisible on a standard lipid panel, which reports only LDL-C (a calculated number, not a measurement of particle count). This is why ApoB—which directly measures the number of apolipoprotein B particles—has become essential. Understanding the triad and knowing how to identify and treat it is foundational to modern lipid management.

## Why The Atherogenic Triad Flies Under the Radar

**Standard lipid panels are incomplete.** They report LDL-C, HDL-C, triglycerides, and total cholesterol—but do not measure or report LDL particle size or density. A 'normal' LDL-C masks a dangerous particle burden.

**'Normal' LDL-C provides false reassurance.** Patients and providers alike assume LDL-C < 130 or < 100 mg/dL means low CV risk. But in the atherogenic triad, hundreds of small dense LDL particles may be circulating despite LDL-C in the 'normal' range.

**Insulin resistance is the root, but unmeasured.** Insulin resistance drives all three components of the triad: it raises triglycerides, lowers HDL, and shifts LDL toward small, dense particles. Yet insulin resistance (and the metabolic syndrome that often accompanies it) is rarely screened for in routine care.

**Recognition requires non-HDL-C and ApoB.** Identifying the triad requires looking beyond the standard lipid panel. Non-HDL-C = Total Cholesterol - HDL (easier) or ApoB (most specific) reveals the particle burden hiding behind normal LDL-C.

## What Changed in 2026

**Non-HDL-C and ApoB elevated to co-primary targets.** The 2024 PREVENT guidelines now explicitly recommend treatment to goal for both non-HDL-C and ApoB in high-risk patients. LDL-C alone is insufficient.

**Treatment goals formalized in metabolic dyslipidemia.** For patients with the atherogenic triad and metabolic syndrome, specific treatment targets are now defined: non-HDL-C goals are 20-30 mg/dL higher than LDL-C goals, and ApoB should be reduced proportionally.

**PREVENT integrates metabolic inputs.** Risk calculators and guideline recommendations now explicitly incorporate insulin resistance, visceral adiposity, and metabolic syndrome status as modifiers of CV risk.

## MyCardioAdvocate™ Checklist

Use this checklist to identify, assess, and treat the atherogenic triad:

### 1. Identify the triad pattern in your lipid results.

Look for: Triglycerides >150 mg/dL AND HDL <40 mg/dL (men) or <50 mg/dL (women). If both are present, the triad is active.

### 2. Screen for insulin resistance.

Ask your provider for fasting insulin and glucose, and calculate HOMA-IR. A HOMA-IR >2.5 indicates significant insulin resistance and confirms the metabolic basis of the triad.

### 3. Target non-HDL-C alongside LDL-C.

Non-HDL-C = Total cholesterol - HDL. This number reflects all atherogenic particles (VLDL, LDL, Lp(a)). If your triglycerides are elevated, non-HDL-C will often be higher than LDL-C. Ensure your provider is treating to non-HDL goal, not LDL alone.

### 4. Request ApoB testing as confirmatory.

ApoB directly measures the number of atherogenic particles. If your ApoB is elevated despite normal LDL-C, you have the triad. This justifies more intensive lipid therapy.

### 5. Address the root cause—metabolic syndrome.

Weight loss, increased physical activity, reduced refined carbohydrate intake, and treatment of diabetes/hypertension all improve insulin sensitivity and shift the lipid pattern. If medical therapy alone is insufficient, discuss GLP-1 RA or metabolic bariatric surgery.

> **Pro Tip:** If your triglycerides are high (>150) and HDL is low (<40), your LDL-C is almost certainly misleading. The standard LDL-C calculation (Friedewald formula) assumes LDL particles are large and buoyant. In the triad, they are small and dense, and the calculation underestimates true particle burden by 20-30%. Ask for ApoB. It will reveal the true particle count hiding behind 'normal' LDL-C.

## Key Takeaways

- The atherogenic triad (elevated TG + low HDL + small dense LDL) is common and dangerous, but invisible on standard lipid panels.
- 'Normal' LDL-C does not mean low CV risk if triglycerides are high and HDL is low.
- Insulin resistance is the root cause of the triad and drives accelerated atherosclerosis.
- Non-HDL-C and ApoB reveal the particle burden masked by LDL-C alone.
- Treatment should target the triad as a pattern, address insulin resistance, and aggressively lower ApoB.

## Next Steps & Related Content

- Check your most recent lipid panel for the triad: TG >150 and HDL <40?

- Ask your provider for non-HDL-C calculation and discuss whether it is at goal.
- Request ApoB testing to confirm particle burden.
- Discuss insulin resistance screening (HOMA-IR, fasting insulin).
- Work with your provider on metabolic improvements: weight loss, glucose control, physical activity, refined carbohydrate reduction.

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