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The Unmet Need in ASCVD: Why Targets Are Still Not Enough

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Dr. Michos:
Welcome to CE with GLC. I'm Dr. Erin Michos, a professor in cardiology at Johns Hopkins University, and today I'll discuss challenges in attaining LDL cholesterol goals in atherosclerotic cardiovascular disease despite statin use.

So lipid-lowering trials have pushed achievement of LDL cholesterol to lower and lower levels with clear evidence of continued benefit at very low achieved LDL cholesterol. LDL-C goals thus have become much more stringent as we learn more about the risk of hyperlipidemia, and these changes are reflected in the updated ACC/AHA 2026 dyslipidemia guidelines.

So goals are back. for very high-risk patients who've had a clinical event like a heart attack or a stroke, or those that have a severe burden of atherosclerosis, such as calcium scores above 300, the LDL goal is less than 55 mg/dL. For high-risk patients who have evidence of coronary disease on imaging or have type 2 diabetes and risk factors, the LDL goal is less than 70. And then for borderline to intermediate-risk patients, such as those that have an LDL greater than 160 or someone who has a high 10-year risk or a strong family history or a high lifetime risk, the LDL goal is less than a hundred.

Now, the guidelines also bring in ApoB goals. Keep in mind that there's one ApoB moiety per atherogenic lipoprotein particles, so that ApoB measurement is like a particle count. So if your LDL goal is less than 55, your ApoB goal is less than 55. If your LDL goal is less than 70, your ApoB goal is less than 70. And if your LDL goal is less than 100, your ApoB goal is less than 90.

Now, if you don't have ApoB available, you can use a non-HDL, total cholesterol minus HDL, and those goals are 30 mg/dL higher than your LDL goal because we know that LDL is calculated or estimated on a lipid panel and reflects the total concentration of cholesterol across all the LDL particles.

But for the same LDL concentration, say 130 mg/dL, you could have different risks depending on your particle count. Because if you have small, dense LDL, you're going to have more particles per a given LDL cholesterol concentration and a higher ApoB. And that's why the guidelines have brought back in ApoB targets, particularly for those who have ASCVD, metabolic syndrome, diabetes, high triglycerides, to help better guide, risk prediction. The 2026 dyslipidemia guideline also endorses at least one-time measurement of Lp(a) in a lifetime as a risk assessment, risk enhancer to help reclassify risk.

Now, unfortunately, despite the fact that we have LDL goals, guideline discordant care occurs frequently. There was a recent publication

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from the Family Heart Foundation analysis that looked at commercial, insured patients, so everybody had insurance. They looked at over 3 million individuals who had ASCVD and showed that despite being high-risk secondary prevention patients, only 41% achieved an LDL less than 70, and only 21%, less than, one and a quarter, achieved an LDL less than 55 mg/dL. If you're looking at persistence of therapy over a 2-year period, only 35% were on lipid-lowering therapy for at least 20 out of 24 months. Shockingly, 28% of individuals with ASCVD secondary prevention patients were on no lipid-lowering therapy, and only 31% were on high-intensity statins. If you looked at metrics of what we consider comprehensive LDL management, having an LDL less than 70, being on a high-intensity statin or being dispensed LDL-lowering therapy for at least 20 months, only 13% of ASCVD patients were getting comprehensive LDL care.

Another recent study looking at US healthcare systems, looking at EHR data, showed that fewer than 25% of US adults with ASCVD achieve LDL goals less than 55, mg/dL. in this analysis, again, only 38% of ASCVD patients were on high-intensity statins, and the use of combination therapy was really low. Only 4% of patients were on statin plus ezetimibe, and only 0.5%, so less than 1%, were on a statin plus a PCSK9 monoclonal antibody. But those patients that were on combination therapy were much more likely to achieve these risk-based targets.

So we know it's important to reach low LDL levels quickly, immediately after an acute event. The data strongly supports an early intensive combined lipid-lowering strategy over this delayed stepwise approach because when you do things stepwise, you lose this time, and it's important to get lower for longer as quickly as possible.

So we have a clinical paradox, and I like this quote from a, a colleague that atherosclerosis represents a clinical paradox. It's potentially the most preventable or treatable chronic disease, yet it remains the greatest cause of disability and death throughout the world. This does not have to be the case.

So in conclusion, if you're trying to aim for these lower LDL targets, like less than 55 mg/dL, the need for combination therapy is inevitable. We now have many tools for lipid-lowering, statins and non-statin therapies, and we know that non-statin therapies provide the same risk reduction per millimole per liter of LDL lowering as statins do. And non-statins are now also recommended by both the US and European guidelines. However, unfortunately, implementation in clinical practice is still far lagging behind guidelines, and there's an urgency to achieve LDL targets as quickly as possible, and evidence-based new approaches include earlier initiation of combination therapy. So in upcoming episodes, you're going to also hear about a new novel therapy for LDL lowering, a CETP inhibitor, obeticholic, which has been shown to have efficacy in reducing atherogenic lipoproteins as well as good safety, with exploratory analysis showing reductions in major cardiovascular events and nuanced diabetes while we await the definitive results from an ongoing cardiovascular outcome trial called PREVAIL. So thanks for joining me today. I hope this information is useful for you in your practice. Thank you.

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