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**ReachMD**
www.reachmd.com
info@reachmd.com
(866) 423-7849

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Grooving Through Lipid Management: A New Rhythm in ASCVD Risk Reduction

**Announcer:**
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**Dr. Gluckman:**
So just for the next 20 minutes, I'm going to bring Erin up. We're going to be doing five-minute, really high-packed, high-yield information about specific topics, and then we'll be breaking out into the case-based discussions.

**Dr. Michos:**
Great. So I'm going to set the stage with discussing the burden of ASCVD really quickly.

# Heart Disease Remains the Leading Cause of Death in the US

I'm sure this is very familiar to this audience that cardiovascular disease remains the leading cause of death in the United States. It exceeds all the rates from all cancers combined, as well as the next eight leading causes of death.

# Death Rates From CVD Are Rising

And you've probably all seen these statistics from the American Heart Association about cardiovascular mortality, that for a while, for over a decade, we were making progress in reducing cardiovascular mortality.

But since 2010, we've been on a really bad uptick that we're not only stagnated, but we're frankly back on a rise of cardiovascular mortality due to the burden and the epidemic of cardiometabolic diseases.

# Lipid Management: An Important Risk Factor for MI

Now we know from the INTERHEART study that nine modifiable factors make up 90% of the attributable risks for myocardial infarction.


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So this is good news for being a preventive cardiologist, that most of cardiovascular disease is preventable.

Among these nine modifiable risk factors, lipids, highlighted there in red, account for about half, or about 50%, of the attributable risk from MI. And this is why lipid management is a central tenet for ASCVD prevention.

## LDL-C Is a Causal Agent in ASCVD Pathogenesis

And we have overwhelming evidence from observational data, genetic data, and randomized clinical trial data, that LDL cholesterol and ApoB are causally related to atherosclerotic cardiovascular disease pathogenesis. And again, that's why LDL is our primary target. And we've been moving in our guidelines to lower and lower LDL goals, as you'll hear about.

## Even Modestly Elevated LDL-C Is Associated With Increased Risk When Lifelong

So, not only does it matter about how high cholesterol is, but it's also the duration of the exposure. To be thinking of cholesterol years like we think about pack years of smoking. So, in this example, the dotted line represents a theoretical threshold of cholesterol years where you would have the clinical onset of ASCVD. Someone said this is about 5,000 mg/dL per year.

Individuals with severe primary hypercholesterolemia, like FH, shown in the red, left untreated, they will cross over that threshold of clinical ASCVD early in life. In contrast, those shown in the green bar, who have lifelong low LDL from favorable genetics, healthy lifestyle, or perhaps earlier initiation of LDL-lowering therapy, they may never cross that threshold of having a clinical ASCVD event.

But I think we need to pay more attention to those in the yellow and the orange with mild or moderately elevated cholesterol that often get ignored. But they too, with enough years of exposure, will also have earlier onset ASCVD compared to their counterparts with very low LDL.

## Atherogenic LDL-C: Lower for Longer Is Better

We see that there's a proportional reduction in coronary risk related to the degree of LDL lowering. So, as the figure moves to the right with greater LDL lowering, we see greater reduction in coronary risk. But duration of treatment matters. For example, if you look at the dotted line around 1 mmol/L LDL-lowering or 39 mg/dL, for that same degree of LDL-lowering, those in the red that represent genetic studies that have had that degree of lowering from birth, they have a 50% reduction in coronary disease. Observational data that's 10-12 years, is shown in the blue line. Randomized clinical trials in the black line, there's still a proportional reduction, but 1 mmol/L lower LDL confers about a 22% reduction. And those patients who have averaged only five years at that lower threshold.

## Duration of Lowered LDL-C Is Also Important

And this slide shows the same thing. If you look at, this is from statin data, but showing for the same degree of lowering, for every 38 mg/dL lower LDL, the more years that people have been exposed to lower levels, they have greater reduction in events. So this is why we need to overcome inertia and get to these lower targets, being lower for longer.

## One Half of Patients with ASCVD Are Not Receiving Statins


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But we're doing very terribly in clinical practice currently. This is data from commercial medical claims data. Everybody in this registry has access to insurance and so access to medicines.

These are ASCVD patients, secondary prevention patients, the highest-risk patients, and yet nearly half were on no statin at all. And less than a quarter were on high-intensity statins. And we see gaps such as women and those with cerebrovascular disease or peripheral arterial disease being less likely to be treated compared to those with coronary disease.

## GOULD Registry: Nonstatin Therapy Is Underused in Patients With ASCVD

And we see there's just a lot of clinical inertia. This is the GOULD registry, US data, patients with ASCVD. So, we're talking about secondary prevention patients.

But two-thirds remained above an LDL above 70, and now we're moving to lower targets, less than 55. But two-thirds above LDL above 70, and if you look at non-statin therapy like ezetimibe, PCSK9 inhibitors, less than 10%. So, underutilization of non-statin therapy.

## Consequences of Nonadherence to Statin Therapy

And there's a lot of consequences to non-adherence or underutilization of statin therapy. The figure on the left is a secondary prevention population. The figure on the right is primary prevention.

But they show that if you have poor adherence or discontinuation, you have greater risk of having a major adverse cardiovascular event.

## The Clinical Paradox . . . and a Call to Action

So, I'll end with this sort of call to action 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, and this does not have to be the case.

## The Burden of ASCVD: Conclusions

So, as I turn it over to the other speakers, just remember that for LDL, lower for longer is better. That non-statin therapies, which you'll hear about, provide the same risk reduction per degree of LDL-lowering. But we have still a major problem with implementation and adherence.

Thank you, and I'll turn it back to Dr. Gluckman.

# Crooning About Current Therapies and Individualizing ASCVD Care

**Dr. Gluckman:**
Erin, that was absolutely fantastic. So we're going to pivot through here, and I'm going to start with a poll.


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**Poll 3**

And this is actually going to get to set the stage for some other discussion this evening. But please take out your tablets or have them available. I am familiar with the latest clinical data on novel Lp(a)-targeting therapies and decisions about lipid therapy intensification in patients with elevated Lp(a).

A. I strongly disagree;
B. I disagree;
C. Neither agree nor disagree;
D. Agree;
E. Strongly agree.

Please key in your answer.

Great. So I'm going to move through my slides and actually not talk about lipoprotein(a). We're going to bring that up in a moment here.

## LDL-C Treatment Targets Over Time

But I just want to sort of highlight the evolution of where we are today. This is a really busy slide, but just pictorially displays from 1988 all the way through what we had up until and through 2022, not inclusive of our document that came out just about two weeks ago, our dyslipidemia guidelines. The movement in what we consider the target or goal LDL cholesterol for those that are at increased cardiovascular risk.

And you can see the evolution, left to right. We're moving to lower and lower targets or goals for this patient population.

## Blood Cholesterol Guideline Recommendations

So, historically we've talked about four patient groups with regard to targeting LDL cholesterol-lowering. We've expanded that in the 2026 guideline. People are familiar with clinical ASCVD, those that have severe hypercholesterolemia with LDLs greater or equal to 190. Those with type 2 or type 1 diabetes.

Lastly, primary prevention. And then a few sort of subgroups, if you want to think of it, those with subclinical atherosclerosis, which are a unique call-out, but a lot of attention is given to those individuals in the new guidelines. And then a focus on hypertriglyceridemia with the new guidelines, now more broadly covering dyslipidemia, not just cholesterol.

## It Starts with Lifestyle Modifications

It starts with lifestyle. So I'd be remiss, we'd all be remiss if we didn't talk about the various ways in which we can help to lower LDL cholesterol. This, you might say, Ty, it's really hard to get patients to do one of these, let alone seven of these things.

But nonetheless, there are meaningful improvements in LDL cholesterol that can be achieved through lifestyle changes. And that's highlighted by the 30-50% reduction in the right lower corner.

## Many LDL-C–Lowering Tools at Your Disposal

So, there are also a number of therapeutic targets or pharmacologic therapies that are available to lower LDL cholesterol.

Erin did a phenomenal job of highlighting the benefits conveyed, associated with this. But these include both oral and non-oral


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therapies, statins and non-statin therapies. We could be spending an hour and a half just talking about this slide alone, but just to highlight that we have a pretty full toolbox of therapies available to us today. They're just underutilized.

## Refining CV Risk in Those with Clinical ASCVD

In terms of understanding how to inform your decision-making, we're increasingly trying to move towards shared decision-making and personalized approaches. Just to say on this slide, and this was brought up previously in previous guidance, but that individuals, even with atherosclerotic cardiovascular disease, can be further divided into those that are very high risk, that have two or more of the conditions, in orange, or one condition, in orange, and two or more, in blue.

But those individuals, in an effort to try and match the intensity of LDL cholesterol-lowering to the baseline risk of the individual, even those with an atherosclerotic cardiovascular disease, greater refinement is needed in better matching two targets like an LDL less than 55.

## Important Additional CV Risk Factors in Primary Prevention

It'd be important to realize that in primary prevention, those with diabetes on the left and those that are on the right that represent those with primary prevention that don't have diabetes, there are a number of factors that should inform your risk calculus in this patient population, whereby the presence of one or more of these should guide you to decision-making about being more aggressive in LDL cholesterol-lowering for your patient population.

I'll just point out on the right-hand side in the most recent guideline, this is by no means plain favorites, but inflammatory diseases like rheumatoid arthritis, lupus, psoriasis, or reproductive risk factors often get undertaken into consideration. They're inadequately assessed that it importantly inform the risk of the patient and the intensity with which we need to more aggressively lower their LDL cholesterol.

## Using Coronary Calcium to Refine CV Risk Assessment

Coronary calcium gets a lot of attention in the new guideline for cardiovascular risk assessment. This is just a pictorial display of the visual appearance of increased coronary calcium. Higher your coronary calcium, higher your atherosclerotic plaque burden, and higher your cardiovascular risk, arguing why in the new guidelines we need to be more aggressive in LDL cholesterol-lowering.

## Goals for ASCVD Risk Reduction Based on Patient Population

And if you're a one-pager and you just want one page to take away to paste on the wall, put on your desk, this is sort of a synthesis of what the guidelines have put together in terms of treatment targets or goals for this patient population in terms of LDL, non-HDL. What's not shown on this are the ApoB goals that are included in the guideline as well across these different strata of populations overall.

## Putting It All Together

So putting it all together, this is a very busy slide, but for those of you who like flow diagrams, just understanding the cardiovascular risk of the population that you're talking about, it starts with lifestyle, it starts with statin therapy, and ideally on maximally tolerated statin therapy.


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Really important four to 12 weeks later to reassess their lipids. Any change in therapy, initiation of therapy, reassessing their treatment response. And those that persist with LDL cholesterol goals, LDL cholesterol levels above goal, intensifying their treatment regimen with non-statin therapy.

And again, don't have the time to go through each one of these branching points in detail. Just to highlight, we need to be doing much better than the slides that Erin highlighted in intensifying both our statin and non-statin regimen.

# Residual Risk Riffs: Monitoring Lipids and Lp(a)

So with that, we're going to pivot over to lipoprotein(a). I'm going to invite Pam up to now present that information.

**Dr. Taub:**
So, Ty gave a really incredible overview of LDL-lowering and our guidelines, how they've evolved. I'm going to talk about a relatively newer biomarker, and that's lipoprotein(a). But we'll start with a polling question.

**Poll 4**
The question is, I am familiar with current guidelines on lipoprotein(a) testing and how to incorporate test results into cardiovascular risk assessment and management.

A. Strongly agree;
B. Disagree;
C. Neither agree nor disagree;
D. Agree; or
E. Strongly agree.

## IMPROVE-IT: Aggressive LDL-C Lowering Does Not Eliminate ASCVD Risk

We know LDL-lowering is important, but we've seen in studies such as IMPROVE-IT that even when we get our LDL to very low levels, in IMPROVE-IT, it was in the 50s, and yet we still had a very high rate of cardiovascular events. So even with an LDL in the 50s with a combination of simvastatin and ezetimibe, there was still about a 33% event rate. And this is because there's other factors that need to be addressed, including lipoprotein(a), triglycerides, and cardiometabolic risk factors such as insulin resistance and hyperglycemia.

## Lipoprotein(a)

So, what is lipoprotein(a)? The way I like to describe it is it's the evil, diabolical cousin of LDL. So it's an LDL-like particle, but it has additional moieties that make it even more powerful in terms of being more atherogenic and more thrombotic.

It's actually very common. One in five people have an elevated lipoprotein(a). So if you have a busy clinical practice, you're going to see multiple people in a day that have elevated lipoprotein(a). And it's the strongest inherited risk factor for both coronary artery disease and aortic stenosis. It does occur more commonly in South Asians and blacks.

## Journal of Lipid Research (March 2016)

And the two main aspects of lipoprotein(a) that you need to be familiar with is it's a very atherogenic molecule. So, it increases the incidence or plaque. It also is associated with faster progression of aortic stenosis.


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## 2026 Blood Cholesterol Guidelines: Lp(a) Is an ASCVD Risk Enhancer

And in the new guidelines, it's been very clearly delineated as an ASCVD risk enhancer.

And the numbers you need to remember are greater than 125 nmol/L or greater than 50 mg/dL in terms of increased risk.

## 2024 NLA Focused Update on Lp(a)

In the 2024 NLA guidelines that Erin and I were part of, we were the first guidelines in the U.S. to state that lipoprotein(a) should be checked at least once in the lifetime of every individual. And this was also in line with what the European guidelines were saying, at least once.

## 2026 ACC/AHA Dyslipidemia Guideline on Lp(a)

Now we have, in the 2026 guidelines, lipoprotein(a) should be checked at least once. So all of your patients should have a lipoprotein(a) measured at least once. What I also love about the new guidelines is they really quantify risk based on level.

So, for instance, if you have a lipoprotein(a) that's 350 nmol, well, your risk of developing ASCVD is threefold higher. And so that's a really nice diagram to keep in mind, because not all lipoprotein(a) of 50 is not the same as a lipoprotein(a) of 350. That person with 350 has higher risk.

What I also like in the guidelines is they give us some actionable items. So when someone has an elevated lipoprotein(a), right now in 2026, we want to be very aggressive with their LDL. It's the concept of two to tango.

When you have both high Lp(a) and LDL, that's when the risk is highest. So we want to reduce the LDL to very low levels in these patients, and this is where the guidelines give us more options. We can now add a PCSK9 inhibitor if we can't achieve lower LDL goals with statin, ezetimibe, bempedoic acid.

## Nurses Health Study: Interaction of Lp(a) and LDL-C

Here is the Nurses Health study, which illustrates the concept I was talking about, the two to tango, which is risk factors don't operate in isolation. They really synergize with other factors. So when you have lipoprotein(a) elevated, LDL elevated, you have a very high risk of future events. And then if you add other risk factors, such as diabetes, that also enhances risk.

## Lp(a) Impact on Plaque Progression

One of the things that we're starting to learn a little bit more about is how does lipoprotein(a) impact plaque. And what we're seeing in some studies is that when you have an elevated lipoprotein(a), there's actually accelerated plaque progression.

We're also learning that many people with lipoprotein(a), even if their calcium score is zero, we should have strong clinical suspicion that they have soft plaque. And think of further imaging in these patients with elevated lipoprotein(a).


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## Must Incorporate Lp(a) Into Clinical Risk Assessments

This is a handout that I give patients in clinic to really talk to them about all of their risk factors. And many of the risk factors are modifiable, as Erin so well described in her talk, especially LDL. But there's factors that right now are out of our control. And right now, lipoprotein(a) is one that we cannot dramatically lower with therapy.

PCSK9 inhibitors do lower lipoprotein(a) about 25-30%. But we have better drugs that are in clinical trials that will get lipoprotein(a) levels to almost zero. But what you can do is really get good control of all of the other factors.

And what I tell patients is if you perfect all of the modifiable risk factors, then the lipoprotein(a) becomes neutralized. So that means getting the LDL as low as you can, controlling blood pressure and other lifestyle modification that we can do to decrease cardiac risk.

## Lp(a)-Lowering Therapies

So, the future is very exciting for lipoprotein(a) lowering.

We have multiple therapies in very late-stage clinical trials. We should have the results of the pelacarsen study or the HORIZON study by the end of the year. And so, hopefully, we will have a therapy on the market within the next couple of years.

And the first trial that's going to read out is the pelacarsen study, which is an antisense oligonucleotide. We also have some small interfering RNAs, which are dosed very infrequently every six months, that are in trials as well. But right now, we do have the PCSK9 inhibitors that we can use. And the guidelines give us a lot of leeway in utilizing PCSK9 inhibitors.

## ASPREE: Primary Prevention with Aspirin and Lp(a) Genotypes

There's also some emerging data that aspirin might be beneficial in patients with elevated lipoprotein(a). Typically for primary prevention, we don't do aspirin.

But in individuals with elevated lipoprotein(a), there may be some benefit. And so, you want to have a risk-benefit discussion with patients about starting aspirin. Again, we don't have the randomized controlled trial data. But remember, it is a very thrombotic molecule. And so this is worth discussing with patients.

## How Does Lp(a) Change Lipid Management?

And so, the way that lipoprotein(a) changes management is we do maximize statin therapy. We consider aspirin. And in those that are eligible, we consider PCSK9 inhibitors. And don't forget, it is inherited. So, we want to test family members. There's been many patients who've had their children tested, and you pick up an elevated lipoprotein(a) at a young age, and we start aggressive lifestyle modification very early and also enroll patients in clinical trials.

Thank you.

# *Bass*line of Care: Challenges, Future Directions, and Holistic Care

**Dr. Le:**


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Okay, it's always hard to follow such great talks. But my job here is to look at the challenges, future directions in holistic care. And so, as you've seen – oh, let me start with poll 5.

**Poll 5**
I'm comfortable using shared decision-making to guide ASCVD therapy intensification when patients are clinically stable with no recent cardiovascular events.

A. Strongly disagree;
B. Disagree;
C. Neither agree nor disagree;
D. Agree; and then
E. Strongly agree.

## Mind the Gap in Therapy Implementation

All right, excellent. Okay. All right, and so we've already seen this.

As the GOULD registry, we need to be doing better. And Intermountain Health is no better, no worse. 50% of patients with ASCVD are not on statins.

Let's just start there. Let's start with the statins, and then we can move to non-statins. But only 23% were on high-intensity statins.

So here's the GOULD registry. 66% remained with an LDL cholesterol greater than 70 mg/dL. These were individuals who were enrolled in the trial to – because they were interested in improving statin therapy.

So we didn't really move the chains much in this group, regardless of knowing what the guidelines were.

**Poll 6**
Poll 6. In your practice, what is the number one reason patients do not reach LDL cholesterol goals?

A. Clinical inertia, unclear next step;
B. Cost, prior authorization and access;
C. Follow-up gaps, lab visits do not happen; or
D. Patient hesitancy, I feel fine.

## Barriers at 3 Levels

So there are barriers at three different levels. The first is at the patient, as you just saw. Your sense is that there's some patient hesitancy. So maybe there's barriers to treatment adherence, safety concerns, maybe a lack of understanding about their cardiovascular risk. Like, why are you giving me this therapy when I don't know? Am I at risk? And so, some of those solutions is a shared decision-making process.

Imaging, maybe, to visualize their disease, rather than just guessing at a percent, addressing those social determinants of health. Then, from the healthcare provider or clinician standpoint, maybe there's some clinical inertia. Perhaps even in those who are enrolled in a trial to improve statin therapy, there's some unfamiliarity with the guidelines, confusion about treatment goals. And so, decision support tools may be helpful. Maybe, you know, we're not very happy about BPAs, but maybe that will help.

Performance audit, like, how am I doing? I think I'm doing great, but perhaps I'm not prescribing as well. Ongoing education.

And then, from a system standpoint, maybe that's where we can support both patients and clinicians by helping with that prior authorization access, removing that fragmented care, and then improving team-based care with multidisciplinary care. Pharmacy integration, etc., with alerts.


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## Interprofessional Care: Turning Intentions into Outcomes

So, interprofessional care. Team-based care is a class one. It is class one, both in the 2019 primary prevention guidelines as well as 2023 chronic coronary disease guidelines. And, of course, it manifests again in 2026.

So really, we need to surround the patient, both from health system and clinician around the patient.

## Practical Implementation Pearls: Turning the Guidelines into Real-world Care

So, what are the practical implementation pearls? Turning those guidelines into real-world care, not repeating the mistakes of, you know, the Gould Registry here.

Well, maybe we motivate with imaging. I think seeing is truly believing. When patients see that coronary calcium and you can put together the risk and the event rates, that helps.

Initiating combination therapy early. Rather than waiting 4, 6, 9 months. By the way, it changes as quickly as 2 weeks with pharmacokinetics and dynamics, but we say 4-12 weeks in terms of labs. So, don't wait to start combo therapy. Follow up systematically, 4-12 weeks. Get those labs. Gamify this. Let patients see the change in labs or not change in labs, the goals, so that they know what to do next.

Address cost proactively. We have co-pay cards. We have ways to help with prior authorization. Those are the things that you should be thinking together as a clinic beforehand, not wait until it surprises the patient and you of cost.

And then leverage technology. Let those systems do the work. We have EMR, EHR. We should be able to leverage those systems to help us.

## Resources for Shared Decision-making

I would be remiss as the editor of CardioSmart to say that we don't have these shared decision-making. We do. We have so much education available for patients, and when you have these already hanging up in the walls of your office, it allows patients, that hesitancy to be removed up front. They're able to see what cholesterol does, how it promotes atherosclerosis, and what coronary calcium is, how statins work to lower that risk.

## Key Takeaways

And so, here are the key takeaways.

Implementation of guideline-directed therapy way lags behind current evidence. This is our biggest challenge. Barriers exist at all three levels.

Multidisciplinary team-based care, no one can do this alone. It is just too complicated. And imaging, use it. It figures highly in the current guidelines. Please, seeing is believing. And then systemic and systematic follow-up and early implementation of combination therapy is critical.

# Masterclass Cases


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**Dr. Gluckman:**
Erin, Pam, Viet, that was fantastic. We're now going to have our excellent faculty move down into the audience. We're going to present some cases. This is meant to be interactive. This is meant to be fun. And so we're going to be presenting some cases.

We need to pick up a little bit of time, and we're going to move maybe through the cases a little bit faster. But we want you to work at your tables to be able to think about this. For those that are online, I'm going to be talking to you as we move through this.

But I'm going to start with three cases.

# Patient Case 1: A.G., 54-Yr-Old Woman

We're going to start with case one. This is A.G., who presents to your primary office for routine examination. No complaints, just trying to be more active. She has hypertension and hypothyroidism. She has a family history notable for a father with a myocardial infarction at 42.

She's on lisinopril at 10 mg a day, levothyroxine, as you can see the dose listed there. Normal intensive, normal heart rate, BMI is 30. Physical exam is notable for a 2 out of 6 systolic injection murmur at the right upper sternal border.

You can read through the lipids there that are listed up there for you. I'll call out her triglycerides and HDL cholesterol in particular. You can read through the hemoglobin A1c, fasting glucose, and the results from her comprehensive metabolic panel.

# Patient Case 1: ASCVD Risk Score Is Calculated

So you, in reading the most recent 2026 dyslipidemia guidelines, decide to implement the PREVENT risk score. You estimate her 10-year ASCVD risk using the PREVENT risk score, and as is shown here, she's got a 2.7% 10-year ASCVD risk. Her 30-year risk is 14%.

I'll just call out very quickly, her current USPSTF recommendations would not likely benefit from aspirin therapy. She has no indication to currently be on statin therapy, as her PREVENT risk score is less than 3%. This puts her in a low-risk category, and her blood pressure is well-controlled.

You can see some of the characteristics of her down at the bottom, which is attempting to summarize her aggregate of risk overall.

# Patient Case 1: ASCVD Risk Score Discussion and Next Steps

And so, primary care, or you all in your clinics, reassure her that she's at low risk because her ASCVD risk score is low, and her LDL is in an excellent range at 44. You encourage her to lose weight, improve her diet, increase physical activity, recommend over-the-counter fish oil.

I'd welcome you now at your tables talking about whether this management is something that you would also share and recommend, and then would the management change based on her lipoprotein(a) level.

We're going to revisit this case in just a moment, and then we're going to bring the panel up to sort of talk about questions that may come up. But we'll pause here and allow you at your table to, in particular, ask the question: would you change anything in terms of her approach, and would the measurement of Lp(a) be of have help to you?

Quiet, but we'll do that. And I'm going to be talking to those of you that are online.

So, in this patient that I'm seeing right now, this is a patient that, while her LDL cholesterol level is in the quote-unquote "desired range," the LDL cholesterol of 44 may be a marked underestimation of her true atherogenic risk overall.

She's got higher triglycerides and a diminished HDL cholesterol. I know, I'm talking to those online. So, if you could just talk at your table, it would be great.


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And so, in my estimation, the LDL cholesterol that's being measured may be, again, an underestimate of their true atherogenic risk. In addition to this patient having multiple cardiometabolic abnormalities, it's really important in this patient to try and focus in on intensifying their comprehensive lifestyle focus. And measurement of lipoprotein(a), like a review of other risk enhancers, is going to be pivotal, even in someone with an LDL that measures at 44, this is somebody who may benefit from pharmacotherapy to even further address LDL cholesterol. And the estimated 10-year risk may be an underestimation of this patient's true risk overall.

So, for those of you that are online and those of you at the tables, please, we're going to give it about another 60 seconds of thought locally. For, again, those of you online and in the room, 30 to 60 seconds, and then we'll bring our panel back up to sort of talk through what they're thinking about overall. I know this may be new for you, but as much as you can talk at your tables, we'll be revisiting this in a moment.

And even though it's quiet, for those of you that are online, give us another 20 seconds, and we'll be bringing people back on, and we'll further the discussion.

So, I'm going to ask our panel to come back up, and while we're doing this, as people are walking back up, I think probably most of you have gotten it. We're going to make sure that the mic is turned on for all of our panelists.

# Patient Case 1: Panel Discussion

Pam, I'm going to start with you while you're walking back up and at least thinking about it, or you can stay down, either way, it's whichever's easiest for you. Actually, we'll have you guys stay out there.

You know, Pam, this is a patient who's got a, quote-unquote, normal or low LDL cholesterol. How do you begin to think about this patient in your practice, and what do you do? Is this a true reflection of their atherogenic risk based on their LDL?

**Dr. Taub:**
Yeah, so at my table, we had a great discussion, and one of the attendees keenly pointed out the first thing that struck him was her triglycerides are elevated. They're in the 380s. And so this is when all your red flags need to go up because when the triglycerides are that high, the LDL, by the Friedewald equation, is falsely low. It fails you. So, whenever you see elevated triglycerides, you want to be cautious about interpretation of that LDL. And so, if you were just glancing at the LDL, you would say, "Oh, the LDL is 44, it's great," but it's not really truly her LDL.

So this is where you want to calculate a non-HDL, which is total cholesterol minus HDL, and her non-HDL is very high. This is also where, per the new guidelines, you might want to think about ApoB in assessing risk. And so this is a great case where if you just looked at the LDL without looking at the entire panel, and also her entire profile, she has an elevated BMI, and she has elevated fasting glucose. So, she has metabolic syndrome. So, she has metabolic syndrome, and she's a very high-risk patient. And this is where, also, you have to be cautious with the risk score because when you put in some of the parameters in the risk score, it gives you a low risk, but when you look at this patient in front of you with a BMI of 30 and elevated triglycerides, that is not low-risk. And this is where you have to look at the risk enhancers that are really well-delineated in our guidelines.

**Dr. Gluckman:**
I love it. Erin, anything else briefly to add?

**Dr. Michos:**
Yeah, so we also agree that the LDL likely is underestimated by Friedewald, so I definitely would measure an ApoB in this patient because I think there's been discordance. Definitely, would measure an Lp(a), but even if the Lp(a) was normal, I think because of the family history and her triglycerides, and I bet anything the ApoB's going to be high, she would have an indication for statin therapy because of the elevated triglycerides. Statin's her first line of therapy.

We also discussed imaging, coronary calcium. This is a 54-year-old woman. It would be less helpful to get a calcium score if she was 34, but in a 54-year-old woman, I would still treat her with a statin, but the calcium score might help guide the intensity of treatment, which is also adopted in the guidelines, that you can use the coronary calcium not only to initiate, but intensify.

So if she had a very high calcium score, certainly above the 75$^{th}$ percentile, you would target an LDL less than 70 compared to less than 100, and then if they had severely elevated score, you could even consider these optional LDL-lowering less than 55.

**Dr. Gluckman:**


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Perfect. Viet, I'm going to come back to you for the next part of this. So, you know, we'll make sure that the volume is turned up so that it makes sure everybody can hear it, so I apologize for that.

Understood. We'll do our best to try and work through it so that the audio is turned up for you in that regard.

# Patient Case 1: Follow-up With A.G.

I do want to continue with this first case, A.G., this is a follow-up to the same patient. She experiences abdominal discomfort and nausea three months later. She's seen the emergency department; diagnosed with a non-ST segment elevation myocardial infarction. Taken to the cath lab, a stent is placed in the proximal LAD. A.G. also has 40% stenosis of the proximal circumflex.

Reviewing the lipid panel now, three months from the prior three months, you can read there, total cholesterol 160, triglycerides 382, HDL 40, LDL is 44. I think we've talked about a lot of this, but her non-HDL is 120. Because of the patient's triglycerides, the cardiologist feels that her LDL is falsely low. This was hammered home by both Pam and Erin in this regard, and therefore her ASCVD risk is underestimated.

At that time, you check an Lp(a). It's elevated at 190. We talked about optimal levels, and to the point that both Erin and Pam brought up, an ApoB is elevated at 105.

So, how does your management strategy change based on this new information?

And Viet, I'm just going to turn to you briefly, just to provide perspective, because I think Pam and Erin teed it up nicely. Any additional thoughts about how this now, she's now presenting, she's a secondary prevention patient, unfortunately. How are you thinking about this patient?

**Dr. Le:**
Yeah, I mean, I think already, within HDS guidelines, we would look at her as an LDL cholesterol less than 55, and it's falsely low because of this lipidemia, the triglycerides. So you have to target the ApoB. In this case, with the discordance, I would shoot for an ApoB that's less than 65 in her.

**Dr. Gluckman:**
Great. So I'm going to actually pivot now to a second patient case, and we're going to go through a similar theme here, and I just want to run through this. And you're going to see, here are some different themes that we talk about, but I think the biggest piece about this is the integration of a lot of different disparate information that helps you understand the risk profile of the individual, where both LDL-centric therapies are initiated, and how we think about Lp(a) to inform risk.

# Patient Case 2: R. S., 56-Yr-Old Man

So this is R.S. He's now a 56-year-old man, South Asian man, non-smoker, sedentary, has a history of hypertension. You can see his systolic blood pressure listed there, metabolic syndrome, and a BMI of 31.

Family history is of a father with an MI at age 52, he's on lisinopril 20 mg a day. You can see the other laboratory data that's listed there, but a fasting glucose of 108 that I'll call out. His lipid panel demonstrates a total cholesterol of 232, triglycerides of 210, HDL of 38, and an LDL of 152.

His Lp(a) is 185. So, you're proactive. You have very much embraced what Pam shared earlier, everybody in their lifetime needs an Lp(a) measured, and his 10-year ASCVD PREVENT risk is 6.7%. This puts him in a category of being intermediate-risk based upon the PREVENT risk score. Would you start lipid-lowering therapy for this patient? If so, how aggressive would you be, and why? Does he have, or what are, risk enhancers that he may have, and is there any additional testing that is warranted based on this?

So, I'm going to ask each of you your respective tables. My mouth will be moving, but you won't hear me because I'm talking to folks online, but we're going to ask you to talk at your table about what would you do.

We're going to have our experts circulate and help you make informed decisions, and we'll come back and revisit this in a moment.


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Thanks so much.

So again, for those of you that are online, appreciate you bearing with us as we're talking in the room as well, but it's notable for this individual, A, this is a person who has intermediate-risk based upon their 10-year PREVENT risk score, but on top of it, there are a number of uncontrolled or prevalent risk factors or risk enhancers for this individual.

They're of South Asian ancestry. They have hypertension and many features consistent with metabolic syndrome. They have a family history of premature atherosclerotic cardiovascular disease, again, reflective of their metabolic syndrome. They have an elevated fasting glucose. Their lipids are abnormal in that they have a low HDL cholesterol and high triglycerides. Their LDL is elevated. Their lipoprotein(a) is elevated.

So, even though they have a 10-year ASCVD risk that puts them in an intermediate-risk category for which there is support for initiation of statin therapy, this individual has multiple other risk enhancers that are putting this patient at increased risk overall. And so, I think collectively in this regard, this is somebody who we want to be aggressive with in terms of LDL cholesterol-lowering therapy.

We're going to talk and revisit in a moment what we would be doing for this patient overall based upon their risk. We're going to come back to this as a larger group in just a moment, so I appreciate you bearing with me. But we're going to continue to revisit this, and we'll be answering some of the questions that you pose online towards the end as time permits.

So we're going to give you about 30-60 seconds more. I hear a lot of discussion at your tables, and we'll come back with our experts to sort of think through what are the things that you're thinking about overall. And for those that are online, we'll be coming back here with our experts in just about 20-30 seconds.

And certainly for the audience, as questions come up, please feel free to type them in. We're going to have a Q&A at the end. So, if we're not able to address questions as part of the discussion, we want to make sure that we have the ability to address them on the back end.

# Patient Case 2: Panel Discussion

So, Erin, I'm going to come to you here. I know you were talking at the table, and I apologize for that, but for the sake of time, Erin, not infrequently you'll see patients like this in your clinic. What stands out for you about this patient?

How do you begin to approach thinking about your therapeutic approach? What are your targets? What therapies do you begin with?

**Dr. Michos:**
They only mention it for extreme triglycerides, but not for cases like this. A class one indication for a statin for intermediate-risk. If the patient was borderline risk, which is 3% to 5% 10-year risk, with risk enhancers, that would be a two-way indication to start a statin.

Clearly, this patient has tons of risk enhancers, family history, the elevated triglycerides, and the high Lp(a). So, clearly, has a strong class one indication for a statin. The LDL is 152. We can get an ApoB. It's going to be high. The LDL is already high.

So, then the question is what the patient's target would be. And so, I would get more imaging in this patient, like a coronary calcium score, not to make the decision about statin, but if he had a lot of plaque, then we're thinking about something like a VESALIUS type of patient who may benefit, and additionally with evolocumab on top of a statin, that has some PCSK9 inhibitors, have some lowering of Lp(a), which is quite high in this person, because he's starting at LDL 152. So, if I start a high-intensity statin, at best I can get a 50% reduction in LDL, and then you're getting an LDL around 80.

But this is a patient who may, with a lot of plaque and that Lp(a), I'd actually want the LDL under 70. So likely a maximal statin is still not going to get me where we want. And so we're thinking, I'm thinking this patient's going to need combination therapy, and I may get additional imaging to determine my target, and the non-statin add-ons, like a PCSK9 inhibitor.

**Dr. Gluckman:**
Those are some great takeaways. Viet, thoughts, additional thoughts?

**Dr. Le:**
Yeah, I mean, that's exactly where I'd be going, is additional cardiac imaging. But, you know, at the same time, we would be left with triglycerides and HDL in a metabolic syndrome patient with a Class I obesity. So I think they meet indications for GLP-1 as well, which, as you treat the weight and the metabolic syndrome by A1c, you'll see a drop in that triglyceride and an increase in HDL.


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Here's a question.

**Dr. Gluckman:**
So, let me restate it for those that are on the audience. I think you were asking the question about risk enhancers can help to inform the risk profile and maybe project someone at being a higher risk than a 10-year risk would predict.

Pam, maybe you can speak to, we have a number of risk enhancers that are listed up there. I shared some of that information. Are these additive, or does it give you a gestalt feeling that the person who has three, four, five risk enhancers as opposed to one is thought of differently overall?

**Dr. Taub:**
Clinically, the more risk enhancers you have, the higher risk you're at. So, one risk enhancer that we really need to focus on is the South Asian ancestry. The guidelines call out both South Asian and Filipinos as having very high risk.

So, this patient has multiple risk enhancers, and the prevent risk score is just a ballpark, and if you put in all of those risk enhancers and incorporate that into your decision-making, the risk is actually much, much higher.

**Dr. Gluckman:**
And I'm going to just highlight and build off of what Erin had shared here, because this is one of those areas where imaging can be particularly helpful, I think the dyslipidemia guideline that came out two weeks ago probably positions the presence of subclinical atherosclerosis as being a key driver of intensifying your therapy and helping to define LDL cholesterol goals more than any other document that we've had. This is this patient, and it's building off of things that Erin shared.

# Patient Case 2: Imaging Risk Stratifies and Drives Action

So, this patient has evaluation. Now the picture that you're going to see in the middle portion on the rightward side represents a coronary calcium scan. We can talk about, do we extend this to CT, coronary angiography?

Needless to say, this patient has evidence of calcified plaque. There's also non-calcified plaque, or so-called mixed plaque, overall. A high coronary calcium score of 287, putting them above the 75$^{th}$ percentile for age, sex, and ethnicity.

I'm going to come back to our panel in a second, but maybe you can just speak to, and Erin, I'm going to come back to you. You've done a lot of work in this regard, but does this reinforce your point about how imaging can be a helpful adjunct? And maybe, just very briefly, without opening up a can of worms, which we can open up if we want to in the Q&A at the end, how do you think about straight up just calcium scoring, and is there any role for a CT coronary angiogram?

**Dr. Michos:**
I mean, I think in this patient we already have enough information that we want to treat very aggressively. We know that if we can get the LDL way down, that not only do we prevent progression, but we can actually confer some regression of the modifiable plaque. In an asymptomatic person, I think I have enough information in this high-risk patient to just treat them very, very aggressively.

When the calcium score is above 300, and we're getting really close here, they have the same event rate as someone with a prior MI. So it's almost the secondary prevention risk equivalent. So, I may be treating this patient like a secondary prevention patient.

So in this patient, I don't think that you would need additional information with the CTA because you have so much disease with the calcium score. But I think the CTA would be helpful in a younger person who's less likely to have calcified plaque, who has other risk factors like high Lp(a) or HIV disease or autoimmune disease, where the calcium score may underestimate the risk or give false reassurance because they don't have calcified plaque, and they may lead to undertreatment, where detection of plaque by a carotid ultrasound or by CTA, could help implement earlier treatment.

**Dr. Gluckman:**
Perfect.

**Dr. Taub:**
And I want to add, this is a patient where the current guidelines would really encourage us to think about a PCSK9 inhibitor for multiple reasons. There's an elevated coronary calcium score, but there's also an elevated Lp(a). And so, when I see a patient with an elevated lipoprotein(a), I always try to figure out how can I get them on a PCSK9 inhibitor because that's what we currently have that's going to


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lower Lp(a) by about 25%.

## Patient Case 2: Additional Questions

**Dr. Gluckman:**
And these are some additional questions, Pam. Great additional comments. You nicely highlighted this.

Maybe, Pam, just one additional point, is this patient, much like our first patient, also had an elevated Lp(a) level. How do you tell people, screen first-degree relatives, siblings, children? What do you inform them about testing and what that means for them in terms of their risk?

**Dr. Taub:**
So, one of the simple ways to think about it is if you have an elevated lipoprotein(a), it really changes your category. If you're low risk and then you have an elevated lipoprotein(a), it takes you to the next level. And so, I think of the multiple risk enhancers, it's a very potent risk enhancer.

And so, when you detect someone with a lipoprotein(a), you want to get all of their first-degree relatives screened. That includes children, siblings. And I'm very aggressive. And because it's one in five people have it, and by guidelines everybody should, I use that as an opportunity to just say all your family members should be informed and screened, cousins as well, but at least the first-degree relatives.

**Dr. Gluckman:**
Perfect. We're going to move on to our third case. I want to keep us on time.

## Patient Case 3: M.L., 68-Yr-Old Woman

So, this is a different patient. This is a 68-year-old woman, M.L., who's a nonsmoker. Past medical history is significant for hypertension, type 2 diabetes, peripheral artery disease. You can see the ABIs listed there in chronic kidney disease. History of a small M,I six years ago treated with a stent to her RCA. Ejection fraction remains in the normal range despite her MI.

She had a recent acute coronary syndrome six months ago and now has an LAD stent to add to her prior stent to the RCA. She has a history of statin-associated muscle symptoms where she's had intolerance to three prior statins, developed significant myalgias each time, especially with higher doses, and has a family history of premature atherosclerotic cardiovascular disease, namely a stroke.

You can see the current lipid-lowering therapy up on the right, rosuvastatin and ezetimibe, and a number of other therapies, including dual antipolio therapy, an ARB, a calcium channel blocker, a beta blocker, and then metformin and empagliflozin.

## Patient Case 3: M.L., 68-Yr-Old Woman (cont'd)

The details about her vitals and labs are listed there for you. I'm not going to read through each of these, but there's elevated blood pressure, elevated BMI, a reduced estimated GFR, and an elevated urinalbumin creatinine ratio. Labs are listed on the right side of pertinence. In particular, elevated triglycerides, low HDL, elevated LDL, elevated ApoB, and a lipoprotein(a) of 125. You can see the hemoglobin A1C of 7.6%.

We're going to let you talk amongst yourselves. There's obviously a theme tonight, besides it being jazz-centric in New Orleans. We're going to ask you to think about what would you be doing about her current lipid management regimen? How would you change it, intensify it? Please talk amongst yourselves for the next couple of minutes, and we'll open it back up.

Thank you.

And again, for those that are online, I think, fairly straightforward. This is a patient with polyvascular disease, the presence of both PAD,


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as well as a prior myocardial infarction. She, in fact, is very high-risk, not only because she has polyvascular disease with both PAD and a prior MI, but has had a recent acute coronary syndrome separate from her prior MI, puts her clearly in a very high-risk category.

Current guidelines would support an LDL cholesterol goal of being less than 55 mg/dL. Her LDL is 96. She's got elevated triglycerides, low HDL cholesterol. Her ApoB is elevated, and her lipoprotein(a) is elevated, all of which may well be contributing to her cardiovascular risk. So, treatment intensification is warranted. Despite that fact, she's on a statin, and likely maximally tolerated statin therapy, but her LDL remains elevated, and so we need to be doing something more.

The question is, what could and should we be doing for this patient population?

So, we're going to take a minute or two for those of you who are online. I'll be quiet just for a short, brief moment while we get the groups assembled here in the room, and then we're going to be coming back in just a minute or two to further address questions that you all have.

For those that are online, please continue to type in questions that are top-of-mind for you, and we're going to do our best to save as much time for Q&A at the end.

So, I've let those online know that we're going to come back in about a minute, so we'll give you another minute at your tables just to sort of think about what you would be doing for this patient with statin-associated side effects, statin-associated muscle symptoms, and the opportunity for intensification of therapy.

Again, as questions come up, please put them in through your tablets so that we can address them in Q&A, and we'll obviously lean on our experts to help weigh in on recommendations here overall.

Viet, I'm going to come to you first in terms of questions. So I'm going to come to you in about 30 seconds first.

# Patient Case 3: Panel Discussion

Okay, we're going to go ahead and get started. I know there's a theme that you've heard a lot about today, of people who have multiple cardiometabolic abnormalities, cardiovascular kidney metabolic syndrome or disease.

Viet, what do we need to be thinking about in this patient who has a lot of complexity but also has had some challenges in tolerating therapy? Where do you begin?

**Dr. Le:**
Lipid, first kind of thought process. You know, we have an Lp(a) of 125 nmol/L. Their LDL cholesterol is 96. This is someone that's very-high risk just by having two different events, but even just one event and multiple cardiovascular risks, they would be an LDL cholesterol of less than 55. And you're just not going to get there adding bempedoic acid, arguably with a stat, and it's a 15% reduction. So you're going to want a PCSK9, which will then hit kind of three things: LDL cholesterol, ApoB, Lp(a).

But this is also an individual that still has a class 2 obesity. They have an EGFR that's low and triglycerides HDL. So, you're probably thinking at least reduce it, icosapent ethyl, and then a GLP-1 through the select trial in order to hit all those things. But we also see that GLP-1s combined with this SGLT inhibitor will be protecting the kidneys as well.

So, a lot of things to think about in this person.

**Dr. Gluckman:**
Great thoughts. Pam, anything else to add?

**Dr. Taub:**
No, I agree. I think that the initial strategy would be to start a PCSK9 inhibitor, but I challenged my table, and I said, let's say that even with the PCSK9 inhibitor, the LDL was 70, then you could also add on bempedoic acid if you needed to get the LDL to less than 55. And there are some patients that do require statin, ezetimibe, PCSK9, and bempedoic acid. And we should be very comfortable using combination therapy.

**Dr. Gluckman:**
Erin, I'm going to let you add too.

**Dr. Michos:**
Yeah, so I just want to, first of all, clarify the ApoB goals. So the nice thing is on treatment is your ApoB goals are the same as your LDL


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goals. So her LDL goal is less than 55, her ApoB goal is less than 55. When the LDL goal is less than 70, the ApoB goal is less than 70. The only difference is primary prevention. When the LDL goal is less than 100, the ApoB goal is less than 90.

So, we want to get her LDL and her ApoB less than 55. So, we all just talked about PCSK9 being a good option. I discuss all the options with patients for individual choice.

She's a Medicare-age patient that sometimes with the monoclonal antibodies, even if they're covered by insurance, there could still be an out-of-pocket cost. So I also discussed the option of inclisiran. A patient with Medicare with supplement, often there could be no cost to them because the way it's delivered, given on-site as opposed to out-of-pocket medicine, it almost can be covered entirely. And some patients might like they don't want to give themselves injections or frequent injections. They might prefer the twice-yearly injections. So, I discuss all the options.

You'd get about a 50% lowering with LDL, with inclisiran, and a similar lowering of Lp(a), about 20-s25%, just to let the patient make the decision about what agent that they would want to use.

**Dr. Gluckman:**
I love that. Thank you all. So we're just adding one more thing.

# Patient Case 3: M.L.’s Lipid Management

If lower is better, and maybe, Pam, I'll have you weigh in on this, is 55 where we should stop? Should we be pushing people to even lower levels? What are your thoughts about that?

**Dr. Taub:**
So, I think 55 is kind of the minimum criteria that we need to meet. It's like getting a C on your report card. I think to really get that A, especially in these very high-risk secondary prevention patients, we want to be driving the LDLs into the 20s or 30s.

That's what I do in my clinical practice. And the analogy I always tell people who say, "Oh, is that too low?" Well, babies are born with an LDL in their 20s, and they do just fine from a neurocognitive perspective. And we've also seen in the PCSK9 trials, when people have very, very low LDLs in the 20s, there's no increased side effects. There's no increased neurocognitive events. So, we should feel comfortable driving it low.

And then we've seen in studies such as PACMAN-AMI, and HUYGENS, where they looked at patients on PCSK9, there's a decrease in the plaque volume, and also the fibrous cap becomes thicker. So, there are a lot of advantages of getting that LDL as low as possible. There's plaque stabilization, and there's also regression of the atheroma volume.

# Patient Case 3: Treatment Considerations

**Dr. Gluckman:**
And I just want to highlight on this slide, we have a huge, really, panoply of treatment options that are available to reduce cardiovascular risk. Some of the therapies that we think about traditionally were developed for other indications, but at the end of the day, they're reducing cardiovascular risk. I think we are blessed in the preventive cardiology field, and we have three real experts in the field here right now, are more the challenge of how do we balance the polypharmacy affordability access-related issues.

It's rare that we've got a toolbox that's empty that we can't reach for in these individuals. And obviously, these are complementary to one another and provide incremental cardiovascular risk reduction overall. I think these themes have largely been covered by the individual experts, just in terms of the range of therapies that we have that focus on LDL and Lp(a) lowering.

Viet mentioned icosapent ethyl based on REDUCE-IT in those with elevated triglycerides, diabetes, or established atherosclerotic cardiovascular disease. We've talked about GLP-1 receptor agonists; could add SGLT-2 inhibitors to the mix. And as we start moving into the CKD and diabetes, adding a non-steroidal MRA, finerenone, GLP-1 receptor agonist. We've got a lot of therapies. And at least the ACCHA, all publicly known, it's on their website, are expecting a cardiovascular kidney metabolic syndrome guideline hopefully later this year.


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And I think we just need to be thinking about all of these three patients tonight that there's a common thread of a wide range of abnormalities that increase cardiovascular risk. Clinical inertia cannot get the better of us, and we need to be doing things to get it better.

## Patient Case 3: Discussion Points

So, I'm going to invite our panel back. This is, again, some of the key points. We'll bring our panel back up to the stage. We're going to be going through those same questions that we asked initially, but I also want to leave time.

There are a lot of great questions that we did not get to tonight as part of these three cases that I just want to feed in the remaining minutes that we have, still have about 20 minutes, to be able to answer some questions related to these cases or things that you all are experiencing in practice overall. These are just a number of the discussion points. We're not going to solve the cost-financial toxicity. Erin brought up some alternatives depending upon the individual patient, and increasingly cognizant of the challenges of adherence with increased pill or medication burden.

## Patient Case 3: Follow-up With M.L.

So, I just want to highlight for this case, ML is started on evolocumab, repeating the lipid panel after six weeks. You can see the improvement in their lipids overall. LDL is now 38. ApoB, to Erin's point, very much goal-wise, should be paralleling up our LDL cholesterol goal. And we've seen significant improvement in their Lp(a).

To Pam's point brought up earlier, while it's one of the more effective therapies that we have for lowering Lp(a) today. Not an approved indication for that. We do have a wide range of therapies in development now that are going to be able to more effectively lower Lp(a). And we all look with earnest at those outcomes trials, the first of which hopefully will be presented this year to help provide context about this.

And, you know, I think of this as sort of a potentially a more complex puzzle that hopefully we on this stage and others out there can help to simplify for all of you. But the number of therapies may even grow that much more. And how do we intersect these therapies? It's easy to say, yes, more. But we want to be cognizant of the challenges for you all as clinicians and for patients as well.

To the point that Viet brought up earlier, the patients also started on semaglutide, improvements in A1C, BMI, urinalbumin-creatinine ratio. And again, our goal as clinicians is to work through a multitude of different mechanisms to mitigate their risk overall.

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