

Lp(a) Clinician to Clinician Transcript

Speaker 1:

Welcome to the American Heart Association's Heart 360° Podcast. This podcast brings together diverse groups of clinical experts from around the world to share their expertise, insights, and models on how to implement guideline-directed medical therapy to combat the number one cause of death, cardiovascular disease. Each episode contains focused professional conversations for providers across the spectrum of care, targeted at improving patient outcomes through quality improvement. And now today's episode.

Jeremy Skinner:

Hello and welcome to today's episode of the Lp(a) Discovery Podcast, a conversation about lipoprotein(a) between a community clinician and an expert. My name is Jeremy Skinner, program consultant and host for this podcast episode. As part of the Lp(a) Community Health Center Discovery Project, we aim to assess current practices and identify barriers to Lp(a) testing in community health centers, establish site-specific improvement goals, enhance clinician knowledge of Lp(a) and its role in ASCVD risk management, and improve patient education to support shared decision-making between clinicians and patients.

Today we'd like to welcome two clinical champions from our Lp(a) Discovery Projects. From Bay Area Community Health in Fremont, California, Dr. Reema Menezes, medical director. And from Massachusetts General Hospital, Dr. Kaavya Parachuri, clinical operations director for cardiovascular medicine. Let's start with some introductions. Dr. Menezes, would you mind going first followed by you Dr. Parachuri?

Dr. Reema Menezes:

Yes, absolutely, Jeremy. Thank you so much. First and foremost, I would like to thank you and the AHA for this opportunity. It is an honor to participate in this discussion with Dr. Parachuri about an extremely important topic. I am really looking forward to gaining some insights from her about lipoprotein(a) and sharing my thoughts on how clinicians in primary care can approach this topic. A little bit about myself. I'm a family med doc by training and I'm the medical director at Bay Area Community Health, which is a large federally-qualified health center in Northern California. We serve a racially and ethnically diverse patient population from the East Bay Area and the South Bay Area communities.

I'm also the clinical champion for chronic disease management at my health center, and I hold a keen interest in cardiometabolic disease prevention and one of my areas of focus at my health center is to improve hypertension and diabetes-related clinical quality metrics. I am passionate about cardiovascular disease prevention and I have been involved in educating providers and staff at my health center about heart disease and its risk factors and sharing evidence-based clinical guidelines and best practices.

My Lp(a) discovery journey began last year when our health center was selected to participate in the Lp(a) Discovery Project initiated by the AHA to improve Lp(a) awareness amongst patients and providers and promote testing among community health centers nationally. So now I am also a Lp(a) provider champion. And I have to admit that prior to this project I don't think I had a great deal of knowledge about Lp(a) and I hadn't been testing. I had heard about Lp(a), but I felt it was a test which was mostly ordered by cardiologists for high-risk patients. I didn't know what I should be doing with the results and how to manage patients with high Lp(a). All that has now changed with the wealth of knowledge that we

have received from the EHA. And currently I have been screening my patients for Lp(a) and I've seen elevated levels on many of my patients and I'm able to better manage them.

I have learned a great deal in the last day and I'm so excited to be here to help raise Lp(a) of Lp(a) among other clinicians in primary care to improve their understanding about Lp(a) and to help prevent cardiovascular risks in their patients. Dr. Parachuri?

Dr. Kaavya Parachuri:

That's a very tough introduction to follow, Dr. Menezes, and I'm very excited to be here and to speak with you about Lp(a) as well. My name is Kaavya Parachuri and I'm a clinical and preventive, suppose, a research cardiologist at Massachusetts General Hospital. So I see patients in primary prevention clinic for various reasons, including cholesterol problems, elevated Lp(a), or generally high cardiovascular risk of unclear causes, which I try and help investigate. I also see patients in a cardiovascular rehabilitation clinic and I'm part of the secondary prevention process as well. So the recovery and prevention of recurrent heart attacks for cardiovascular health. Lp(a) is a very near and dear topic for me as I do a lot of research in this space as well, and it's been really exciting to see some of the knowledge and discoveries that we've gleaned from various clinical trials and biobanks and other research repositories start to filter more into clinical awareness and clinical practice as well. So I think this is a great time for us to talk about how this is not just for the cardiologist, but is for all physicians, especially primary care physicians and providers.

Jeremy Skinner:

Thank you both so much. So Dr. Parachuri, would you mind starting us off by giving us some background on Lp(a) and the importance of testing?

Dr. Kaavya Parachuri:

Lipoprotein(a) or Lp(a) is a type of lipoprotein that consists of low density lipoprotein called LDL, that's attached to a unique protein called apolipoprotein(a). What sets Lp(a) apart from other lipid particles is its genetic basis. Levels of Lp(a) are largely determined by genetics and remain relatively stable throughout an individual's life. In fact, Black persons of African descent and South Asian populations are reported to have higher levels of Lp(a) than any other ethnic group. Despite being discovered decades ago, Lp(a) has been underappreciated in clinical practice. It's been linked to an increased risk of atherosclerotic cardiovascular disease or ASCVD, such as coronary artery disease, stroke, and even heart valve diseases, yet it's not routinely measured in most lipid panels. For that reason, it's really exciting for us to be talking more about it today to help address and hopefully reduce this gap in care. And I'll hand it over to Dr. Menezes to talk a little bit more about why we are here today.

Dr. Reema Menezes:

Thank you, Dr. Parachuri. Absolutely. We are here today because there is a critical gap in awareness about Lp(a). 20% of the global population has elevated Lp, and our goal today is to shine a light on this common yet often neglected lipid, explain why it's important for patient care, and explore the latest research that can help all clinicians identify, assess, and manage Lp(a) levels effectively. So Dr. Parachuri, let's dive right in. Lp(a) was discovered many decades ago and still since its discovery there has been a lot of interesting research that has been ongoing and we know a lot about Lp(a) now than we knew before, although some things are yet to be discovered. I have been reading a lot on this topic lately and I see a lot of missed opportunity for screening for Lp(a), especially for primary prevention of ASCVT. There are clinicians and healthcare providers who are not aware of this lipoprotein(and those who are aware

may not understand how to manage patients' elevated levels. Could you explain what Lp(a) is and what risks are associated with elevated Lp(a) levels?

Dr. Kaavya Parachuri:

Sure. That's a great question to start us off. So as we were talking about earlier, Lp(a) is a cholesterol particle that is very similar in structure to the LDL molecule, which is often thought of as the bad cholesterol that circulates in our bloodstream. Both LDL and Lp(a) contain the ApoB-100 [inaudible 00:08:38], which is the particle that we believe is most likely to be driving some of the atherogenic properties that we see of both of them. However, Lp(a) also has an apolipoprotein(a) molecule, hence its name, and that apolipoprotein(a) is structurally similar to plasminogen and therefore we think it might promote more of a prothrombotic state. Another important aspect of Lp(a) is that it has a number of Kringle-IV domains that repeat. So individual patients might have different numbers of repeats and that causes different isoforms of Lp(a) in terms of how big or how small those molecules are. And larger isoforms are generally thought to have more retention and degradation in the liver. Therefore, larger isoforms have lower plasma levels and less risk associated with them.

Another aspect of Lp(a) is that it has high levels of oxidized phospholipids and these promote inflammation and calcifications which have a large driver of not just pro-inflammatory burden, but atherosclerotic cardiovascular disease, and make it an independent risk factor for ASCVD. I think it's challenging because there's been no consensus among medical organizations about universal screening for Lp(a). There are some relative indications we think of patients with family history premature ASCVD. A personal history of early ASCVD without major risk factors should be another red flag to think about out-of-the-box risks to consider. That being said, across European versus American guidelines, there is really a wide spectrum with the European Atherosclerotic Society saying back in 2022 that we should be testing everybody once per lifetime. But the AHA guidelines, even in 2022 saying we only test if there's a personal or family history of ASCVD or if you're cascade screening within a family with elevated Lp(a).

Dr. Reema Menezes:

Thank you, Dr. Parachuri, for the excellent explanation and for your insight. So from what I'm understanding, Lp(a) is like a triple threat. It is pro-atherogenic, it's pro-thrombotic, and it's pro-inflammatory. And studies have shown that it is six times more atherogenic than LDL on a pro-particle basis. And you did share some screening guidelines. I did want to ask you about the NLA, National Lipid Association guideline. I think there was a shift from secondary prevention to more of primary prevention and they're recommending screening for Lp(a) once in your lifetime for adults. Can you share a little bit about the NLA guidelines from 2024?

Dr. Kaavya Parachuri:

Yeah, actually, Dr. Menezes, the National Lipid Association has always been ahead of the curve in terms of Lp(a) recognition of a cardiovascular risk factor. So even back in 2019, the NLA was advocating for us to test everyone once per lifetime. The tricky aspect of this, we can all agree that there is meaningful information in many patients. However, there are disparities in terms of insurance coverage of this test, especially in the primary prevention space that has sometimes led to lower adoption of this guideline, especially given the AHA's recent guidelines don't yet incorporate screening universally in the United States. I'm hopeful that this might come in the near future, but we have to wait and see.

Dr. Reema Menezes:

Absolutely. And I'm hopeful too to see an update whenever that comes from the AHA and the ACC to support the European, the Canadian, and the NLA guidelines. One of the things that I feel is that it could be not having clear screening guidelines may have been one of the barriers for screening, but we can at least depend on the NLA guidelines to improve screening for Lp(a). What I wanted to ask you is about cascade screening. What I feel like it's really important to know about cascade screening because screening for Lp(a) does not just reveal an individual's risk, but it also helps to reveal a family's risk. So can you speak a little bit about cascade screening?

Dr. Kaavya Parachuri:

Definitely. We've talked about how Lp(a) levels are stable over the lifetime, and that's due to its autosomal codominance inheritance patterns that we see with Lp(a). Most of the genetic influence that we see about Lp(a) levels are based on the Lp(a) gene primarily, or secondarily on the ApoB or ApoA genes. Because of this, cascade screening can be really high yield. When we talk about cascade screening, this is testing siblings, parents, and children, or first-degree family members of patients who have elevated Lp(a). And screening is thought to be highest yields if the level is above 100 milligrams per deciliter or above 200 nanomoles per liter. However, we advocate cascade screening with anybody with an abnormally elevated Lp(a).

Dr. Reema Menezes:

Thank you so much for sharing. So cascade screening would include first-degree relatives like parents, siblings, children of those patients who have elevated Lp(a). So that would help us to identify so many more patients. And we are lucky that now there are ICD-10 codes for elevated Lp(a). And for those who are interested, those codes are E78.41 and for family history of elevated Lp(a), that code is Z83.430. And I also wanted to share that this is a pretty inexpensive test for a highly prevalent condition. One in five people have Lp(a) and insurance coverage is getting better. So I think it's a no-brainer that we should be screening more folks to help find out what their overall cardiovascular risk is, including Lp(a), because it is an independent risk factor. I also wanted to ask you about diagnosis. What are the assays available to diagnose Lp(a)? Can you share a little bit about that?

Dr. Kaavya Parachuri:

That's a great question as well. There is some confusion about what's considered a high Lp(a) value and this is because there have been historically two different measurement options and there isn't an exact conversion between the two. One of them is mass, and so that's nanograms per deciliter. And the other assay is concentration, which is reported out in nanomoles per liter. And this has to do with that isoform different sizes that we notice with the Lp(a). So different isoforms might have different mass and give you variability, whereas with concentration, it's felt to be more standardized across individuals and across populations. The European Atherosclerosis Society suggests an interim conversion factor of two to two and a half to provide some guidance in interpreting data from the clinical studies that are out there, but there's no clinical conversion from one assay to the other.

When we think about interpretation of these, we consider the threshold of low risk to be under 30 milligrams per deciliter or under 75 nanomoles per liter. And there's usually an intermediate risk category from 30 to 50 milligrams per deciliter or 75 to 125 nanomoles per liter. And then into the high risk range anybody with an Lp(a) above 50 milligrams per deciliter or above 125 nanomoles per liter. We consider that a risk enhancing factor and it's been incorporated into many of the risk calculator algorithms that we use today. So another scenario that I often use in my clinical practice is to look at

Lp(a) in patients who might have an intermediate risk on the traditional PCE or the traditional AHA, ACC calculators to help re-stratify them as higher or lower risk from that intermediate bucket.

Dr. Reema Menezes:

That is really great information. I just wanted to add that I believe the NLE is suggesting to look at Lp(a) as a continuum of risk. So patients are still at risk if they have moderate levels. There are studies that are showing that if they have moderate levels of Lp(a) and risk factors, they could still be at risk. But those patients who have high Lp(a) levels, they are so much more at risk of having cardiovascular disease. So thanks for sharing that. I also read that there's a lot of variability in the tests at certain points in your life and in certain ethnicities. You had alluded to this earlier. Can you share a little bit more?

Dr. Kaavya Parachuri:

Yeah. There are some non-genetic factors that can affect Lp(a). Some things increase Lp(a). These can include menopause, of use of hormone replacement therapy, chronic kidney disease, or hyperthyroidism. Conversely, liver disease and chronic inflammation can give you a falsely low result. So although we generally say that this is stable over lifetime, there are those patients in whom a repeat test might be indicated if they're not at steady state in terms of management of these co-risks. So if they have treatable thyroid disease, they start renal replacement therapy, or they started renal transplant, you might want to recheck Lp(a) in those patients. It can be up to a 20% change related to those non-genetic factors.

Additionally, the distribution and median values vary across different ethnic population cohorts, and this can be up to a three-fold difference depending on your ethnic background. So Black individuals or individuals of African ancestry have the highest distribution and median values followed by South Asian, Hispanic, white, and Chinese. The ethnic background effect is mediated by the genetic ancestry, so it's hard to disentangle those two. Research studies though have shown that the ASCVD risk increases similarly across individuals in each group. So it is a continuum of risk with high values meriting consideration no matter your ethnic or genetic background.

Dr. Reema Menezes:

That is great information. Thank you so much for sharing that. And it is a non-fasting test, but if you order it with the lipid panel, then you would have to order it fasting.

Dr. Kaavya Parachuri:

You're right, that testing is routine non-fasting blood draw. So it should be very easy to add on and have it drawn at the next set of labs. Because it is stable, because it is a chronic risk factor, it doesn't have to warrant another fasting blood draw. It can be added on to a routine or annual tests that you might be doing as a primary care provider.

Dr. Reema Menezes:

Great, thanks for that information. From what I understand, once in a lifetime, but there are certain diseases or conditions, especially menopause, chronic kidney disease, or certain other risk factors where you can order it more often. Can you share a little bit about how would you manage elevated Lp(a) levels and how would you convince providers who say that there's nothing they can do about elevated Lp(a) levels?

Dr. Kaavya Parachuri:

I do want to validate because it can feel frustrating, Lp(a) isn't significantly modified by diet or exercise, so it does sometimes feel like there's nothing to do. However, we can still modify other risk factors in the understanding that having a high Lp(a) means that we need to target lower thresholds of the modifiable risk. So intensifying your diet and lifestyle, intensifying your other cholesterol management, really focusing on that LDL threshold to lower it further from where you would've previously been aiming for.

Patients with elevated Lp(a) also derive a pretty clear benefit from statin treatment even though the Lp(a) itself is not effective at the statin, and that gets at that intensification of modifying the other ASCVD risk factors that the patient may have. So lowering their LDL, addressing any diabetes risks, blood pressure that isn't well controlled. All of these things do still add to the cardiovascular risk, and Lp(a) can be a really good motivational factor in patient-based or in shared decision-making with a patient to help intensify their regimens.

Dr. Reema Menezes:

That's a great approach. And I would like to just share that since I have started checking for Lp(a), I have always used that as a risk-enhancing factor. See, I have a patient who I am doing a 10-year ASCVD risk calculation for if they are at... Especially in primary prevention, if they are at that borderline or intermediate risk, if the patient also has elevated Lp(a), I am going to intensify their treatment. If they're already on a moderate intensity statin, I may change it to a high intensity statin or add combination treatments like Ezetimibe. So can we talk a little bit about combination treatments in primary prevention?

Dr. Kaavya Parachuri:

Yeah, so getting at the treatment considerations for patients with Lp(a), pharmacologically, we don't have a lot that has been FDA-approved that effectively lowers Lp(a). As I mentioned, the four statins and Zetia as you mentioned as well, or Ezetimibe, which is generic name, have no effect on the Lp(a) value in and of itself, but it can lower risk by lowering LDL. Similarly, bempedioic acid isn't known to change Lp(a), but it lowers cardiovascular risk. High dose Omega-3s can lower Lp(a) by around 5%, and then more novel agents such as PCSK9 inhibitors can lower Lp(a) values by 20 to 25%. There are also siRNAs that are directed at PCSK-IX, also siRNAs that are directed at Lp(A) that lower Lp(a) values by 70 to 90%, but these are not FDA-approved yet. There are large trials such as the Lp(a) HORIZONS trial, OCEAN(a) and Acclaim, Lp(a) that are investigating whether the use of these medications can help reduce cardiovascular outcomes in the longterm. They haven't reported out yet.

The other FDA approved therapy that is available for Lp(a) is apheresis, which can lower Lp(a) values by 50 to 85%. This is essentially dialysis for cholesterol. So it's not something that's going to be palatable for most patients to come in for weekly treatments, but it can be useful in those who are high risk or have recurrent events despite modifying or addressing all risk factors that are able to be addressed with alternate pharmacologic therapy.

Dr. Reema Menezes:

Those are great insights. Thanks for sharing. So essentially we need to focus on all the other risk factors to reduce the overall cardiovascular risk for patients, reducing the LDL cholesterol, taking care of patient's blood pressure, obesity, helping them to not smoke or focusing on smoking cessation to reduce that overall cardiovascular burden, is what I'm hearing from you.

Dr. Kaavya Parachuri:

Exactly. Lp(a) is the canary in the coal mine. So it's that warning sign that hopefully motivates the patient and the provider to really address all of those risk factors. The other aspect is we don't know what to do about Lp(a) in terms of what is sufficient in terms of risk reduction. Is a 20 to 25% clinically meaningful in terms of outcomes or do you need 90% reduction? Is it a threshold-based talking about those cut-ups, knowing that it's a continuum of risk should we aim for Lp(a) targets the same way that we do with LDL? Is very unclear at this time. And so we live in exciting times where we're going to find out more about these as the clinical trials are poured out. But in the absence of data, we're left with dealing with the traditional risk factors that we know how to deal with and are able to manage.

Dr. Reema Menezes:

That's a great point. And since you spoke about LDL targets, I wanted to know, there is some suggestion that for primary prevention we should target LDL-C less than 100, and for secondary prevention we target LDL-C below 70. But if there is Lp(a) in the picture, there is some consensus that the lower the LDL the better. Could you speak a little bit about what LDL targets you use when you have a patient with Lp(a)?

Dr. Kaavya Parachuri:

I completely agree with what you said, and I think that my handling of Lp(a) is that I will ratchet down one level in terms of goal for LDL. So if the patient's target would have been 100, I will bring it down to 70. If it would've been 70, I'll bring it down to 50. There are a lot of papers and trials and sub-analyses that have shown there really is no such thing as too low of an LDL. So there is no concern about being very low and there is still incremental risk benefit by pushing LDL lower and lower throughout these trials. So whatever you're able to do to lower it and using the Lp(a) as justification for intensifying pharmaceutical therapies or lifestyle is a really great method of reducing some of that sixfold, some of that risk that we see.

Dr. Reema Menezes:

Great. And since you mentioned about lifestyle, let's not forget a heart healthy diet and physical activity, which is the cornerstone of ASCVD prevention. I also wanted ask you about treatment options. It's an exciting time for Lp(a) treatments because there are so many drugs in clinical trials and there is one, the antisense oligonucleotide Pelacarsen that is furthest along and maybe there may be an outcome soon, hopefully within the next couple of years. So can you share a little bit about the new research that has been going on with Lp(a) therapies?

Dr. Kaavya Parachuri:

I'm happy to talk about that. I am actually one of the investigators on the Lp(a)HORIZONS trial, which is looking at Pelacarsen in patients with elevated Lp(a). As an investigator with the Lp(a)HORIZONS trial, I think that it's going to be really exciting to see what the outcomes are when they're fully released. There are many different trials out there in addition to Pelacarsen, the OCEAN(a) study, which is also close for recruitment, and we'll hopefully report out in the next two years or so. And the ACCLAIM-Lp(a) TRIAL, which is looking at another novel molecule lowering Lp(a) is still recruiting. For those out there who are listening who have elevated Lp(a), there's still time to get involved in these studies. By doing research and evaluating the outcomes longterm, we'll have a better understanding of what to target and how aggressive to treat both Lp(a), LDL in patients longterm.

Dr. Reema Menezes:

Thank you so much for sharing those newer therapies. There is also some new evidence coming out regarding aspirin in primary prevention of elevated Lp(a). Can you share a little bit about that?

Dr. Kaavya Parachuri:

That's a great question. I think that right now there still isn't a ton of consensus. Aspirin is one of those really hot topic things that comes up in cardiology every couple of years. Everyone should be on aspirin. Nobody should be on aspirin. Maybe some people, but who? At this point, I don't typically recommend aspirin as primary prevention for patients who have elevated Lp(a). If I'm thinking about pharmacologic therapy, I'd rather reach for a statin or to focus on those lifestyle modifications, which you so clearly elucidated earlier as something more discreet that they're able to work on. Whereas aspirin might give a false sense of security. There might be some benefit, but it hasn't really been shown to have longterm outcome data yet. So I focused on the things that I think are going to be the highest yield.

Dr. Reema Menezes:

That is very helpful to know. So we spoke about a lot. We spoke about the risks of having elevated Lp(a), we spoke about screening for Lp(a), diagnosis, how to re-stratify, risk factor modification, cascade screening, and the newer treatments. Is there anything else that you would like to add regarding Lp(a)?

Dr. Kaavya Parachuri:

I think that sometimes patients can be a little bit anxious about getting their Lp(a) checked because they don't really know what it means for them. And having a high value can also add to that anxiety burden. As a researcher, and I do a lot of work in genomics, we like to use a phrase, DNA is not destiny. So even though Lp(a) is genetically encoded, it is stable over the lifetime, that doesn't necessarily mean you're guaranteed to have a heart attack. There are still a lot of modifiable behaviors, modifiable risks that are out there for patients. And so I just want to emphasize that there is a lot of positive things that we can do to address this.

Dr. Reema Menezes:

That's great. Yeah. So you're not stuck if you have this risk factor, there's a lot that one can do to reduce their overall risk by focusing on other risk factors and leading a healthy lifestyle. Can you also share a little bit about what would you tell patients who are not willing to take statins to lower their LDL cholesterol? Do you have any tips for us?

Dr. Kaavya Parachuri:

It's a tricky conversation. It is a shared decision-making between the patient and their provider. I try to set goals that are reasonable, and so if a patient is really hesitant or resistant about a statin, I give them two months, three months to really effectively change their lifestyle. So increasing the exercise, cutting out saturated fats, and then we can repeat the LDL to see what that improvement is with the agreement that if we don't reach that certain threshold that we have decided on together, that we would start a statin. And statins don't have to be all or nothing. So you're not necessarily starting to map the max dose high-intensity statin, sometimes it does take a gradual initiation of lower doses for patient to be comfortable.

Statins have been around for decades. They have really good safety and efficacy data. That being said, they do get vilified in the media. So it can be hard when people are Googling things to get past all of the

negative reviews that are out there. But the people who do really well on statins aren't on the internet talking about how wonderful their atorvastatin or their rosuvastatin is doing for them, they're just living their lives.

Dr. Reema Menezes:

That is really important, yes. And shared decision-making, it's that clinician-patient discussion of risks and benefits that is so important to have that partnership with the patients so that they're on board with the treatment plan. So I am really happy that you mentioned that. Could you also share about calcium scoring, coronary [inaudible 00:32:05] calcium score is one of the risk enhancers in the AHA, ACC guidelines? When do you use this test?

Dr. Kaavya Parachuri:

So calcium scores are another really good risk assessment tool that's out there, especially in that group of patients who might be at intermediate risk or unclear risk because their risk factors aren't included in the clinical risk calculators that are currently in use. I use calcium scores as a zero-not-zero scale in helping decide with a patient about statin initiation. So if a patient's already on a statin or there's already clear benefit for a statin, I don't reach for the calcium score unless it's something I can use to help motivate the patient to come around to taking a statin medication.

Dr. Reema Menezes:

That is really helpful to know because if a patient is reluctant to take statin, then maybe this test could be helpful so that they can find out if they already have ASCVD, which is not manifesting. So thank you so much for all of those insights. It was really interesting to learn more about Lp(a). Can you share any last thoughts for this?

Dr. Kaavya Parachuri:

I really think that knowledge here is power. And so bringing awareness to both patients and providers about when to check Lp(a), what values to be worried about, and what to do to reduce the risk, that you don't have to be worried anymore is really important. And getting out there, talking to different providers, getting this information out there is really important. I'm excited to see what the next couple of years bring in terms of new therapeutics, and I think that now more than ever we should have a good sense of the patients who might benefit from those therapies as they come to market.

Dr. Reema Menezes:

Thank you so much. And I'm just going to add that cardiovascular disease is the leading cause of death and disability in men and women, and we all know that the improved cardiovascular disease outcomes, we need to focus on primary prevention for our patients. There's a lot that we can do as healthcare providers. We've just learned that Lp(a) is a common genetic independent risk factor for coronary artery disease, and we can help our patients by screening more, by early intervention and risk stratification, and aggressive risk factor modification. We could increase patient's engagement and awareness by shared decision-making. And screening could also help identify families at risk with cascade testing.

Lp(a) is definitely actionable and clinicians in primary care have more frequent contact with patients and have an active role in preventative health of patients. They also have a trusting relationship with patients and are well-positioned to raise awareness. So I'm just going to end by seeing that it would be used as to take action and continue this conversation. There are great initiatives like the Lp(a) Discovery

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Project and organizations like AHA who have been helping to promote Lp(a) screening and awareness and providers could go on their website if they're interested to learn more about Lp(a).

Dr. Kaavya Parachuri:

Yeah, the AHA toolkit is both for professionals and for patients are excellent resources to learn more about Lp(a), and I can't thank primary care providers enough. There are not enough preventive cardiologists to manage all of the cardiovascular disease that's out there. So your efforts as the primary patient's longitudinal care providers are really important in mitigating some of this disease risks that we see out there.

Jeremy Skinner:

Thank you both so much, Dr. Menezes and Dr. Parachuri, for sharing your valuable insights and care considerations and perspectives on lipoprotein(a) as a whole and lipoprotein(a) community health centers. To learn more about lipoprotein(a), listen to the Lp(a) podcast series and watch professional education presentations at www.heart.org/lpadiscovery. Thanks for your time.

Speaker 1:

Thank you for listening to today's Heart360! Episode. Today's content discussed current barriers and care and how these barriers have been addressed in the communities featured. These views do not necessarily reflect the American Heart Association, American Stroke Association's official position. The association does not endorse any product or device. For more quality improvement information and resources, visit www.heart.org/quality.