ADAPT PTP Functional Medicine Q&A with Tracey O'Shea

Wednesday, March 10, 2021

1. How would we treat for oxidation and [low-density lipoprotein] (LDL) receptor activity? Very high LDLs but low triglycerides. LDL peak size 227.3, HDL large apolipoprotein B [is] 113, and lipoprotein(a) is 38. No metabolic syndrome. (7:15)

Tracey O'Shea: Hello, everybody. Happy Wednesday. I hope everyone is having a great week. I'm going to give it a little bit of time. I know it can take a couple of minutes to get people in the door. I don't have any pre-submitted questions today. So [I] don't have anything to work off of until people start to file in and share their questions as we normally do in the session. If you're just coming in, I was saying that I don't have any pre-submitted questions. So we are able to dive right in. If people have questions, you are free to go ahead and start putting those into the Q&A section. [I'll] give everybody some time to come up with questions.

Maybe I'll just chat a little bit about some of the things that I've been seeing in my clinical practice. I know there's been a lot of discussion around hydrogen sulfide [small intestinal bacterial overgrowth] (SIBO). So that has been a bigger exploration and topic in our practice. And I'm in the works of updating the SIBO section, lesson seven, to reflect the new tests. And I've sat in on a couple of Dr. Pimentel's Q&As, just reading the room and figuring out what the community as a whole is using. I have to take some of that in perspective because a lot of the people involved in those Q&As are more in this—they're gastroenterologists. So they have a little bit of a different approach. But it's been quite interesting to see what they're using for hydrogen sulfide and developing a protocol on our own that includes some of the nutrients to reduce hydrogen sulfide levels in the body to help reduce [symptomatology] as well as experimenting with low-sulfur diets and what that means for people and how that might be able to integrate into treatment.

So that should be coming soon. It often takes four to six weeks at least for us to get all that developed and get it produced. But I did want to let you know that that's in the works. And it's definitely on our radar, and we're starting to utilize those protocols in practice already. So I'm at this place where I'm just waiting for people to finish and work through those protocols as a way to see how effective they are and what we're seeing clinically. So just some updates on the hydrogen sulfide SIBO stuff and the ibs-smart test, looking for post-infectious [irritable bowel syndrome] or post-infectious SIBO with anti-vinculin antibodies and anti CdtB antibodies. Chris has already done a podcast with Dr. Pimentel that really explains a lot of the background

behind it and what the theory is, and what they're using in practice. But now we're starting to actually do those tests and starting to use prescription and herbal prokinetics in an effort to jump-start the motility of the small intestine. So we'll touch on that a little bit in the updates for SIBO as well as what we're seeing in practice of actually using that information. So, lots of cool stuff [is] happening on the SIBO front, and I will make sure to give updates as we go.

Let's see, trying to think if there's anything else. I still don't have any questions yet, which is totally fine if you don't have any questions. But I definitely don't want to bogart the Q&A with too much discussion if you have questions. So I'll give everyone a little bit of time to put some questions in. [It] looks like there might be a question about cholesterol, but I have a feeling [you're] probably just taking some time to write that out. So I'll give you some time, Patricia. Cholesterol has also been something on my mind lately, and I've been doing some diving in and trying to navigate the lab testing and the markers and what the numbers mean and how aggressive to be, and it's a pretty complex system, and how all of these are interwoven and connected is intricate. So we're all learning as we go. But I think we've started to refine that process a little bit, or at least I have in my clinical practice, and [I'm] feeling a little bit better about being able to navigate those markers and which labs we need, and which labs we really don't need and which are lacking scientific validity, I think, is really where we're at.

Pete Attia and Dr. Dayspring have a lot of podcasts together, but the most recent one from September, I think, the last update was really helpful for me from the perspective of the preference for apolipoprotein B over LDL particle number and why. So I think that will also be [included in] some updates that we'll be coming out with.

Let'' see. Patricia, I see that you just say re: cholesterol in your question and answer section. So I don't know if you had a specific question about cholesterol or if [you] just [want] me to talk about it. I'll give you a chance to maybe, okay, here we go. Yeah, I just want to make sure.

"How would we treat for oxidation and LDL receptor activity? Very high LDLs but low triglycerides. LDL peak size [is] 227.3, HDL large apolipoprotein B [is] 113, and lipoprotein(a) is 38. No metabolic syndrome."

Let's break this down a little bit. So I'll tell you where I'm at, what I was mentioning after reaching out to Dr. Dayspring, and what his thoughts and approach [are] as far as which labs to use. So, I did some split sampling of oxidized LDL between Labcorp and Quest because I was just getting these gigantic numbers in Labcorp that were way off track with all my other numbers. I'm talking, like, 1000 for oxidized LDL, and things just felt really off to me. So I did some split sampling and then asked Dr. Dayspring what his opinion was. He essentially was like, these are just non-comparable between labs because the methodology that's being used in every different lab and the primers that they're using are so different that there's just really no way to be able to do split samples and compare oxidized LDL.

And on top of that, he also does not feel like it's a valid lab marker. Scientifically, there's not a lot of evidence to support what we think that that's showing and the connection between atherogenic risk or oxidation. I think Chris Masterjohn actually also sent out an email, if you're part of his email threads, saying, "Yeah, it's a cool number. But at this point, there's really not a lot of scientific literature and evidence to support the validity of that marker and what to even do with the markers." So, at this point, I would say that I have just decided to take oxidized LDL out of the running and the mix, because I just don't think it's reliable. And I don't think that we can really use it as a way to guide treatment.

So [it] makes it a little tricky, because I've been ordering it for people and then I have to go back and explain, "Well, thanks for getting this lab, but this is an ever-evolving topic, and we're constantly learning, and thanks for doing the lab. But after really looking and diving into this, I think the evidence just doesn't support us to use this marker." So I hope that answers your question from an oxidized LDL marker perspective. You have very high LDL but low triglycerides. You have some information about your LDL peak size, your LDL size, and your apolipoprotein B and lipoprotein(a). So if people are listening that have a little less information and knowledge base about cholesterol, I really do encourage you to go through Peter Attia's work, [and] listen to Chris's podcast with Dr. Dayspring. But that most recent September 2020, I think, [podcast] with Dayspring and Attia really give the most updated information.

So the markers right now that I'm using to track and use for treatment [are] apolipoprotein B and lipoprotein(a). So one note about lipoprotein(a): depending on which lab you're doing, it could be nanomoles per liter or milligrams per deciliter. So I think you do have to pay attention to that and do your conversions. Now, it took me about 15 minutes to find a way to do this conversion, and it is not easy to find online how to convert the units. Tricky. So what I'm saying is that depending on what those units are, it might mean something different. So, after also listening to Dr. Kara Fitzgerald's and Dr. Dayspring's podcast discussion, they talk a lot about lipoprotein(a) and the numbers and where they won and where the atherogenic risk is. So, I'm using lipoprotein(a), as I said, and apolipoprotein B.

I think that the goal, the number on Labcorp, I think, is less than 90 for apolipoprotein B. Dr. Dayspring and a lot of his discussions on podcasts suggest even less than 60 is a safe place to be if there's [a] history of cardiovascular disease, or significant familial risk, or lots of other markers that are indicating high cardiovascular disease risk. So, I haven't really come to a number just yet, but I'm probably targeting 70 to 80 is probably a good safe spot. Again, big picture, right? We're not just using one number; we're using the collaboration of lots of different numbers. So I'm paying a little less attention to the size, the LDL peak size, and HDL size. Again, Dr. Dayspring does a really good job of explaining this in a couple of different podcasts with various people. The number of HDL, there [are] some questions about if you go too high with HDL, is that really as protective as we once thought? And the question is, maybe, maybe not. We just don't know because it's not necessarily the quantity of the HDL, but the quality of the HDL. Are they doing their jobs? And the large versus small, I think, can matter. But as of right now, I don't think that there's a test that tells us [the] quality of HDL. I've asked him

before, and it just doesn't exist yet. So I don't know that we have a lot of information about being able to really pick through the HDL size and number and to really know how effective that HDL is just yet.

So, in that context, I would really pay attention to the apolipoprotein B and your lipoprotein(a). We know that lipoprotein(a) is probably a lot less likely to be impacted by lifestyle factors, supplements, diet changes, [and] getting rid of infections. So I usually only check that once. Sometimes I have gotten into a habit of checking it once a year or so because I have seen it change. I have seen it shift and go down. I think that's rare. But I have seen it shift. There is some literature about niacin and some other supplements to reduce lipoprotein(a). I haven't really done that yet in practice because I was always under the impression it was futile to do that. But I'm interested in it. And so I think I might add a couple of things if someone really does have a high lipoprotein(a). And again, that range for talking to or looking at Dr. Dayspring's work and Kara Fitzgerald's conversation and Peter Attia, less than 8 less than 10 of the milligrams per deciliter unit. So a 38, right, if that's nanomoles per liter; I don't want to confuse you too much, but I think the units do matter because the range that I'm most familiar with is the milligrams per deciliter. So you'll want to convert that number.

If we can't really push on the lever for lipoprotein, okay, yeah. So if the lipoprotein(a) of 38 is nanomoles per liter, I think what I ended up coming out to, I think that this is right for the conversion, is multiplying that times 0.4. So that gives you [a lipoprotein(a)] of 15 for 15 milligrams per deciliter. So that's pretty close, I think, to optimal range. Maybe that creates a little less risk on this spectrum when we're evaluating all of the numbers together. Even lipoprotein B of 113 is higher. I don't think it's astronomically high, but it's definitely higher than optimal. So the question really is how would we test for [it]. Okay, so that was the question, how do we test. I don't think you test for oxidation and LDL, to be honest, at this point. Like I said, I don't think there's enough evidence to support using the oxidized LDL. So I think you're doing [a] good job; I would stick to the apolipoprotein B, lipoprotein(a).

I often have also been doing myeloperoxidase, LP PLH2 fibrinogen activity as additional markers for [the] ecosystem of the vessel. I don't know if the validation of those [has] changed. I know that those were recommended at some point, and I haven't gotten there just yet. But I think that those are at least some supporting markers that I'll use when I'm testing for cholesterol and cardiovascular disease risk. So if I had someone [who has] an apolipoprotein B of 113, a lipoprotein(a) of 15, I think it will really depend on what else is going on with that person. Do they have significant familial risk? Did someone die of a heart attack before the age of 50? First-degree relatives. What's their Framingham risk score? Or the Reynolds risk score? Really looking at all of those pieces. And what's their primary [symptomatology]? Are they there because they're worried about their cholesterol? Then I might dive into that earlier. If they've got other things going on, then I think it's important to look at all the potential—I'm going a little bit off [on] a tangent, but no one else has written any questions, so I hope it's okay that I'm just going to talk about cholesterol. But when I'm looking at reasons for high cholesterol or reasons for high [apolipoprotein B], I will give people the list, the rundown.

We're talking about genetics, so there can be some genetic component. Usually, I think that that's a little bit more likely for people who have, as we know, familial hypercholesterolemia, a high lipoprotein(a), numbers that are way high. Total cholesterol is 300 to 400, [apolipoprotein Bs] that are pushing 200. These are, I think that's generally a little bit more clear. But I think there's still a lot we don't know about the full capacity of the genetic implications for cholesterol.

So we've got genetics; we have poor thyroid function. Thyroid hormone is required to activate LDL, so I think that I am checking thyroid function, making sure that that's optimal. I think gut infections can absolutely impact cholesterol. It doesn't happen all the time. But I definitely have had a handful of people where [they've] had pretty high numbers, and we've treated SIBO, gotten rid of the [*Helicobacter pylori*] (*H. pylori*), [gotten] the gut optimal, and that was the only thing we really shifted except for maybe some nutrients, and the numbers really came really back down to optimal level. I don't think that's the majority of the cases, but I think it's at least worth doing for multiple reasons. So we've got genetics, poor thyroid function, gut infections, chronic infections also, viral, tick-borne illnesses, those types of things. Metabolic dysfunction.

As you guys definitely know, insulin and leptin resistance, blood sugar dysregulation look a little bit different as I think you probably know that pattern, right? High triglycerides, low HDL, that's definitely going to give me much more of an indication that we're working with something from a perspective of metabolic dysfunction and insulin resistance. And that's probably where I'm going to put my focus mostly first to see if we can manage that stuff, see what happens to the rest of the cholesterol numbers. I've also seen HPA axis dysregulation and dysfunction play a role. These kind[s] of cortisol mismatch. I don't know that I've seen that as much in practice or if it's just really difficult to discern all of the things and which one is really having the biggest impact. Because as you know, the goal is to make your patients feel better, and sometimes we don't have the luxury of doing really individual treatment regimens to keep the variables perfectly separated. That's not always the reality of the situation. So I think it is a little hard sometimes to know which one of these imbalances is having the biggest impact on your cholesterol numbers.

But cortisol HPA axis, I think, can have an impact. And then, of course, heavy metal and environmental toxicities. So I listed just about everything that we're really focusing on from a Functional Medicine perspective. So if the numbers aren't astronomically high, like in this case, an apolipoprotein B of 113, I probably would work my way down this list and say, "Okay, let's make sure your thyroid is functioning correctly; let's get the gut optimized, make sure that you're detoxing, make sure that your hormones look good, make sure your metabolic function is good, and then kind of retest things." And if things still aren't budging, then maybe start looking at the heavy metal toxicity, environmental toxins, chronic infections, mold, going to those level two, phase two things, if that's in your wheelhouse. And then, if I walk away and everything looks good and is optimized, I also am looking at diet a little bit. I think most people aren't really that sensitive to saturated fat. But as we all know, there is a small percentage of

people that are, and that's usually the first question, "Are they on the carnivore diet? Are they on a keto diet with high saturated fat levels?

Just understanding the connection between that and the cholesterol doesn't mean—I think Chris sent out that article about is this just a compensatory mechanism for having high levels of fat in your diet? Does it really infer atherogenic risk, and what are the connections? And I think there's still a lot of questions between those things. And I don't think for every single person, that answer is the same. But sometimes I will do, if there's a lot of risk, a lot of family risk, a lot of cardiovascular risk markers, I really ruled out and crossed out a lot of other imbalances, then I will have people do a 30-day really strict hardcore Mediterranean Paleo diet where they pull out at least the major saturated fats that I have seen clinically impact numbers, which would be coconut oils, full-fat dairies, [medium-chain triglycerides] in their coffee, that kind of stuff. I usually still have people eat red meat if they're fine, just having more of the lean cut [meats]. And I do that for 30 days, and I retest labs, and I see what level of impact are we having?

It needs to be pretty significant in order for me to make a decision to say, "Okay, you're pretty sensitive to saturated fat per this diet. Does that mean that this infers risk? I don't know 100 percent. I can't say that for sure." But there are people that I see that make a really drastic difference for, and that's the template diet that we stick with because they don't really want to be on supplements, they don't want to be on medications, they have a pretty high significant family history, and that's maybe where we end up, and that really controls their diet, and I'm still really comfortable with the Mediterranean Paleo for someone to be on long-term. I think it's still nutrient-dense.

And then I think at the end of the day, if numbers are still really high, and I haven't gotten any budge in movement by going through my list, then we have a conversation about supplements and red yeast rice and tocotrienols and try that. And [we] usually have pretty good success with a supplemental protocol, if that's where we ended up. That was a very long answer to your question, but I hope it was informative and helpful, and maybe how to approach that and those numbers.

All right, I'll give everyone a little bit more time if anyone has any additional questions. I don't want to end the session early if people have additional questions. But if not, we'll maybe just go through our day. Any other questions? Anybody that's sitting in? I know we don't have too many people participating. But I just want to make sure there aren't any other questions.

All right, well, thank you so much for showing up. I know everyone's really busy, and we're all trying to make it work with our lives and our schedules. So I see you, and I appreciate you, and keep up the good work. I know you are really working hard through these lessons, and it'll all pay off, I promise. It's really fascinating to see us all work together and explore and learn. So I'm excited to be part of it with you. Have a great rest of your day. I really appreciate it. Don't forget that there [are] options to submit questions to me through the Q&A section. So if you

have questions throughout the week, feel free to send them my way. All right, everybody, have a great rest of your day.