

ADAPT Functional Medicine Q&A with Chris Kresser

Wednesday, May 26, 2021

1. Is it difficult to get the amount of [thyroxine] (T4) right in a growing child? This is a 10-year-old girl who's been diagnosed with autoimmune thyroid Hashimoto's [disease] (I assumed? 00:47). What could be the reason for the rise in [thyroid stimulating hormone] (TSH), which is at 14.4 now, when the T4 is looking good and previous blood levels have been stable? [This] 10-year-old [was] diagnosed with Hashimoto's [disease] two years ago, one year after being diagnosed controversially with celiac disease, and then had a high TSH of 20. Free T4 was 10.7; free T3 [was] 5.3. [She] has been on [an] altered T4 dose since. (01:42)
2. When you prescribe several supplements at once, do you introduce them one at a time or start them all at once? I'm just wondering if they're started all at once. If a patient has a reaction or positive response, how do you know what's contributing to that? (05:43)
3. The course laid out straightforward treatment protocols. However, it's not easy to gain access to all the supplements from Australia. Wellevate and Fullscript don't ship there. I've been directed by your team to Emerson, which has some but not all of the supplements suggested. Do you have any other suggestions for those that are studying internationally or a list of alternative supplements? I want to get the best for my patients. I'm sure there are others in the same boat. (09:15)
4. You mentioned [alkaline phosphatase] (alk phos) and [mean corpuscular volume] (MCV) being potential markers for thyroid dysfunction. What's the relationship there? (13:08)
5. Can you review lactate dehydrogenase (LDH) and how it's related to blood sugar control? If all markers of blood sugar are normal, but LDH is really low, is there [a] concern there? (16:54)
6. In your experience, if all other symptoms are supported, can autoimmune hypothyroidism in children burn out, and can we stop medication? (21:13)
7. Is continuing to take the T4 making it difficult to see remission? (25:32)
8. Follow-up on LDH. What would you consider to be really low? The patient I'm referring to has an LDH of 69. Is that really low? (26:42)

Chris Kresser: Hello, [and] welcome to the Q&A for May. Only one question [was] sent in, and it looks like maybe a few people [are] on the call. So, Ben and David and Teresa, go ahead and put any questions you have in the Q&A box.

And then I'll start with this question that was sent in by Angela. So her question is, **"Is it difficult to get the amount of T4 right in a growing child? This is a 10-year-old girl who's been diagnosed with autoimmune thyroid Hashimoto's [disease] (I assumed? 00:47)."** The second part of the question, **"What could be the reason for the rise in TSH, which is at 14.4 now, when the T4 is looking good and previous blood levels have been stable?"** And then there's some detail here. **"[This] 10-year-old [was] diagnosed with Hashimoto's [disease] two years ago, one year after being diagnosed controversially with celiac disease."** So it just looks like there was some disagreement among clinicians there on celiac [disease]. **"And then had a high TSH of 20. Free T4 was 10.7; free T3 [was] 5.3. [She] has been on [an] altered T4 dose since."**

Okay, thanks for that question. I'll say that this is not a hard and fast rule, but almost always, people with Hashimoto's [disease] and autoimmune thyroid conditions do better with the T4, T3 combo because they have trouble converting T4 to T3. So some of the things that inhibit conversion of T4 to T3 would be inflammation is one of the top issues, gut issues, because a lot of that conversion happens in the gut and the liver. So if someone has any preexisting [gastrointestinal] (GI) issues, which this girl certainly does with a potential diagnosis of celiac [disease], that's going to be a problem.

The nature of autoimmune disease itself is that this is a low-grade chronic inflammation. That would be even more true with celiac [disease] because that's another autoimmune disease in addition to Hashimoto's [disease]. So inflammation is almost certainly an issue here, and that's almost certainly reducing the conversion of T4 to T3. And then nutrient deficiencies, so iodine, selenium, and zinc being the big ones, but there are others, as well. And nutrient deficiency would not be unusual in the case of a child with celiac disease.

There's a number of things to consider there. But in general, I would say that if you haven't already tried T3 containing medication, so either natural desiccated thyroid that has both T4 and T3, or a combination of synthetic T4, like levothyroxine plus synthetic T3 Cytomel, then that's the number one thing that you might want to explore in that situation.

The other consideration, of course, is the autoimmunity and whether that's been adequately addressed because this is somewhere where Functional Medicine differs considerably from conventional medicine because in the conventional model, [if] someone has Hashimoto's [disease], they don't really care about that. They're still just treating with thyroid hormone; they're not really doing anything for the autoimmunity. But if we look at it from a Functional Medicine perspective, Hashimoto's is an autoimmune disease, and the thyroid malfunction is really just a symptom of that underlying autoimmunity. So if you're only dealing with the symptom by altering thyroid hormone levels with medication, you're not really treating the Hashimoto's [disease] from a functional perspective.

So I would consider an autoimmune protocol (AIP) diet, although that can be difficult for a kid this age, and you want to definitely work with a nutritionist and make sure she's getting all of

the nutrients that she needs. You might want to consider things like glutathione and curcumin, which have an immune regulating effect, possibly even low-dose naltrexone, which I think is pretty safe even in kids this age. So there are a number of things you can do to help regulate and balance the autoimmunity, and that's going to help improve the conversion of T4 to T3 and it's going to generally help any external support, whether that's pharmaceutical medication, or herbs or supplements or whatever, have a better effect. So hopefully, Angela, that gives you some food for thought for this case.

Teresa asked, "When you prescribe several supplements at once, do you introduce them one at a time or start them all at once? I'm just wondering if they're started all at once. If a patient has a reaction or positive response, how do you know what's contributing to that?"

Great question. There's no right or wrong answer. It depends what you're prioritizing. So if somebody is suffering a lot and they're in need of quick relief, then we will often have them start the supplements all at the same time. Because in that case, we're prioritizing rapid improvement and we don't care as much if we know specifically whether supplement A was more effective than supplement B or C. We're just looking for effectiveness in general. And then we can figure out which supplements were the most or least effective later when we start titrating them, taking them off of the supplements.

So if somebody, for example, let's use someone who is in a Crohn's disease flare and they're having multiple watery bowel movements a day or blood and mucus in their stool; they're really suffering. Their gastroenterologist is recommending more aggressive medication, maybe a biologic like Remicade or Imuran or maybe even surgery. I've had patients over the years who've been in that situation. And in those cases, we want to just get them out of that flare as quickly as possible. So we're not as concerned, in that case, with exactly which of the supplements is going to do that most effectively. I'm just going to use the ones that I will typically use in that situation and know as a group tend to be most effective. And then once they're in remission, ideally, or they're at least out of that flare, we can start walking back some of those supplements to determine which are the most effective.

On the other hand, if someone is not in those kinds of dire straits, and particularly if it's somebody who has reluctance to take a lot of different supplements, or is the kind of person that likes to isolate variables, then I'll generally suggest that they add them in one by one. And I will often ask my patients what they want to do. I'll even frame it in a similar way to what I just did for all of you. So I might say something like, "There are a couple [of] different ways of doing this. Neither is right or wrong; [it] just largely depends on what we're prioritizing, what's most important to you. We've got this protocol; we can just start them all at once. That will move us through it more quickly, and you have more of a chance of getting a good result, but if you prefer to do it in a more sequential fashion, [we can do that]."

Or here's another good example or another good consideration. If I have a patient that's super sensitive to supplements, and they know that historically, then we're not going to hit them all at once with all of those supplements. We're going to add them in one day at a time or one supplement at a time.

Okay. Ben asked, "The course laid out straightforward treatment protocols. However, it's not easy to gain access to all the supplements from Australia. Wellevate and Fullscript don't ship there. I've been directed by your team to Emerson, which has some but not all of the supplements suggested. Do you have any other suggestions for those that are studying internationally or a list of alternative supplements? I want to get the best for my patients. I'm sure there are others in the same boat."

Yeah, Ben, thanks for that. We get this question periodically, and the reason I provide specific protocols in this program is not actually because I expect you to use the identical supplements that I use in my practice, although I certainly don't think that's a bad idea, of course. But I do it because I'm a big believer in practical learning and case-based learning, and I think the best way to learn is just to get started and start using protocols rather than a more didactic approach that doesn't actually teach you specific supplements.

So, the reason I mention that is because there are lots of different versions of the supplements that I recommend that can be as effective in protocols. And indeed, I've used different supplements over the years. When I find one that works, I tend to stick with it. But if something new comes out that looks like it might offer an additional benefit above and beyond what my current supplement in that category offers, then I'll try it. And so, if you look at protocols, even in this course, we've changed and updated protocols over time.

I think a good way to start, Ben, is to look at the ingredients of the supplements in question, the ones you're not able to obtain from Emerson or that are part of the protocol that you can't easily get the same exact one in Australia. And then look at what you do have access to through whatever distributor that you tend to use in Australia, and see if the ingredients are pretty similar in lineup. And then also, ideally, you'd want to choose from a reputable manufacturer. Because as you know, there's a lot of shenanigans in the supplement industry. And when you look at companies like Thorne and Pure Encapsulations, and some of the other brands that I recommend and that we talk about, they have independent third-party quality control; they list COAs on their website, and you can just be a lot more confident that you're getting what the label says you're getting, and that there's been research and testing that has gone into the product.

So don't be too discouraged if you can't find the exact products. Know that I do change my own use of products over time. I'll use different products, if the product that I like is not available or out of stock, and I don't worry about it too much. Because it's pretty clear to me after more than a decade of doing this that there [are] many paths to the top of the mountain and that you can get really good results using similar products that are not the same ones that

I'm using in a protocol. But my hope is that in giving you these protocols, more than just expecting you to use the exact same products, that you see how I assembled the protocol. And we talk about this in the course, but you get a sense of how to put together a protocol yourself so that if those certain individual products are not available to you or become unavailable, you can swap them out and you know what role they're playing in that protocol. That's the most important thing. So hopefully, that helps, Ben.

Next question from Theresa. "You mentioned alk phos and MCV being potential markers for thyroid dysfunction. What's the relationship there?"

There's not a lot written on MCV and why hypothyroidism could potentially lead to a higher volume of the red blood cell[s]. I don't actually know what the mechanism there is, and I don't know off the top of my head what the mechanism is for alk phos either. I've done some research and tried to get more clarity on that, but I don't find very much that's written about it. What I would also say is that those are secondary or maybe even tertiary markers of thyroid dysfunction.

So I would never in a million years diagnose somebody with a thyroid problem just because they have elevated MCV and alk phos. It's more the kind of thing to just be aware of like supporting markers. Like if you're seeing some weirdness in the thyroid panel, and then you also happen to see that alk phos and MCV are out of range, and then you also happen to see that their free cortisol is significantly higher than their metabolized cortisol. And if you're not already at that point in the curriculum with the DUTCH test, you'll get there. Those are all supporting signs of a thyroid issue that would give you some clues that that might be going on, especially if the blood markers are equivocal or just starting to become slightly abnormal. Then maybe you're catching that person at an earlier point in their progression toward hypothyroidism. And they can be helpful in that regard. But beyond that, I don't think they're super useful.

It's also helpful to know, like, I've had a number of patients over the years, maybe you have, too, that just have randomly high MCV with no other indication. They're not anemic; they're not [vitamin] B12 or folate deficient or [vitamin] B6 deficient. They don't have any of the other hematological disorders that would lead to high MCV, and they've just got this randomly high MCV. And then, especially if they're not aware of a thyroid condition, that can be a red flag and a reason to at least test for hypothyroidism to cast a wider net and see if that's a reason for that elevated MCV. That has happened to me in a couple [of] cases where I ended up eventually diagnosing someone with hypothyroidism because of a high MCV. But that was only ever when they brought in their own bloodwork and I hadn't yet done the full case review panel. This is a good reason why we do the comprehensive blood panel up front because you catch things like that.

And certainly, if you know about the relationship with MCV and a thyroid panel, and you only happen to do a [complete blood count] up front, then that could prompt you to go on to do a

thyroid panel. But that rarely happens for me because I virtually always do the full comprehensive blood panel up front.

All right, guys. Anyone else? Any questions for me? Teresa, “Can you review lactate dehydrogenase (LDH) and how it’s related to blood sugar control? If all markers of blood sugar are normal, but LDH is really low, is there [a] concern there?”

Good question. So I would say it’s similar to what we just talked about with MCV and alk phos as thyroid markers. Lactate dehydrogenase requires a consistent supply of glucose entering the cell in order to maintain normal LDH levels. And so my understanding is that if LDH is low, then that could signal a problem with glucose entry into the cell. And that problem could be something like insulin resistance, reactive hypoglycemia, you might see in patients as a result of that. But again, if you’re thinking about blood work more as in patterns than as individual markers. There are certainly some individual markers; if the only thing you see is that marker being really high or really low, it’s still a big cause for concern.

For example, if you see [vitamin] B12 being really low and nothing else is out of range, which would be unusual, but it can happen, then you’d still consider that person B12 deficient and you’d want to do something about that. Or if you see that fasting glucose is 140 but somehow, A1C is normal, which, again, is unlikely, but I’ve seen that, that’s still enough to trigger a red flag and make you want to take action. But in general, we’re going to see patterns. So we’re going to see that maybe fasting glucose in this scenario might be a little higher, or it might even be a little low. And then you might have A1C be a little bit high. You might see triglycerides high because this person might have metabolic issues. And [high-density lipoprotein] is maybe a little bit low. You might see some difference in uric acid. If you have them do some post-meal blood sugar testing with the glucometer, you might notice that they have a post-meal blood sugar spike followed by maybe a three-hour value that’s actually low. So you’re seeing that reactive hypoglycemia pattern.

That’s usually what you’re going to see. And if they have just low LDH, especially if it’s low in the functional range, and there’s nothing else wrong there in the sugar picture, then I wouldn’t worry about that. If the lactate dehydrogenase is very low, so let’s say it’s well below the lab range, then that’s a different scenario. So that could be, and you’re not going to see this much in clinical practice. I’ve probably seen it once, maybe twice. But that could be lactate dehydrogenase deficiency, which is a genetic condition. That’s typically a glycogen storage disease; it’s caused by mutations in the *LDHA* or *LDHB* gene, or both. They’re inherited autosomal recessive patterns, so that person will always have a low lactate dehydrogenase.

And generally, you’re going to also see high creatine kinase (CK); you’ll see increased lactate and pyruvate, which isn’t typically tested. But [for] kids with metabolic disorders, [I] often might order that kind of testing for them. And then in a pretty large percentage of people, you’ll see myalgias, exercise-induced muscle fatigue, muscle spasms, and things like that.

All right, so Ben is actually Angela. Thanks for clarifying, Angela. “In your experience, if all other symptoms are supported, can autoimmune hypothyroidism in children burn out, and can we stop medication?”

That’s why I think it’s so important to treat the autoimmune part of it, especially in kids. So if you’re able to remove the agents that are provoking an autoimmune response. Let’s say it’s gluten. That girl had celiac [disease] and maybe was still eating gluten, and she’s responding well to a gluten-free diet. And so it’s entirely possible that on a gluten-free diet, her immune system is going to be better regulated, and it’s not going to keep attacking the thyroid.

And so I think it is possible and plausible that in that situation, you may see a spontaneous, what would be called in conventional medicine, spontaneous remission. I don’t think there’s anything spontaneous about it, because it’s caused by removing the offending agent. But that’s what they call it in conventional medicine. You can see that in kids. I mean, with Graves’ disease, even in adults, about 30 to 40 percent of patients, depending on the study you look at, will spontaneously remit. So that’s one of the, you have to be careful with Graves’ [disease]; if somebody[’s] TSH is super low and their T3 is super high, you have to intervene to prevent a thyroid storm. But if it’s just a mild incidence of Graves’ disease, I will often take a wait and see approach before, and I’ll let people know that those rates of spontaneous remission before they get their thyroid gland ablated radioactively or they take potentially toxic medication. So that’s just something to keep in mind.

As for stopping thyroid medication, I’ve had patients, I have a few, actually, just staying on the Graves’s [disease] theme even though it’s less common, the Hashimoto’s [disease]. I had a woman in her late 60s who had severe Graves’ [disease] her whole life. She had exophthalmos, the bulging eyes, all the classic symptoms. She’d been on, I’m trying to remember which [medication] she was on. I can’t remember which one, but it was for like at least 30 years, and she was extremely skeptical. And frankly, I was extremely skeptical, as well, that she would be able to get off of the medication. I think it might have been methimazole. Yeah, I think it was that. But we got her on a full AIP; we got her on [low-dose naltrexone] (LDN), glutathione, curcumin, [and] we did a whole autoimmune-focused protocol. And she got off the methimazole and was able to fully maintain a normal TSH and T4 and T3 levels just by taking LDN.

For kids, I think it’s even more likely that you can stop medication if these other factors are addressed. And it’s not like antidepressants, thyroid medication, where you have to titrate really slowly and all that. It still can be a good idea to titrate, but generally, there’s no dependence that forms or anything like that. So it’s totally possible to get off of that.

In the follow-up from Angela, “Is continuing to take the T4 making it difficult to see remission?”

It could be. Generally, we'll keep someone on medication until we're pretty certain that they're stabilized, and then we'll start to do experiments of them stopping their meds and seeing if anything changes with their thyroid. And again, unless they have Graves' [disease] and there's a risk of a thyroid storm and stroking out or something like that, for someone with hypothyroidism, the risk of just stopping their medication for a period of time under supervision is not that high. They typically will just, if they still need it, they're just going to start to experience symptoms again, which can range from mild to moderate to severe. But you can usually prevent severe [symptoms] just by intervening sooner. If they start to notice the early signs of hypothyroidism or symptoms, then get them back on the medication.

Teresa says, "Follow-up on LDH. What would you consider to be really low? The patient I'm referring to has an LDH of 69. Is that really low?"

That's pretty low, so I would retest that at least once more and see if it's still that low. And if it's consistently below the lab range, then you might consider referring them out to get some genetic testing to see if they have lactate dehydrogenase deficiency. Especially if they have any of those symptoms that I mentioned like muscle myalgia or exercise-induced muscle fatigue or muscle spasms. That's what's going to happen if there's not enough LDH.

Okay, anybody else? You're welcome, Teresa. I hope that's helpful. I'll just wait another couple [of] minutes in case anybody does have a question. Okay, folks. Well, thank you so much for being here, and I'll see you next month. And in the meantime, if you have questions, Tracey is, of course, more than capable of answering them. And I look forward to seeing you on the call next month in June. Take care, everybody. Enjoy the springtime.