Live Case Recordings #3 - Case Review Part One

Chris: Okay. Hello there. All right. Let me tell you a little bit about what we're going to do so you have an overview of the appointment. We're going to start by me going over the underlying patterns I identified in your blood work, all of your tests, and your paperwork. Then I'll make recommendations for further testing based on what I discovered, and then we'll talk about the first phase of the treatment plan. You, at any time, can ask me any questions you have that come up, and then there will be some time at the end as well for you to ask any questions that we may not have had a chance to cover.

Jonathan: Okay. Great.

Chris: Okay. I'll send you a report of findings after the appointment, which just outlines everything that we talked about, and then you'll get a really detailed follow-up from my staff outlining all of the next steps that we talked about taking.

Jonathan: Awesome.

Chris: Great. Okay. So, we'll start with the blood work. Pull that up here. All right, so your uric acid was low. The first thing I want to do is retest that just to see if it is a consistent finding or if it was just a one-time thing. There are a number of different causes for low uric acid. The most likely, in my opinion, is related to low levels of antidiuretic hormone, which we'll talk about in a little bit. That is one of the markers we use to assess chronic inflammatory response syndrome, which we talked about a little bit in the initial consult, the possibility that you were exposed to some kind of biotoxin in the freshwater, the pond or the lake; mold; or some other toxin like that which is triggering a systemic inflammatory response. One of the things that happens in that condition is that your ADH levels, antidiuretic hormone, drop, and that can actually lead to low uric acid. If I was a betting man, that would be what I would assume.

Jonathan: Okay.

Chris: But I want to retest it first of all to see if that is the case. I don't think it's anything to be concerned about. It's just one manifestation of this whole pattern that we're seeing with you.

Jonathan: Okay.

Chris: Next, your vitamin D levels are what I would call borderline toxic. Most nutrients we want not too little and not too much. We want to be in that kind of Goldilocks zone. Your 25(OH)D, which is the main marker that is used to measure vitamin D status, is at 90. It's 89.7, so it is basically 90. In the conventional model, conventional doctors would say anything over 100 is toxic.

I think there is pretty strong evidence to suggest that toxic effects can happen at levels over 70. Your 1,25 D, which is the more active form, is well outside of the range. The upper end of the range is 79.3, and you are at 119. Are you supplementing right now with vitamin D?

Jonathan: I have on and off. Right now, I'm not. I don't think at the time that I was getting the testing done with you that I was, but I'm not positive.

Chris: This is not really well understood, but there are some theories of immune dysregulation that could explain high calcitriol in particular, which you have, the active form of vitamin D. For example, the condition called sarcoidosis, which is an autoimmune disease that involves quite elevated levels of calcitriol, active vitamin D. In some cases, 25(OH)D, the less active form, will be actually low in sarcoidosis. I'm not suggesting that you have that condition, but it does indicate that in some cases of immune dysregulation that can cause these abnormalities in vitamin D levels even without supplementation. For now, I definitely don't want you to be supplementing with vitamin D. You don't need that. We may want to retest these markers after we address some of the things that we're going to be focusing on to see if those levels normalize, which I would expect them to, but if they don't, then we will probably want to do some further investigation there.

I'm going to actually add 25(OH)D and 1,25 D retest to your report of findings here. We'll do that after the SIBO treatment, I think.

Okay, so next is your aminotransferases. These are often referred to as liver enzymes. They were a little bit elevated, not outside of the lab range but above what I consider to be optimal. There are a number of potential causes of this as well. One is poor thyroid function, which we know you have, and there is some evidence that your thyroid is still not optimally functioning, which we'll come back to in a second, and we'll talk about in a moment. That alone could cause the problem. Certainly any kind of impaired detoxification ability or other conditions such as hepatitis, which I don't think is likely in your case; autoimmune liver conditions, which is pretty rare and again not the most likely cause. There are a lot of potential things that could cause this, but again, my guess is it is related to the thyroid, given that we already know that you have that and given that the enzymes are not particularly elevated. It may also be related to CIRS, chronic inflammatory response syndrome, if that is present for you. We've seen that in other patients who have CIRS and don't have any other obvious cause of elevated liver enzymes. Nonalcoholic fatty liver disease is probably the most common cause statistically of mild elevation in these liver enzymes, but there is no indication that you have that given your normal blood sugar and normal weight. It's not likely for you. It's likely to be another cause.

You have elevated alkaline phosphatase, just barely elevated, but this is an enzyme that is produced in a lot of different tissues such as the bone, the liver, and the gut. Whenever we see it elevated, we do a follow-up test called alkaline phosphatase isoenzymes, which tells us where the tissue is breaking down. First of all, we could do a follow-up, and it might be normal. This happens all the time where we see a transient elevation in this, and when we do a retest, the levels are normal. If the levels are still elevated, we want to know that, and we want to know where is the

breakdown occurring. You have elevated liver enzymes; it's possible that we'll see something happening in the liver. There is another marker of liver function that we ran called GGT, which is normal. That, to me, suggests that there isn't a structural breakdown of tissue happening in the liver and nothing serious going on there, but I want to retest the alkaline phosphatase and then do the isoenzymes so we can see where the tissue breakdown is occurring, if it is in fact.

Next is thyroid. I know you were diagnosed with hypothyroidism, and you're on WP-Thyroid now. Do you remember what your TSH level was when you were diagnosed, before you starting taking this?

Jonathan: I don't remember. At my first diagnosis, that was 2013. I know that my levels of TSH have drawn I think as high as around 10. I think the last time we had it checked it was—I've got actually the paper right here. My mom is sending it to me. Let's see. It looks like—does that 0.3? It looks like it was 3, but that doesn't seem to be—that doesn't make sense to me because—

Male: It was 07/21/2016. That is not the most recent.

Jonathan: The most recent was around maybe August, and I think I remember it being like 5 point something.

Dr. Nett: Chris, in July it was 0.3, so I don't know if there is a lot of fluctuation based on immune response.

Chris: Yeah, possibly. You're at 4.27 now. The lab range for TSH is misleading. The typical range is 0.5 to 4.5, but when you look at studies of people with normally functioning thyroid, their TSH ranges between 0.5 and 2 or 2.5 at the maximum. For optimal thyroid function, we like to see patients with a TSH in that range between 0.5 and 2.5. When it's higher than that, it suggests that despite the thyroid medication you're taking, your thyroid is still not functioning optimally. As Dr. Nett was just alluding to, in some cases where Hashimoto's is present, you can see a pretty significant variation in TSH levels. They just bounce around a lot. If we were to test it serially every week over a period of 12 weeks, you might see it go from 0.1 to 10 to 5 to 2 to 7, and what is happening there is that is just the relapsing-remitting autoimmune process happening. When the immune attack escalates, the TSH will go up, or it will go down depending on what is happening. If there is a release of thyroid hormone into the tissue because of the immune attack, then the TSH will actually go down because you'll have excess thyroid hormone. After that immune attack passes, the TSH will go back up because there has been a release of thyroid hormone. It goes in a cycle. I don't think the solution is taking more thyroid hormone right now because your free T3 is at 3.5, which is already toward the upper end of the range. If you took more thyroid hormone, you could quite possibly move into the hyperthyroid state. I think that we're just picking up variation that is related to immune dysregulation with the TSH. Your free T3 levels are optimal, and I wouldn't want to see them go higher. I think the way to address this is to focus on the immune dysregulation, which is really the root of the problem. Addressing all of the triggers that could lead to immune dysfunction is probably the way that we're going to get your thyroid more regulated.

Jonathan: Okay. At that time when we got that test done with you guys, I think I was on a slightly lower dose. Now I'm on a little higher of a dose. I don't know what impact that has on your thought process, but just so you know.

Chris: It makes me want to retest your TSH and T3 levels sooner rather than later just to make sure that you're not going into what we call facetious hyperthyroidism, which is a medication-induced hyperthyroid state. You were already at 3.5 with T3 with that lower dose when you took this test. I think it will make sense to retest your levels a little bit sooner rather than later just to make sure you're not going too high.

Jonathan: Is what you're saying is based on the TSH it would look like I'm hypothyroid, but based on the free T3, it looks like I'm almost hyperthyroid?

Chris: Exactly.

Jonathan: Because I'm feeling okay.

Chris: Based on these labs, your free T3 is high-normal. It's inching up. If it went above 4.0, you'd be in the hyperthyroid state. It seems unusual to have high TSH and high free T3 because, as you understand already, TSH is inversely related to T3 levels typically, meaning as T3 levels increase, TSH would go down not up. We see this phenomenon in patients with autoimmune disease. It's that the TSH is just fluctuating pretty regularly, so we just caught it in a moment of time. Who knows? The next day we could have tested it, and it might have been 0.3 again. There are studies that have shown that kind of variation can occur in this situation.

Jonathan: Okay.

Chris: We don't typically hone in and focus on that right away because there are so many other things that affect thyroid function, especially when the cause of the thyroid dysfunction is autoimmune, as it is in your case.

Jonathan: Okay.

Chris: Regarding triggers for immune dysregulation, since we've mostly made it through the blood work now, there are many different possible triggers of immune dysregulation, but of course, one of the main ones that we look at first is the gut. We did a SIBO breath test for you. We also did stool tests and urine organic acids tests to look at your gut function.

The breath test was strongly positive for SIBO. You had what is called a classic double peak, which is just the way that the results look on the chart. I don't need to go into the detail on that, but the significance is that when you see a double peak on the breath test results, you can be even more confident that they are accurate and positive. It strengthens the finding, basically. You had an increase in your hydrogen levels from 4 at baseline to 42 at 60 minutes. That is a pretty significant

increase. Then your methane levels were elevated right from the get-go. At baseline, they were 25, and then you had a peak of 49 at 60 minutes as well. The significance of that is that studies have shown that methane production leads to decreased intestinal motility and constipation, and I know that has been an issue for you. With this elevated methane, that is almost certainly contributing to the constipation picture.

Jonathan: Okay.

Chris: That is something that could definitely be related not only to your gut symptoms but also to the immune dysregulation and the skin stuff because there are a lot of studies that have shown very strong correlations with various skin conditions and SIBO. For example, acne rosacea in one study, it was 100 percent correlation, and 100 percent of patients with acne rosacea had SIBO, and a large number of them improved significantly when the SIBO was adequately treated. If someone comes into the clinic with skin conditions as a major complaint, one of the first things we do is look at their gut, and this is why.

Moving on to the stool tests, on the Doctor's Data stool test, there were a couple things there to talk about. Generally, your beneficial bacteria were pretty good overall except you had low levels of Lactobacillus, which is an important species, and no growth of Enterococcus. You had some commensal imbalanced flora present, which are bacteria that normally reside in the intestinal tract, but if it becomes overgrown or overrepresented, it can be a problem. No parasites, which is good. No fungal overgrowth, which is good. You did have elevated levels of lysozyme, which is a marker of inflammation in the gut. Your secretory IgA levels, which are also markers for inflammation or activated gut immune defense, were a little bit low. That, together with the lysozyme, suggests that there is some gut inflammation happening, which isn't surprising with SIBO. The pH, which is the acidity and alkalinity of the stool, was low, and that also typically reflects either dysbiosis, fungal overgrowth, or some issue in the gut.

Then you had positive for occult blood in the stool, occult meaning hidden, or in this context, dark. I will say that with Doctor's Data, this occult blood marker I've found it to be not very accurate. For that reason, we often will do a follow-up test with LabCorp for their occult blood assay. At least nine times out of 10, if not 19 times out of 20, it is negative, and we just move on. There have been a few cases where it has been confirmed positive, and because blood in the stool is something we would want to know about, that is the follow-up test I would like to do as a precaution, even though I think it is unlikely that it will be positive.

Jonathan: Okay.

Chris: Next was the urine organic acids profile. That looked pretty good overall. You just had a few markers that were in the high-normal range and a couple that were low. The two that were low were reflective of amino acid insufficiency, and that basically may be caused by not digesting protein properly. You already know that about yourself, which is why you are taking the HCl. Given that we know that you have SIBO, it's not surprising that you're not absorbing and digesting protein very well

because protein digestion happens in the stomach and the upper part of the small intestine. Nothing to be done particularly about those markers other than to address the SIBO.

You did the dried urine hormone test, and a few things came up there. The first is that you had high levels of free cortisol, but you had low levels of total cortisol. Free cortisol is only about 3 to 5 percent of the cortisol that is produced in the body, so it's not the best marker for overall cortisol production. That is metabolized cortisol. With you, you have high levels of free but low levels of total or metabolized cortisol. The most common cause of that is hypothyroidism.

Jonathan: Okay.

Chris: Or poor liver function. We have indications that you may have both things going on. This isn't really so much an issue with excess cortisol production in your case. It's a problem with cortisol clearance. Does that make sense? Your HPA axis is making probably the right amount of cortisol, but you're not able to metabolize it and excrete it through the urine as well as you should be, so you get a buildup of that free cortisol because of an issue with metabolism that is caused either by poor thyroid function or poor liver function, impaired detoxification. It's not so much an excess cortisol issue as it is a problem with you being able to get rid of it effectively.

Jonathan: Okay. So, do you think—you said that the levels of cortisol were low that were metabolized?

Chris: Yes.

Jonathan: But how would you expect that to make somebody feel, with having free cortisol?

Chris: High free cortisol can cause some of the symptoms that we would associate with HPA axis dysregulation. Cortisol plays several roles. First of all, it turns off the inflammatory response, which is good, but when it is persistently elevated, it can actually cause inflammation and interfere with inflammatory cytokine signaling, so it can cause problems with immune function. It can cause all of the symptoms of what we associate with a stress response, so anxiety. It can interfere with sleep, certainly, which we'll come back to in a second. It can disrupt blood sugar. High cortisol is associated with high blood sugar and other unfavorable metabolic changes. It is associated with a lot of chronic inflammatory conditions. Part of the reason I think that the problem is more with metabolism than it is with production is your 24-hour free cortisone is normal, and if anything, it is toward the low end of the normal range. That further indicates that the problem is more clearance of cortisol than it is overproduction.

Jonathan: Okay.