Introduction to Diagnostic Dilemmas in Tick-Borne Disease

This is Doctor Talks, real talk from real doctors on the issues that matter to you most. Hello, everyone. My name is Dr. Richard Horowitz, and I am co-host of this Doctor's Talk of the Healing Lime Summit.

And it is my great pleasure to introduce a good friend of mine who we've known for a long time, Dr. Christine Green. And we are going to be discussing today the specific topic of diagnostic dilemmas in tick-borne diseases, a really, really important topic.

Otherwise, the most commonly misdiagnosed disease is that mimic Lyme. So this is a really, really important topic that I'm happy to have Chris here. And just to give you a brief background, and Chris will explain afterwards a little bit more detail about her background, she's an expert in chronic vector-borne diseases, including persistent chronic Lyme.

And the vector borne illnesses that she diagnoses and treats are relatively new. And the scientific evidence, obviously, is still emerging. And I had hired and brought Chris on to the HHS Tick-Borne Disease Working Group when I was co-chair.

Chris worked with me when I was co-chair of the other tick-borne committees subcommittee when we were dealing with co-infections. So we've worked together intimately.

And Chris, it's a great pleasure to have you here. Please tell people a little bit about your background and how you got into this. Oh, thank you. Indeed, we've worked together a long time.

And I got into tick-borne disease when a patient walked into my office in 1989, who looked like this 30-year-old young man had MS. And it turns out, long story short, after a lot of neurologists, he didn't have MS.

And in the way for me of many things, he brought me a newspaper article and said, could I have this? And it described a patient very much like him. I looked up the author of the article that was quoted, who told me to treat him for Lyme disease.

And if he had a neurologic condition, nothing would happen. And if he had Lyme, he'd get well. And the young man got well. So after that, I began to see patients in my family practice who had Lyme disease.

And over time, the complexity of the disease became apparent to me. And I have spent time in the literature. I have spent time writing articles. I have been on various boards, including the ILADS board, which is the International Lyme and Associated Disease Society.

Right now, I'm an education director, a co-director for Invisible International, and we do continuing medical education for physicians and care providers in Lyme.

So that's how I got into it. Academically, it's a fascinating disease, but in terms of patient suffering, it's a terrible disease. And we have a lot of work to do to really identify it early, treat it early so that we don't get the chronic limes we're seeing and tick-borne disease.

We tend to say tick-borne disease now because there's more than lime in those dirty little ticks. Of course. So actually, the way you got into this was actually someone who was misdiagnosed with a disease, which in fact was an autoimmune disease MS.

That's actually one of the doorways you entered. So why don't we start off with that question, because that's really what we're talking about today. What do you see being the most commonly misdiagnosed diseases where Lyme is

underlying the pathological process?

Dr. Greenu2019s Path Into Lyme and Chronic Tick-Borne Illness

So it's interesting because I now see patients who have been sick for a long time. And one of the things I do is I list every diagnosis they walk in with.

And they're usually 10 to 15 diagnoses that they supposedly have. The most common is a chronic persistent debilitating disease, fibromyalgia, migraine headaches, chronic fatigue syndrome.

These are syndromes. These are things that have no cause. You simply look at the patient and say, I'm sorry you have this syndrome and we're going to do our best to make you comfortable, but we can't cure you.

which is a mistake because Lyme is a curable situation and tick-borne. The second most common misdiagnosis is under a mental health umbrella, depression, anxiety.

I have had patients, this drives me crazy because I have three children. I have had patients told that the reason they're anxious and sick is because they have three children.

just the patient is not feeling well and they look depressed. And if you don't do a proper diagnosis, you can accidentally assume they're depressed because they're sick and feeling bad.

The third category is autoimmune. Again, autoimmune disease in general doesn't have a cause. It is believed to be the immune system getting completely confused and attacking itself.

And so I have patients diagnosed with lupus, rheumatoid arthritis, or most often non-specific collagen vascular disease, i.e. they don't find a marker, but they think it must be autoimmune because it looks autoimmune.

Right. No, that's exactly right. And by the way, the patients come in and tell me exactly the same thing. It's almost always I've been diagnosed with chronic fatigue syndrome, myalgic encephalomyelitis.

I've been diagnosed with fibromyalgia. And with the psych, you know, it's it's, of course, much more even than anxiety, depression. It's obsessive-compulsive disorder, it's psychosis, schizophrenia.

Once we start getting into Lyman-Bart, you start seeing these really severe neuropsychiatric and the patients are coming in on loads of psych drugs, but they never got to the source or sources of why these people are ill.

So I agree with you. And even what's interesting, and I think this is an important point, Some of these people do have what I would call a true autoimmune illness, like I've had patients come in with rheumatoid arthritis where they had not only rheumatoid factors, and we'll get into this in detail, which are non-specific inflammatory markers, but they had CCP, right?

They were cyclic citrullated peptide positive. They actually had what we call true rheumatoid arthritis, but they weren't responding to methotrexate and they had migratory pain.

And the migratory pain is the hallmark of Lyme. So what it was is, yes, they had rheumatoid, But they got bit by a tick. So they had more than one thing wrong with them.

And I think sometimes doctors and patients forget you're allowed to have more than one thing wrong. But Lyme can be underlying a lot of these illnesses.

Exactly. And treating the underlying can relieve the symptoms of the autoimmune diagnosis. Yes. No, that's a great point. So tell me, when you're taking a patient history, how do you distinguish Lyme from some of these other diseases?

Like what role does duration and onset of symptoms play?

Common Misdiagnoses: Fibromyalgia, Psychiatric, and Autoimmune Labels

How do you make the differential? So one of my umbrella concerns in medicine is that The time with the physician has been cut to two minutes sometimes.

And I think of myself as a classically trained doc. I think the history makes the diagnosis. And it's not just Lyme, it's any disease. Lyme maybe is more important because there's become a huge dependency on laboratory values.

And the testing in Lyme is just plain lousy. but the history will give you the diagnosis. And so the timing's important and the development, the course of symptoms developing is important.

Most usually there's an onset for a patient. It's not mandatory for the diagnosis, but most usually the patient will say, I got sick in June of 2020. And you know, I thought I had a virus and it went away.

And then I got another virus and I thought it was something different. And then I got another one and I realized it's the same thing coming back. And what I would tell you And this is clinical impression.

This is not peer reviewed literature. I think the most common picture is that it keeps coming back and it gets closer and closer together and can end up never leaving so that you get this evolution of, gosh, I heard all over and I had a headache.

And I think I had a low fever. I felt like I had a fever. I had sweats. I felt really bad. I got over it. And then it came back. The other nature of Lyme.

and tick-borne disease is it is an infection. There is a real pathogen in the body and Lyme and Bartonella for that matter can go anywhere. They do not have a tissue tropism that's only for one place in the body, right?

They, Lyme especially, you watch that little spiral key, it can zoom around. It's one of the fastest bacteria known. And it can go neuro. It can go to cardiac.

It can go to the bladder. It, of course, can go to the soft tissue. It loves that connective tissue. Bartonella, similarly, can make a blood vessel in any tissue.

I've been told by Dr. Ed Breitschward, who's an expert in Bartonellosis, who really brought it, the modern Bartonella to our attention, that in a lab, he hasn't found a tissue that Bart won't invade.

So what you see in the course of this is recurring symptoms, but they begin to go elsewhere. You know, it started with a headache and a low fever and achiness.

And then, oh my gosh, you know, my ankle hurts so much. I couldn't walk on it for two days. And you get this course of migrating through the body. You'll start hearing autonomic symptoms.

This bug will go into the central nervous system. It will go into the brain, the spinal cord, hence my MS patient. It will go into the vagal nerve. into that autonomic nervous system, and it will trigger problems along the way, mass cells, et cetera.

So you see this recurrent set of symptoms that begin to have other symptoms connected with them, which is why often the patient is identified as a malinger or someone who worries too much about their health or something like that, because how could this be?

And the way it can be, and we can talk more about this later, is the nature of these pathogens, they are stealth pathogens. They have the ability to get into the body and evade immune detection so we don't wipe them out.

And while they're evading immune detection, they go, that place over there looks really juicy to me. I'm going to go set up something over there. Right, so the point you're bringing with these clinical presentations is like Lyman-Bart, it goes everywhere in the body.

So for central nervous system, headaches, light sensitivity, sound sensitivity, dizziness, memory concentration problems, sleep disorders, neuropsych, depression, right?

Because it gets into the central nervous system. The autonomic that you were describing, that's dizziness, standing, palpitations, fatigue, brain fog, anxiety, which we're seeing with long COVID, right?

The lymph nodes, you'll sometimes see large lymph nodes with BART, sometimes with Lyme, gets into the heart, can be second or third degree heart block, right?

The pulmonary is more the babesia, but even the gut, right?

How Lyme Presents: History, Recurrence, and Migratory Symptoms

We're seeing gastroparesis with the autonomic nervous system, chronic constipation, muscles, joints, and the migratory aspect again, which you and I just mentioned, is kind of a hallmark symptom that I think helps the patients and the doctors to diagnose it because there's only really seven diseases in medicine when we When we published our screening lime questionnaire back, I think it was 2017 with the researchers from New Paltz, we found there was only seven diseases in medicine, lupus being one of them, by the way, that temporarily caused migratory pain.

We're going to get into this later. I joke with doctors, unless you're at the bottom of your medical school class, you should be able to differentiate gonococcal arthritis, and ulcerative colitis Crohn's, and acute rheumatic fever, and Reiter's syndrome with these sausage digits, and acute hepatitis.

You should be able to differentiate these things normally from Lyme. I mean, there's a very different clinical presentation. So the migratory is really common.

As you said, it's multi-systemic. good and bad days comes and goes. And then there's a point, as you said, it all comes together, right? Where people start getting sick day by day and it just, it doesn't go away at that point.

nature, it has been called the second great imitator after syphilis. And what does that mean? It goes anywhere it wants to in the brain. The brain is how we perceive, right?

So I used to say, and I sometimes still do that, my Lyme patients are the best worked up patients in the world because they all have this pain, they all have this pain in their liver.

and I'm looking at the liver and I'm looking at the spleen, and they have a radiculopathy. They basically have an infection in that little radicular root, which Lyme loves to go to, and it's giving them their symptoms.

And that is extremely common. I think I should have said radiculopathy is one of the major missed ones. No, and for people who are not physicians listening, we're talking nerve pain, right?

We're talking neuropathy, a specific type of nerve pain. And you're right, I mean, it's burning, tingling, numbness, it has a very specific clinical characteristic.

So when someone comes in with neuropathy, right, you're right, it can look like liver pain or something else. But once you ask the patient the clinical characteristics and it's neuropathy, then you've got to do a differential to say, all right, what's causing neuropathy?

Is it lime? Is it barred? Is it heavy metals? Is it mold? We've got to go through the pieces, carpal tunnel, pregnancy, hypothyroidism. You have to go through the differential of what causes neuropathy.

Well, I tell patients this is like shingles. Shingles infects that little root. And just like there's a distribution to shingles, there's a distribution to that radiculopathy.

And there is neuropathy and radiculopathy. So that's right. Risk factors that increase the likelihood someone's going to get it. What do you think might be a reason why somebody might get a more severe case of it?

Do you think there's people who put out more carbon dioxide heat, they attract the ticks? Do you think it's hormonal in some, immune dysfunction? Do you have a general sense of the major risk factors why some people get a lot sicker?

So I think one of the areas in medicine that's very exciting right now is what's called precision medicine, where we're having the ability to look at your genetics versus my genetics.

And one of my favorite papers that actually changed how I behaved in Lyme is written by an evolutionary biologist, Paul Ewald, who talked about in this case, chlamydia pneumoniae and the APO4 gene and smoking, and talked about how bugs evolve for different genetic systems.

They're successful and different. And so there is some thought, it's not just me, and I would say it's not my original thought, that people who are hypermobile and have Ehlers-Danlos or hypermobility somehow don't deal as well.

with this spirochete as other people. If there is certain autoimmune tendencies, HLA-B27, if there are some family histories and those people end up with Lyme or Bartonella, and I think it's, oops, I think it's different for different bugs, for different infections or pathogens, Yes, I have a feeling I can get Lyme and get over it because I've had an EMF and I took my dutiful month of antibiotics.

But you got it early, which is part of the reason. I got it early. As did I, by the way. I got it almost 30 years ago, got it early and been fine. And of course, that's the problem.

It's the chronic states that are the problem for people. Right. And the missed people, And perhaps if I had been missed, you know, there would be a problem because that's part of it too.

But I suspect that there are people who are in more trouble and I think we need to keep trying to identify who those are. Great. Just going to have one quick question before we take a quick break.

These co-infections, you've been discussing them. Just tell us briefly, like if somebody has Babesia, what are the most classical symptoms you're seeing?

How are you differentiating BART from Lyme when you're doing it? There are hallmark symptoms you're seeing that are really standing out. Yes, there are.

And I hasten to say that this is really hard to document in the literature. There's very little in the peer-reviewed literature that says, here's the pure Babesia patients, and here's the symptoms.

Again, Dr. Breitschwert, Dr. Bozziani have done good jobs in Bartonella giving case reports. So we have some information from that. In a Babesia patient, Babesia is a red cell parasite.

It is in the same group as malaria. And just like malaria, you get sweats and chills with it. It's not the rigors, usually, of malaria. It's the exceptional patient who gets that lull of it.

But it's drenching night sweats. or the coldest people I've ever met have babesia. I mean, I remember when I began this, I'd have someone say, you know, we pile on three quilts and an electric blanket and she's still cold.

So temperature intolerance, babesia also has a reputation of causing depression or anxiety. And I think that when I see Herx's, and we can talk about that afterwards, I think that's probably true, that for some people that's in the area of Babesia.

No, absolutely. I mean, it definitely makes the patients worse. So we're just going to take a quick break at this point. I just want to thank everyone for joining us today.

If you found this conversation helpful and insightful, great. If you're a summit purchaser, stay right here. We're going to come back and we're going to dive into details regarding our conversation on the differential diagnosis of Lyme.

If you're not a subscriber at this point, please click on the button below or on the side to get access to this important information, because we have some of the Lyme experts in the world with us during this Healing Lyme Summit.

And I think you're going to get a lot out of it to help your health and hopefully the health of your loved ones. If you are watching this, again, thank you for being a valuable member of this community.

OK, so let's just take a pause. So Chris, if we dive back in here, you were talking about Babesia and things making worse. And I agree with you. I don't even see the classical babesia of the red blood cells bursting apart, this hemolytic anemia.

Babesia Symptoms and Air Hunger

I've only seen it twice in all the years. It seems like when the bugs come together, when Lyme Babesia Bar comes together, the babesia doesn't act like it does in the textbooks where you read about the red blood cells bursting apart and the kidney failure and the problems with the liver.

It's only really the immunosuppressed patients with congestive heart failure in the hospital who seems like they're getting this. linectomy. Yeah, but it's true.

It's confusing for docs because you read it in a textbook and it's like, why am I not seeing hemolytic anemia with the red cells bursting? And for some reason, the way the immune system's reacting, we don't see it in the same way.

So it's a good point. It definitely makes everything worse. And I also see a lot of that air hunger, that shortness of breath and cough, we can't explain apart from the larial of the day sweats, night sweats, chills, and flushing.

I would say that's a, it's a pretty classic presentation, even though I've never seen an ARDS, an acute respiratory distress syndrome, either from COVID or by the way, or from Babesia malaria, even though it's reported, I've never seen it in our patients.

I don't know if you. And I think the air hunger is interesting to me because in little children, you see them in the course of the visit, they will be taking this big breath.

And it is. It's not shortness of breath as we sometimes see in an asthmatic, et cetera. The patient will say, I just don't feel like I can get breath.

Right, and the differential is you got to make sure, of course, they don't have emphysema, they don't have asthma, right? They're not bronchoconstricted.

They don't have a bad post-nasal drip, right? I mean, it's always differential diagnosis, right? It's always the same thing with it. Yeah, and many of my bad Babesia patients do end up seeing the pulmonologist because, and again, especially early on, I wondered if somehow they acquired asthma, they acquired bronchospasm from Babesia.

And interestingly, I worked with a pulmonologist who started to see many of my patients. And he said, Chris, the problem is their diaphragm is weak. They get a weakness in their diaphragm.

He had this special test. And that was often what happened with the Babesia air hunger patient is they couldn't bring down that diaphragm to really expand the lungs.

And as treated, they'd get better. as they were treated. And the vagus nerve, of course, does get affected, right? You were saying it before with the Ellis-Danlos patients.

You were saying the ones that have that predisposition. The ones who get dysautonomia, right, with the autonomic nervous system effect with POTS, they have the roughest time, right, with the vagal dysfunction.

And certainly the diaphragm can get affected. So, yeah, it's a good point. Let's go on to Bart for a second. We were discussing Bart. What are the hallmark symptoms you like in Bart when you're making the differential diagnosis?

So again, there's always this trouble of how many pathogens are active in a given chronically ill tick-borne disease patient. My colleague, Betty Maloney, whenever we were having a discussion and I say, oh, everyone's got at least two or three of these, she said, no, no, Lyme alone can cause this, and here's the data, and it can.

But I think most often when you have a chronically ill patient, they have several things. So I talked for Ilad that I would use the word the flavor of, the flavor of Bartonella walking into your office.

Because again, we can't write these things in stone. So what the I think Probably going to end up patho-pneumonic signs of BART is TRACS or STRIA, whatever we're going to name them.

And it's one of the few, yes, it's a diagnostic in this tick-borne disease. And just for the audience who, again, are not doctors, you're talking these either horizontal or vertical stretch marks that almost look like you lost weight or gained it, right?

They could be white, they could be purple, but they're just like the EM rash, you know, the classic rash of Lyme is the zero theme of my brand's bullseye, although it's half the time not a bullseye, but the classic rash for Bartonella, right, are these type of stretch marks, right, that we call them, right?

And they often don't go in the line of the stretch marks, which is one thing, but they sometimes do. They start out red, they'll fade to white in most people.

And you often see them in young men, in teenage boys, and they suddenly blossom straight horizontally across the back. So what does a Bartonella patient look like as opposed to the Lyme or the Babesia?

Bartonella Hallmarks and Severe Neuropsychiatric Disease

But psychiatrically, I would tell you when I see someone who says to me, Dr. Green, I get so irritated. This isn't me. I mean, my four-year-old will do something four-year-olds do and I'm so irritated about it.

Or the person who says, I get rage. I shouldn't be raging about this thing. I do then start thinking, I'm going to look hard for Bartonella at that point.

There is a very nice paper, I think Mosigini and Breitschert were both on it, which is a rheumatology practice. in Maryland, I believe, where they looked at Bartonella patients and they looked at the subgroup that had been diagnosed at some point with Lyme.

And that combination, Bart and Lyme, had worse joint problems. They had more persistent, painful joints. I'm sorry, I should move my computer cable. So I do think that if I see someone who has particularly refractory joint problems, in spite of being treated for what I think is an underlying Lyme, I will start thinking of Bartonella too.

Right, so to sum up, so basically what you're saying is, and I agree with you 100%, it's the most severe patients that come to see you with the most severe neuropsychiatric, the most severe joint pain, the most severe neuropathy, right?

In the paper we just published in Microorganisms about three months ago, we also found it was the ones with the most severe autonomic neuropathy, like the most severe resistant pots, the neuropathy, the bottom of the feet,

right, Joe?

this years ago, and I have to admit I wasn't sure it was true early on, and yet it is, right? It really is something you hear from these patients. I don't see the large lymph nodes as much, and I occasionally will see granulomas on the extensor surfaces with Bart.

I've seen it in a couple of patients, but it is really the severity. The eye problems they describe I haven't maybe seen you know retinal occlusions and arterial I haven't seen those as much or even thank God so much optic neuritis but I would say it is the severity of the neuropsych and the pain and the neuropathy and in our study that we just published also Immune deficiency, the ones who had the most Bartonella species, if you had Vinsoni and Elizabethae and Hensley and Quintana, Baxilliformis, multiple species with Babesia, those were the ones actually who had the worst immune deficiency, low IgG levels, low subclass.

So now I'm looking for like, hey, you have immune deficiency, gee, I wonder if Bart. I used to think it was just Lyme. I just learned this even a few months ago when I data mined for that recent paper.

Well, and definitely these pathogens we're talking about are stealth pathogens. They have the ability to both manipulate and suppress certain parts of the immune response.

So I do think you can get an acquired immune deficiency. Very hard to, and again, if you aren't a doctor out there, very hard to get that treated, to get your IVIG for that because it's mostly for primary immune deficiency.

But yeah, I think you do see it. Now question also, I'm seeing a lot of mycotoxin illness lately in our practice with mold toxicity. Are you seeing it?

Are you seeing that in your patients? How much are you looking for it? Do you find the symptoms are overlapping or getting worse or that it's interfering with your success?

Can you speak about that a little bit? I can, I wanna say one more thing about BART. BART is known to cause pericarditis, myocarditis, just like Lyme is.

So when I have cardiac symptoms and I will always, always screen for BART as well as for Lyme. So mycotoxin illness. So I long before I was introduced to Lyme, I worked with patients who had a colonization, who had infection of various tissues with mold, with aspergillus, penicillium, and I developed a great deal of respect for mold as a problem.

The toxic mold, i.e. so those little guys are living in us and they can make toxins, or they're living in our environment and we're acquiring those toxins, is is clearly a problem for many people.

Can I distinguish the patient? It's a trick question, by the way, because it's almost impossible, I think. I agree. The one thing I thought about, when someone tells me they feel poisoned, they feel a kind of illness that is just, they're just sick, I do start thinking of toxins, including mold toxin.

I start thinking that's not your classic description of your tick-borne disease patient. So that's the one thing. Obviously, there's evidence that mold toxin can mess with the immune response, which could perhaps augment or support the infection.

I have a colleague that I work with, with Invisible, Charlotte Mao, who is a pediatric infectious disease doc. And she said, you know, I'm beginning to think that the contribution of mold is that it hurts the immune response or it poisons the immune response and allows that tick-borne disease more free rein.

Oh, no, there's no doubt. I mean, the gliotoxins specifically are affecting immunity. And I think the reason this is so important for people who are attending this Healing Lyme Summit is that if you've not been tested from old and you have found that, hey, you've done some of the classic treatments for Lyme, for Bartonella babesia, and you're not better, you should absolutely, absolutely check it.

Because I had a talk with Neil Nathan a little bit earlier about this. I was not a full believer. I believe people have been exposed. But in my world, usually when we cleared the infections, they got better.

But lately, there have been some very resistant patients that would get better. But they weren't improving the same way. They denied mold exposure. We tested them.

They were loaded with it. We pulled it out. And all of a sudden, that was the missing link. They said, oh my god, I'm like, I feel almost 100% better.

So it's tricky. The only thing I would say that maybe is a clue to it is the chemically sensitive ones that walk into a room and they can smell the mold and they get, that's because most people don't do that.

Those ones that are mold toxic, more than not, I hear that from them, like, I can't live in this apartment. I walk in, the husband smells nothing, right?

The kids smell nothing. They think the wife is great. And yet they're very sensitive to it. That's something I would say maybe we see more of. Yeah, I agree.

And I was going to say, I have a patient who, fits your description. He got temporal lobe epilepsy as his presentation of Lyme when he was at Boy Scout Camp.

Mold Toxicity and Overlapping Chronic Illness

And he is quite mold sensitive. And he discovered that he couldn't go into the refrigeration area in a grocery store. He kept saying to his mom, I can't be here.

Something's wrong. Something's wrong. And it turns out The malt specialist said that's where the malt is in the grocery store, is in that refrigeration section.

So I was going to say that one of the ways you might be able to distinguish is if the patient's telling you there are certain locations that make them sick.

That should not happen with pure tick-borne disease, right? Good. Let's get back a little bit to what you brought up earlier because we haven't gone into detail on the rheumatological diseases.

If somebody's coming in with joint pain or they're coming in, let's say you thought it might be lupus or not or MS or not, how are you doing the differential for those as opposed to Lyme?

Again, remembering people are allowed to have more than one thing wrong with them. What do you like as far as diagnostic markers or clinical markers? I do the classical autoimmune testing.

I'll look for lupus markers, rheumatoid factor, CCP, I just do that whole list. In terms of clinical exam, you mentioned, and I think this is very important, and this you presented with your validated questionnaire.

I remember this. I think it was 2015, by the way. You said there are three things, migratory arthralgia, migratory myalgia, and migratory neuropathy. You get all three of those.

If you carefully read your paper, you've probably got it slammed up for lime, right? It's true. You're right. And it's an unusual one. You won't see that with lupus generally.

You won't see it with rheumatoid arthritis because it's bilateral symmetric. Exactly. And, of course, as we said earlier, the problem with Lyme, though, and it's the same thing with long COVID, these people are getting huge amounts of autoimmune markers.

I mean, anti-thyroid antibodies, anti-myelin antibodies, anti-glangulicide, ANAs, anti-nuclear antibodies, rheumatoid

factors. So the problem is, if you don't recognize that Lyme causes this, you might think, Oh, it's a nonspecific autoimmune illness, right?

But the CCP has to be positive with rheumatoid. Volupis, I like the double-stranded DNA in Smith antigen, right? I think that's a good way to differentiate that.

The DNA, I would say, has been demoted to an inflammatory marker. And then you have to go from the ANA to tell that. Andrea Gaito has given a couple of presentations.

She's a rheumatologist who's treated Lyme for a long time. And a couple pearls I've picked up from her, the knuckles, these DP joints are more likely to be reactive in Lyme, whereas, of course, your MP joints are more likely to be rheumatoid arthritis.

The late Lyme patients, they're all red. I call it striping. You get a stripe here, you get a stripe there. They can be 23 years old, and that's what their hands look like.

The other thing Gaito said, and occasionally I find this, she said in rheumatoid arthritis, the lower patellar ligament is sore. In Lyme, it's the upper.

And occasionally, it's very specific that I find that. So the rheumatologists who are trying to distinguish this, she did one other, it wasn't a study.

She presented three patients at ILADS early on who had rheumatoid arthritis and had Lyme. Unequivocally had both things. And she treated just for rheumatoid in a group, just for Lyme.

But it's when she put the two together that they got better. And I found the same thing that I found that the, for example, the rheumatoid patient I had that did not respond to ARAVA, Enbrel, steroids, methotrexate, failed everything, Rituximab.

It was only, and he had migratory pain. After we treated the Lyme, interestingly enough, and we gave him methotrexate, the methotrexate worked. So it was like you had to clear the underlying infection for the autoimmune issue, right, to finally get better.

And I mean, from the beginning in Lyme, comments have been made that the amount of inflammation for the amount of spirochete found is out of line. So Lyme is going to inflame more the underlying inflammatory condition and you remove one layer and then they can respond to their anti-inflammatories.

Differentiating Lyme From Rheumatologic and MS-Like Disease

No, good. Same thing. Let's go on for a second to MS for that patient you initially saw with MS. It's very tricky because you get demyelinating lesions in the central nervous system.

You'd get myelin basic protein, oligoclonal bands in the spinal fluid. What do you like about, I mean, apart from the migratory aspect, which we talk about, anything with MS for you that is more specific that helps you with the diagnosis?

It's the big picture. It's that you aren't just getting the demyelinating symptoms. You are getting, and you said, except for this, but you are getting the migratory arthralgia.

They have dysautonomia. It's that pattern recognition that you're looking for. They have more than what you expect from the MS patient. With the dysautonomia, you often see in Lyme a stiffness, stiff man syndrome, which has been promoted to stiff person syndrome, at least by Stanford.

And I think While with MS you do get some of that rigidity, it's very again localized to where those demyelinating lesions are, when it's a more general picture.

Now that said, if I had a positive Lyme, and the patient looked exactly like they fit MS only, I would still treat the Lyme. As you say, they can have more than one thing and sometimes to your surprise, I mean, I've had rheumatoid factor positive, CCP positive people treated for Lyme and those things disappear.

Right, and in this case, they discovered in the last couple of years that there are Epstein-Barr virus variants that are now associated with MS, apart from the older ones like low vitamin D and chlamydia pneumonia, right?

I mean, Steve Phillips, when he used to lecture, would talk a lot about the fact that it was in the Faroe Islands, right, that they never had MS, and then the British invaded, and all of a sudden there was MS, meaning There's an infectious pathogen here that seems to be right.

So over the years, we've discovered it. The one thing that I think, and I'm curious if you've seen this also, the one thing I learned that I'm curious if you agree with this, when you do the MRIs of the cervical, lumbar, and thoracic spine, the one thing I don't see with MS, with MS, I usually will see that there is demyelination in the cervical and thoracic with lime when i've looked for it.

I've seen the white spots in the brain and i've seen it where there can be a lot of them and you can't just differentiate from the corpus callosum outward how many are there.

But they don't generally, with Lyme, they don't generally demyelinate the upper part of the spine. And if I see demyelination in the cervical or thoracic, it says to me, that may be an MS picture.

I'll look at the amount of myelin-basic protein and the amount of oligoclonal bands. But since you get it in both Lyme and an MS, it can be a little tricky.

Do you find that that's the same with the MRIs? Have you seen that? I will watch, but I have not observed that. But now that my eyes are open, I will look and see.

Great. Good. So what else do you think as far as differential diagnosis? We were talking earlier about chronic fatigue, myalgia, myalchid cephalomyelitis, fibromyalgia.

Now, they can be caused by viruses. Years ago in California, they were talking about HHV6, EBV. I mean, so, and interesting, I think, Now, with COVID being out and the flu, we're starting to see reactivation of other infections.

In fact, my wife recently had COVID for the second time, got over it, got the flu, gave her some Tamiflu, got over it, and then reactivated some other stuff underneath and was complaining of trigger points and stuff.

And I was thinking, gee, it was all the foods and the mast cell and trigger points. And I realized after she went on some fancyclic for viruses, this thing she's been complaining about and these trigger points went away.

And I realized, in some of these Lyme patients who've been complaining of chronic fatigue fibro, they do have underlying viral infections. Sometimes they reactivate and it's not one thing, right?

And it's an important point to keep in mind that this is a soup of viruses and bacteria with Lyme and BART and fungus and mold, right? And parasites. It's usually never one thing.

It's a combination of all of these things. So make sure you look at Zofluza as opposed to Tamiflu, which works on the RNA and appears to have some activity against COVID.

Yes, so before I treated Lyme, I practice in what I call the shadow of Stanford. And I learned from a very wonderful doctor, Jose Montoya, how to recognize reactivated virus in the chronic fatigue patient and to treat it.

So I think that both virus, and tick-borne pathogens can mess with immune response enough that you get reactivation of things that we normally carry around our immune system very carefully controls.

I mean, we are a soup or a stew, however you want to think about it. So I have always looked for reactivated virus in my chronically ill infected patients, whether the infection began with a known tick bite.

If they've been sick for more than a year, then I will start looking for the reactivation. I'll tell you, interestingly, since COVID, I have many, many more positive PCR to Epstein-Barr, to herpes 1, to CMV.

Herpes virus 6, I've also been seeing reactivate some of these patients. And those patients won't get well till you treat the virus. I mean, I tell them, I say, look, we don't even know if a virus is alive.

This is a philosophical debate, right? It's just this little thing that keeps making more of itself. And then your poor immune system is seeing more of it and reacting more.

So yes, I think when you have, if you're the patient and you're chronically ill, And, or if you're the doctor and you're trying to figure it out and you think this is an infected patient, definitely look at all those things we've all had.

Americans have mono by the age of 40, 75% of us by the age of 20. Almost all of us have HHV6 by the age of three. CMV, I find more likely to be in people who have been in medical facilities.

It's a little less common than your EBV. herpes may be inherited through the mother's DNA. I mean, herpes 1 is present in almost everyone by six months.

So yeah, I think reactivation of underlying pathogens, including chlamydia and pneumoniae, including mycoplasma, which might be a tick-borne pathogen, but we certainly, 40% of the world has it, and it's not all from ticks.

We get it by coughing in grade school. So yeah, the co-infection is not just co-tick-borne infection, it's other active. Right. And I think you brought up a good point.

A lot of these patients come with very high Epstein-Barr virus titers or HHV6, whatever. But it is the PCR, right? It's the pieces of DNA in the blood where they give you the logarithmic amount that says, oh, it's reactivated.

And that's kind of how we know when to use the antivirals. And I must admit, there is definitely a subset of patients in it.

Viral Reactivation and Complex Co-Infections

You're right. I've seen the same since COVID. Definitely more viral, maybe I'm looking for it more, but definitely more viral reactivations. So if someone's not getting better from your standard Lyme Babesia bar treatment, you got to look for mold.

You got to look for the viruses, do the PCRs for it. You got to check this just to make sure because the problem with chronic fatigue and fibro is that Fatigue, joint pain, muscle pain, sleep disorders, brain fog, dysautonomia are all the same symptoms.

And it could be caused by a virus, and it could cause by Lyme, and it could be caused by Bartonella. So it's really amount of teasing it out, right, as we're talking about in doing a differential diagnosis.

Yes. And I think the immune system has a limited way to respond. If picture, right, your IL-6, your TNF alpha, you're going mandatory intracellular pathogens.

By the way, Chris, you froze just briefly. So if you can just go back a sense, just slow it down a little bit. You know, everyone's having problems, by the way, even with line connections.

Just go back and just repeat what you just said a little bit about the IL-6 and how people are getting it. So the immune system has a limited number of ways it can respond to a problem.

If the response, if the infection or the problem is inside the cell, that will be a certain response versus in the blood or in tissue. If it's inside the cell, Whether it's a virus infecting or Bartonella infecting, you're going to get the same cytokine pattern like TNF-alpha.

There's different ones for specific organisms. Lyme will have TNF-alpha and IL-6, but it will also have IL-10 sometimes because it's manipulating our response.

But so yes, it's very hard to distinguish what thing is causing the symptom. As you say, it's brain fog, it's arthralgia. I want it clear though, I don't require a PCR positive to treat virus.

Early in the chronic fatigue story, when it was thought that it might be Epstein-Barr causing it, It was noted that if early antigen and nuclear antigen are very high at the same time, antibodies are very high at the same time, that that's a dysregulation.

Even if the virus reactivates, you get early first and it goes down and the nuclear comes up. Now that's been mostly considered not a good diagnostic tool.

Personally, I've used it from the beginning. I think it's a good diagnostic tool. Well, you learn by me, you learn from one of the experts Montoya was one of the guys who was first talking about HHV six and chronic fatiguing illnesses.

So you really did learn from one of the experts early on. And so if I think, if the IgG's to viruses are very high, I don't think it's just a past infection.

I think it's a reactivation. And as you treat, you will see those numbers come down. And I have, it's probably only a handful over the many years. I have a group of patients who every time they stop their antiviral, they would start getting sick again.

And finally, when they came to baseline levels, right, to that level that most of the people have, they could stop. Right. And I've seen exactly the same thing, by the way.

There are some people that just have to stay on Chronic Fempscyclovir or whatever to be able to stay well. Otherwise, they just keep reactivating. I agree.

I've seen the same. So we're almost out of time. So just a quick question. Let people know, how can they contact you? Do you have any upcoming seminars, anything you want to tell people about before we finish up?

Closing Remarks and Contact Information

How can they contact me? Best to contact my office. I practice in Palo Alto, California, where we have tick-borne disease all year long. It is not a summer flu out here.

And probably best is to go to Christine, C-H-R-I-S-T-I-N-E, greenmd.com and put in inquiry in there. I do not currently have any coming seminars. I'm probably going to be at the AAFP presenting information for doctors at a booth in September.

And is Invisible International doing anything new apart from the doctor training and those programs, anything new coming out from Invisible International?

Oh, good question, and I'm a terrible advertiser. So Invisible is mostly generating our CMEs. We also have not webinars, we have competitive solutions.

We kind of put out a problem and we get teams to compete. And we do have, we are funding a few teams to generate their solutions to different problems in life.

Great, good. Well, Chris, this was great. I want to thank everyone for attending this particular session of the very important differential diagnoses for how you differentiate Lyme from other diseases.

It's my great pleasure to be with you, Chris. Chris, you've been a good friend for a long time. We've worked together being in ILADS, and I'm really glad you could take the time today to make it with us.

So I just want to thank you again and wish everyone well, and hopefully you'll be tuning in for another one of the Doctors Talk on our Healing Lyme Summit, and we hope to see you soon.

Thank you so much. Thank you. And thank you for doing this. It's really important that we get this information out so people treat early. If you think you're blind, go see someone.

Thank you for tuning into Dr. Talks. We hope today's episode has enlightened and inspired you on your path to optimal health. Each day is a new opportunity to make choices that empower your well-being.

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