Inside Bartonella: What Every Lyme Patient Needs To Know Transcript

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Introductions and Bartonella overview

Hello everyone. My name is Doctor Richard Horowitz and I'm the co-host of the Doctor Talk Healing Lyme Summit 2.0. It is my great pleasure today to introduce to you Doctor Robert Moses.

Bob has been in clinical practice for quite a while, doing Bartonella, just as I have. And today's talk is going to be on Bartonella signs, symptoms, lab testing, treatment.

Bob, thank you so much for joining us today. It's great to have you here. Thank you I appreciate it. Yeah. So yeah, I, just, give you, some background, on myself and a bit of personal history.

So I grew up in the Hudson Valley just south of you. Oh, I didn't I didn't know that. Town of Newburgh. Got it. And, went to a very large public high school there, which had a very strong academics and AP programs and such.

But it was a microcosm of of everything that a city could have. And, after that, I went to the six year bachelor's MD program with Union College and Albany Med and, so, you know, with that background, I, I always had an interest in research and investigative medicine. So, after, medical school, well, before medical school ended, and during medical school, I was doing lots of research and transplantation immunology.

Richard Horowitz's background and research path

So I'm basically an immunologist at heart. And and so, from med school I went and did a research residency. Well, let me backtrack before graduating. That's what I did a year and a half at the NIH, at this part of the, first Howard Hughes Research Scholars Program.

And from that I went to a research residency at Yale and, just coincidentally, the happen to have discovered Lyme disease there. And at that time, there was nothing controversial about it.

But the questions always came up, you know, how do you know the treatments? And I've had, you know, the symptoms or, you know, the usual questions we now have and, and we're beginning to settle, some of those questions.

But in the research residency, I continued my immunology interest and which I had expanded on when I was at NIH. I was in doing some theoretical modeling of, peptide antigens, to be able to selectively modulate the immune system.

And then, and, you know, in the research residency, I did a postdoc as part of that in molecular biophysics, again, trying to look at the peptide MHC interactions, of the immune system.

So some I characterized that as a biophysical, chemistry for immunology. And then, I chose the specialty rheumatology because I thought that would be the closest, fit with my interest in autoimmune diseases.

And so I really went deeply down the pathway of, autoimmune disease, only to kind of realize that almost all of them are driven by undiagnosed persisting infections.

Which we are, which we all come upon after a while. Right? But with many of these, right, with many of these infections. So you kind of you kind of back end into it as a rheumatologist, kind of finding that infections were behind a lot of the joint pain and rheumatology issues.

You were saying. Exactly. Yeah. So it's fascinating about that is that rheumatology cut its teeth on the concept of chronic infection, like causing rheumatoid arthritis.

And the original, drugs used to treat RNA were actually anti invectives at the time. Things like heavy metals, like arsenic, like gold. And then, even more recently, if you look closely at the effects of different drugs that are used as first line drugs like methotrexate, that's, folic acid inhibitor, much like bactrim.

If you look at, sulfur San Jose, that's a combination of an antibiotic sulfur drug with aspirin, you know, and plaque. You know, we in fact, rheumatologists will say, oh, well, let's try an anti-malarial drug when it's not malaria, you know, so there's a long history of this, in medicine.

And some had been used as an anti-inflammatory by rheumatologist as well. So it's really fascinating to look at where it began. But then over the years, you know, more of the research went toward immunology and much of the research money and, the came from, or for the study of the immune mechanisms.

So the emphasis started shifting a bit. And, the biologicals, when they started rolling out, they provided a great deal of, funding to academic centers to develop these.

And I think that's been, skewing some of the view as well. At the time, I was a rheumatology fellow at Yale, and I did a second rheumatology fellowship at NIH later, at that time, the number two clinic at Yale ran, but ran the Lyme disease clinic.

And as rheumatology fellows, we actually carried a line pager when we were on rotation. So it's one of the few places where rheumatologists actually do get some experience treating Lyme disease.

And in most of other, most other facilities, it's, treated by infectious disease departments. Of course, I think that's helpful to, to keep in mind. And this goes but this goes back how many years ago that you were doing this right now?

Oh, my. I finished my residency there, in 1991. Right. Okay. And, and I did then I went from that. I went to I had two options. I, I applied for and I got a, a, physician scientist training award, to stay at Yale.

And I had an offer to take a senior staff fellowship position at the NIH, and I chose to do the latter, eating for a wide variety of reasons. So, now and within rheumatology, I was more interested in immune mechanisms.

And then I found that physiologically, the vascular system was the most affected and it was the most ignored of the systems. Everyone focuses on the arthritis, but patients had lots of cognitive issues.

And many of these are have some some component of most of these autoimmune diseases is also a vasculitis. And so I became very interested in vasculitis and then and super vascular disease.

Discovering Bartonella and early cohort findings

So I took a big detour from practice for a few years, developing, ultrasound methods, Doppler ultrasound to look at cerebrovascular disease. And, and ironically, then I started finding that infections.

I was looking for infections that could cause vascular diseases. And, that's when I learned about Bartonella and perfusion. And so I reentered the world of Lyme disease from totally different directions of, of cerebrovascular disease. Wow.

Very interesting. Yeah. Because I was actually curious how you got into Bartonella as much as as much as you did over the years. Now at knowing understand how you kind of kind of backed into it right over time.

So the way it started was also quite interesting because I had a patient who was a psychologist, and she had, I don't know if this or no was the cause, but she, had a diffuse, the usual chest x ray pattern, which actually resolved as we treated her for now.

And, and I don't know if, if that's the explanation, but anyway, she had done some research and she said, well, the

guy you really should speak to is that brighter?

And, so, and then I also heard I got this from another, contact of mine, a veterinarian locally, who was doing lots of work in Lyme disease. And part.

And so she, also mentioned ed things. So I called Ed one day and, we still joke about it that we had a two hour long, first phone call talking about, and I, I love the way he started.

He said the more I study this micro, the less I understand that. And I said, okay, this is the kind of guy I want to work with. So, yeah, he he's been absolutely amazing researcher with Ricardo, Maggie and the rest.

I mean, what a what a team he's got there. I mean, I've been doing this. So then I started sending him specimens for his research study, and, you know, and, we had some patients who made donations to support his research program.

And then after a while, I figured I just keep sending him specimens until he said, stop. So we, sent a total of, almost 300, specimens, over a few years and beginning in 2007, which led to the publication in 2012 describing that cohort of patients who we considered I considered as those who had the diagnosis of Lyme but weren't necessarily getting better, but I characterized them as having small vessel disease because if you're studying history professor of medicine, you will understand what how small vessel disease is manifested from a central nervous system point of view.

So for example, executive dysfunction, difficulty, you know, processing information, the word finding all the things that we all know very well that we see in our patients. So, we, we published a paper, paper in 2012, in the CDC on journal Emerging Infectious disease.

And the big lesson there was that, well, first of all, the title of the paper was that it was an alignment demic region. And, the details, are, in fact, that these patients had, tested positive or clinically felt to be positive for Lyme, mostly through serology.

But through the testing that NC state was doing, we found that, first of all, no single test gave you the full answer in most patients. So there was some overlap where a few patients were positive on PCR and serology, but for the most part, they were not.

And so we learned the lesson that you really have to look at the serology and the molecular and you can't trust the negative result. Right. And that and that's primarily because and I know from Ed's research and everyone else is that the number of Bartonella species that we keep finding every year just keeps going up and up and up.

And I think years ago, it might have been 13 that it was 15, that it was 17. I mean, he's over 20 at this point as far as pathogenic species. So, so so question the the most common ones that you're finding and of course you're, you're running T labs at this point.

I know you're doing the fish test, which we've had great results with using your, you know, your T, your t labs, bartonella fish. That is picking up Hensleigh Quinton of the axilla form is, Vin.

So and I subspecies Elizabeth. It's picking up a broad range of species. Correct. Yeah. So so you kind of hit the nail on the head here in terms of our laboratory challenge.

So we designed the the RNA fish test to be very sensitive. But it's specific because we we have a nucleic acid probe. So then the question is, you know, if it's a way more sensitive than PCR, you know, what do we use to actually verify that the target we think we are seeing is actually the target?

Or to to answer your question, what is the sequence? What are the sequences that we are detecting with this probe? Right. So that's that's a diagnostic challenge because PCR we before I started running our lab, I thought a PCR would be have a sensitivity of maybe 10%.

And and by the way, if think Galaxy and NC state are doing their their way ahead in terms of improving the sensitivity. But there's some fundamental issues with PCR that, that make it very hard to use it as a sensitive test.

These are the inhibitors in the serum that you're referring to specifically, or. Those are big ones. And that's what, techniques like, digital PCR or droplet digital PCR are trying to overcome.

But there are some other molecular biology issues as well. And there are some extraction issues as well. So like there we think the answers are there, but they just have to all come together.

And I think that's going to happen pretty soon. I think PCR sensitivity soon will be, much improved. And so but we need that because when you have a test that's that beats others on sensitivity.

How do you how do you how do you verify that. And how do we know what sequences we're seeing? We want to know the sequences because if nothing else, we can improve the probe design.

Right. But we can also know which strains of Bartonella were the PCR. Brilliant. We're able to find. So I think that's important. On the other hand, when as a clinician, we don't know enough about how to treat these, to have different treatments for different species or different strains and different strains within the species can also have different, responses to antibiotics.

So I think that, we're going to need another leap of technology to, to be able to, to know what my what antibiotic to use tool for which microbe. So clinically, for example, I think, you know, knowing that there is a strain apart on is good enough.

Testing challenges and T Labs diagnostics

But different strains may make different species more than others. Like there are some species specificity in terms of the pathogen. The city. Right. So at this point in your chronic Lyme population.

And when I was I want to talk about, by the way the, the symptoms that you're also seeing because the overlap obviously is very strong with Lyme disease.

Right. I mean the main things that stand out, I mean, it's a chronic, fatiguing musculoskeletal neuropsychiatric cardio. It causes the same symptoms, but a lot of times we'll talk about the pain in the bottom of the feet that Joe Boris Garneau had discussed years ago, which I do find, you know, is true.

And in the paper we published last year, we found that the neuropathy was much stronger, especially in those that didn't even just have one bite species, but multiple species with multiple baths, their neuropathy, their autonomic neuropathy was worse.

Much worse spots, their neuropsychiatric symptoms was worse. And their immune deficiency, we found these were the ones that, you know, had chronic variable immune deficiency on either subcu immunoglobulins or IVIg.

So it was clear the Bartonella was causing very similar brain fog, neuropsychiatric, but it was almost like the neuropsychiatric was ramped up in the Bart patients.

Much worse, with the ones with schizophrenia and really severe. You're you're finding pretty much the same type of symptom kind of overlaps. But worse with Bart.

Yes. Yeah, pretty much so I think as a general rule, in the same way we think about contractions, I think you can become infected with multiple species.

Restraints of movement and a lot more of these or any of these, and, and I think that we know that, not only in the 2012 paper, but we regularly see that, serology is negative in maybe 40% of people, usually adults who've had it for a long time.

They have no antibodies. Spagnola but they develop it about 3 to 4 months into treatment. So it's very helpful to monitor serial ifa's, to, to be able to show that immune, reconstitution.

And that then also overlaps with heard some reactions because now you have an immune system that's been restored, that's able to see and respond to various organisms.

But I think the immune suppression of the Bartonella causes also, I think, makes it much harder for them to deal with other infections. You know, for example, and really any, any other infection because it is immune suppressive.

Now, in kids, however, the serology is usually positive just because I think kids aren't they haven't if they have guano, they haven't lived with it for that long to suppress their immune system, except when they acquired it in utero.

Right. And you know, and the problem with immune deficiency and I you know, for those listening, right now we're speaking with Bob Moses. He's been an expert in Bartonella for many years and has developed, some excellent lab testing through T labs for checking for Bartonella and also strains of ABC.

We'll talk about, in a second. But what we're discussing here is the fact that Bartonella is overlapping a lot of Lyme, and we're finding it in our cases now.

We're finding it at least 80 to 90% when we go looking hard enough for it. The interesting part about what you said about the serology, and I found exactly the same thing.

One of the papers, the initial papers we published in 2016 and an autoimmune case with Besets, this patient was sero negative. And it was kind of a strange case. The rheumatology.

Right, which you would know, this should have probably put her on an episode a long time before, but was using all of the, you know, TNF alpha to intercept steroids, methotrexate because she had a rheumatoid overlap with besets, but she was never on DAP.

So we put her on doxy, rifampin DAP. So and part of the protocol I was developing in 2016. And when we went into the intracellular compartment and I can't remember why, I decided to add present amide.

But when I was looking up the TB and persistent drugs and, you know, Bartonella of course, was discovered by Hopkins around the same period of time in other universities to be a biofilm, persistent bacteria, which we didn't know, like Lyme.

I added peers into my thinking, hey, it works in TB. It shortens the course. Let's see what happens. And this woman's besets autoimmune lesions cleared within two months.

And her part, which was sero negative, turned positive after going into the intracellular compartment and kind of pissing off the bugs. We also found tularemia titers that went from 1 to 20 to 1 to 320.

She was like hiding tularemia inside the intracellular compartment. I sent her, in fact ID to confirm this, and her Hvy six titers went up fourfold to 1 to 1280.

So it was like she had all these viruses and infections hiding in the intracellular compartment, which you and I see, I think, in clinical practice all the time.

But it was fascinating to see how you go in there with just one other drug, because it only happened after the Xenia mite was added. That's now you know why I use it, although I need a much larger trial because I really have no idea how well it truly works for Bart.

But I do use it. But fascinating that you're right. I mean, zero negative patients turned zero positive, and sometimes it requires kind of going into the intracellular compartments to kind of flush it out.

Yeah, absolutely. I think, you know, a history of our treating these conditions. You know, there's initially the focus was so, much more on Borrelia that we were using, a lot of selfless forms like IVs, reaction, etc..

But these don't penetrate cells very well. And I think people may have been getting better because we were chasing the microbes into the intracellular space with these treatments.

So I guess because because reception doesn't go into the intracellular space, of course. Yeah. So, you know, I think, so one of the big treatment shifts has been to try to rely more on the, the drugs that do go into the uterus, hyper space and positive light is excellent for that.

So so these and then also do combination therapy to hit the microbes from different mechanisms. But it's really remarkable how many other, issues occur like these viremia virus infections and so forth in a patient is already predisposed.

And, you know, speaking of the viruses, the place where this gets really complicated now is that because Bartonella causes immune deficiency and Lyme causes in efficiency, now we're finding more toxins, like in a large percent of our patients with Clio toxins that causes immune deficiency.

And then they get long-covid where they get T-cell exhaustion on top of the B-cells being affected. And it's like, I don't think people realize they're walking around immune deficient, which is why they're having such a tough time with all of these infections that are getting in.

Right. And I think if, you know, when all the research is eventually done to look at, long Covid, we're going to find that a lot of those patients may have have had these, some of these infections that were not previously diagnosed.

And that and that's showing up. I spoke to it. In fact, Bruce and I will be doing an interview. Bruce Patterson will be doing one soon. And I'm pro and I'm looking in the literature, I, I agree with you.

It's they're finding Borelli and Bartonella showing up in some of these long Covid patients. So through his radiance diagnostic profile when we do is 14 cytokines the the VG, GF is showing up on almost all of my patients.

And we don't know with the vast groups. And you talked about vasculitis earlier and Bartonella causes it. We don't know if the vasculitis is from Covid the spike proteins, is it from Bart, is it from Covid and Bart.

Other factors. So it's it's kind of complicated. And I sent that to Bruce and he said, yeah, we kind of got a it's going to take some time to pull this apart to figure out what's doing what.

You know, I think the good news is we have, we're able to even notice these things. Right? So it's it's a lot to try to put together. And we we have all these techniques and insights staged, but we just have to come up with a more comprehensive plan and just so many aspects to consider.

So on the point about, co-infections, so what I saw in our original cohort and it's data that I presented, but I would never, publish this additional data.

It's from that 2012 cohort that we published in Emerging Infectious Disease. The, bringing all the patients, had a different signature on the western line for Borrelia. They had a lot of it.

So it was more of an IBM positive IgG negative pattern. So it just speaks to, the fact that the exam is maybe, falsely positive in a chronic insulin patient.

Symptoms, serology, and co-infections

And it doesn't mean that they don't have really it. But every time we treated patients who have that, the exam resolves. And the I never see were converted.

So my my understanding we by the way we published the same thing back in I think it was 2019, in the International Journal of General Medicine and Health Care.

We found the same thing and Hopkins found the exact same figures. It was 45% of our patients were IgG, CDC positive Western blot or immuno blot. My understanding, though, from Nicole Baumgarten, what we were seeing is that the B-cells, when Borrelia was affecting the B cells, it was kind of knocking out a subset where it wasn't making IgG and then kind of shifting over to an exam pattern.

So I'm not sure the role of whether Bartonella is in fact causing that with Lyme. I mean, I don't know. I mean, do we know that for sure? I'm not sure we have the data yet, do we?

I agree, we don't have the data. I certainly do that, that the germinal centers were affected and there was never an isotype switch. Yeah. So, yeah. But then the more interesting thing lately is, a few months back or last year, I was visiting, head and, the team in Galaxy.

And, so the question came up and asked me if we're seeing what our prevalence rates were in terms of Barn owl on PBS year. And, I noted that we were seeing higher prevalence rates in our specimens for those than we were seeing for Borrelia.

And he said that they were seeing exactly the same thing. So what we have now realized over the past year or so is that are known to be ever seen together almost always.

And we don't know why, but they're they're commonly contracted. And while the interesting part that came from my side of the woods, where, you know, from the Institute of, Ecosystem Studies with regard failed, he had published got it had to be 8 or 9 years ago that in the ticks when they're transmitting Borrelia, though, we are seeing bbca being transmitted a large percentage of the time.

And by the way, speaking of that question, since we're bringing up a Busia, the other call, I, which you've got a, you know, a test for and we are finding it from your lab.

Are you finding at this point when you're looking at your fish testing for quality or for these other BBC species, 50, 50 or the colleagues are starting to kind of pick up more than the other species, like, what are you seeing at this point?

So the history this is really, quite interesting. So we, I have a colleague who was very concerned that a patient had the Baeza and he started, calling and inquiring, and he, he spoke with, East Stroudsburg University and, they said that they're seeing relatively zero recoil and, 40% of the tests to be passed.

So with that in mind, I thought, okay, well sooner or later we're going to be seeing this in people like you can't be in the ecosystem and not show up.

So we developed a probe based on that idea. And I do think these probes, you know, sometimes we see spots that are very bright and sometimes we see spots that are clearly there, but they're not as bright.

So I feel like maybe if it's a slight there's a slight sequence variation. We're getting, like less probe binding. So it is. So that might be a clue that we're seeing some variability.

But we, we started testing with that and found, it quite surprising. We, we actually got some specimens from, John Scott that tested positive and, we.

John Scott from you talked with Canada. Yeah, yeah. And then, and then after he published. So we were working with that probe, in the research phase before that paper was even published.

And then when it was published, and then subsequently the PCR study did was published, and began to get more attention. And we had developed the, the visa or the quality probe sequence based on the paper that had top

published in the recently at the Islands meeting, they presented, you know, Ricardo presented on the visa and and so, I do think the evidence is building that it's there.

And again, if it's in the ecosystem, it shouldn't be surprising, you know, that we would find it. Now, to answer your question, we we, we thought, okay, well, if this probe is picking or recoiling in some variants, what is it missing?

So we then set out and developed a genus probe. And when we started looking at specimens that were negative for to correlate, but where the blood smear showed inter RBC inclusions that were suspicious for the BCA, we then tested those using the the visa genus probe.

And we found that there was another that of those specimens. They were about 55, 60% positive. So where you. Said that that might have been micro or some other, the species that.

Exactly. Now, I do think that the Order quality probe picks up microbial because we actually did some sequencing studies with, Drexel where they, where they did find, microbial.

And when you're doing sequencing, that's you introduce a whole nother layer of, of questions because like PCR is sensitivity can be low. And a lot has to do with how the specimen is prepped and even the molecular sequences, some of them just don't read.

Well, when you're doing, sequencing. So but we do know that it can pick up, my Crotty. And then the other thing that was confusing is that the serology that we would see usually was a Duncan serology that would associate with the probe positivity.

Or really, you were getting positive Duncan eyes because, as you know, I published something like two years ago and I said, listen, we don't know if it's cross reacting.

Yeah. You know, because some of these patients in the study I published came from the West Coast. So it wasn't clear, but I was finding, at least at the time, done county positives up in Maine.

But of course, you know, who knows what the cross-reactivity was because the CDC had only found 13 positive cases. But I think that all the doctors were looking I know.

So again, it gets back to the fact that, you know, we're trying to use serology and maybe we're stretching it sometimes, like you, I don't think you can necessarily go in to find the strain or the species with with serology very reliably, at least on this case.

Right? I mean, at least from our perspective as clinicians. I mean, it still told me, okay, they've got day sweats, night sweats, you know, fevers, flushing, air hunger can catch my breath.

Unexplained cough classic. The busiest symptoms where all the underlying Lyme symptoms got worse. So at least we knew to treat right at this point. And as Tom had discussed during the islets conference, and I'm confirming it also, I mean, the defense protocol does seem to be helpful.

In a lot of these patients that have the I wouldn't say it's 100% effective, but I'm definitely finding it to be more effective than some of the older protocols with Mapmyrun, as if through Max or clinic.

Why not? Right. Yeah, I agree with that. And in fact I've kind of given up on the older protocols. But I do believe that, you know, now that we have more sensitive testing and we can actually see what's going on.

I think we're really at the beginning of, of trying to optimize protocols. I think we just have to, you know, if you can see the microbes directly then you're not misled by an immune response. And and I think that, you know, I've been seeing patients clear there, these BCI using it's not so I often start with premier clinics.

But I check their Cyp2D6 to make sure they convert it to the active form, otherwise to panic mode, but to panic

when usually is the end point for me of the treatment.

And then, based on some of the protocols being used in research at Yale and Tufts, maybe adding and so the on to to one if needed. But I aim to get three consecutive negative, tests. When I'm treating.

Yeah. We, we had a patient who was fish positive times five and he did the. Yeah. I mean you couldn't believe it. And then finally actually he ended up clearing the infection after two.

But it's true. We're using it with Mapmyrun. The the problem of course, with the insurance is I'm not sure if Mallon is as they when we you know at the islets conference it looks like defend Quinn alone may be helpful, but we just don't have large enough studies to know.

Is it best with a topical and does it work with a tobacco and Pragano Mallon do you have to answer it right?

Treatment strategies and antibiotic protocols

I mean, there's still a lot of unanswered questions, but at least the patients clinically do seem like they're getting better. Yeah, I couldn't agree more.

I just think, we just needed to have the visibility of the, of the microbes, to know what we're actually. What are what our endpoints are. So I know the way I talk to patients is to say, hey, you've reached your clinical endpoint, but what we don't know yet is if you've reached the laboratory endpoint, right.

And we think we're not sure yet, but we think that it's necessary to also reach the lab endpoint to minimize the risk of relapse. So, you know, we're at the frontier of this.

Yeah. So so by the way, in your Bartonella patients that you're seeing because we are finding the rashes and quite a bit of these patients. Right. Sometimes they're, you know, purplish and perpendicular skin plates, but a lot of times whitish. Right.

They change color over time. I know that Ed had published a study in other people about the neuropsychiatric effects of Bart, and I couldn't agree more, as well as even cancer overlaps with melanoma.

And, so, I mean, I am inflammatory breast cancer. I think there's some issues that we need to flesh out in the cancer research to you finding those those three, those classic stretch marks in a lot of your patients.

Also. We do find them a lot. And I I've actually been kind of maintaining a healthy skepticism, but I keep convincing. I keep getting more data that convinces me that those are almost always, Bartonella. Yeah.

Every time we, Because we have, just like we do, a blood, spearfish, we do the same, probe on, skin biopsies. And every time I've looked at one of those lesions by a skin biopsy, it's been positive.

And as a control, we get, a biopsy from normal appearing. We scan right next to the lesion, maybe a centimeter away. And the density of burning, in the lesion is always considerably higher.

But it's also present in the, nearby skin biopsy. Interesting. Which I, by the way, I had the same skepticism you did early on because I tried finding it.

This is many years ago thinking, come on, it can't. And then over time, of course, I convinced and what finally convinced me is the ones that were perpendicular to the skin planes.

Like. Exactly. That's right. Right. Because if you're losing weight and gaining weight, you can't tell. But if it's perpendicular skin planes, that makes no sense whatsoever.

They tend to I think they tend to follow, discipline. They have it horizontally in their back, which I think is from a

growth spurt. Usually in young men, young male teenagers.

But I think it's hints of image follow the lymphatic lines like, especially when you see it around the waist. So I maybe that's the explanation. Waist, buttocks and inner thighs are probably the most common right that I've seen.

I've seen back. I've seen also across the breasts in certain women too. Right. That yeah, very classic. So yeah, we, we know that from the skin biopsies.

And so we're also beginning to look at nerve demyelination. We have an assay for that shows demyelination using confocal microscopy. We're doing a study with, Katz on that. And, and so we think it shows, demyelination before, EMG nerve conduction studies, and I think some patients for some reason have a higher, they have more neuropathy in their symptom complex that they present with.

Yeah. As I said, the worst neuropathy patients in my practice, not even a doubt. They're always overlapping. Lyman Bart. And of course, the problem is regarding neuropathy, the mold.

Getting back to the all with moldy long-covid and even heavy metals mercury, lead, arsenic. Right. They all cause neuropathy. So we're finding, apart from the usual causes, you know, diabetes and carpal tunnel and hypothyroidism, the rest we've got a lot of overlaps causing neuropathy in these patients.

And I you know, I think when they did the NIH trials years ago, we're designing a thanks to you, by the way, for those people who are listening who don't know this, Bob helped to get me in touch with the consulting company that I'm now using for Garland Consulting that we met this morning.

Bob, I have all my applications in at this point. To get the first funding we're looking for, you know, a funding grant starting January 15th. We have to put it in.

But but, yeah, we're moving ahead. And so I want to thank you for getting me in touch with them. And, our statistician, Nicole Close looks like she's going to be working with us.

And she knows Bayesian statistics, by the way. So she said to me, I gave her all your stuff on, you know, the statistics that you love. And I said, she said, we're going to look at the end points how we have to do it.

But but ultimately, the toughest part of designing this trial is trying to figure out what happens on combination therapy to just prove it for chronic Lyme, since almost all of these patients have Bart and Bbca overlap.

So the only thing I can think of is I hate to be fish negative with negative symptoms, that are suggestive. Like I don't have pain in the bottom of my feet.

I don't know how. Well. So I'm going to avoid this doing the trial because you want to get a clear trial for chronic Lyme. And I think they had the same problem when they did the double blind, multicenter placebo trials years ago with the NIH.

You know, God knows how many people had co-infections and overlapping factors on the M6 map causing inflammation or downstream effects that just weren't checked.

Yeah, that's that's, a new biology. The reality of the biology is that these are complex infections. As you have, published and stated over and over again.

Yeah. And so I think, maybe we need to redefine what we look at and maybe you treat a broad cohort and just look to see if at least the brilliance is extinguished, with the treatment that, reverse the problem.

And, and that's what I'm, you know, fortunately, because Monica Embers from Tulane published a study last year that rifampin and dabs on killed Borrelia in the mouse model.

At least I have a culture model, a mouse model, an animal model, and then, you know, retrospective studies when we when we apply for the grants or. Yeah, that's great.

So, so regarding the treatment protocols, I know everyone's doing different things for Biden. You know, I've published on it with like nine weeks of oral dapt shown in combination, with these two week pulses.

Are you finding I don't even know how much you're even using this myself. What are you using at this point in your practice to be able to get these patients better?

The history of this is that I started, I interviewed, veterinarians to see what they were using. And I considered the different options. And so it kind of came down to some macrolide and, going alone.

So as a starting point, but, I, the conclusions I stay away from for long term, treatment because of, not just the known tendinopathy risks, but also because they're known to shorten telomeres on chromosomes.

So, I've been using maybe. That's one I had not heard before. Yeah, I'm glad I'm hardly using them at all anymore. But thank you for sharing that. Yes.

Who knows. You combine it with something that increases, but, the, I kind of looked at as if for mice and, and the veteran experience was that there was a little more resistance with that.

And, like the risk for mice and, because it seemed to do better, but also because it has an interesting property that can help our patients, which is it's an inhibitor of liver C1234, you know, a similar to the effect of like grapefruit juice.

And what's interesting about that is it actually indirectly boosts, the levels of cortisol. So certainly it's chronically ill patients are adrenal insufficient.

And so without having to give them hydrocortisone, you kind of, you know, give the mice and can give them some adrenal support indirectly and almost as a side benefit.

Yeah. But by the way, we find literally almost 100% of all of our patients have adrenal dysfunction. I know no person I check at this point that has normal adrenal function on a DHEA, cortisol a salivary test when we look.

Yeah, I think if you've been chronically ill, like you can't expect them to be normal ever. And then the tests are often misleading for adrenal because the in a sick patient level should be high higher than normal.

But so coming back normal in a situation is low. And it's just it's often misinterpreted. But Clarissa was one, part of it. And then, I wanted an antibiotic that would penetrates and also rifampin was the initial choice. And later, and, and we're sort of camping, you're inducing several liver sided rooms, and with that, you're now lowering cortisol, which probably overcomes the inhibitory effect of the class of mice.

And so now you have a patient with low cortisol that goes lower while they're having a high number reaction in which they need higher than normal levels of cortisol.

So you're literally creating adrenal crisis. At the same time you're causing hurts. And and so this therefore, in a patient who's been chronically ill, I find it very hard to use Roehampton without a little bit of physiological doses of hydrocortisone.

Yeah. You know, the way I've gotten around it, by the way, is glandular. I have found that some of the glandular is out there, as I mentioned, makes one, called adrenal live.

It's from places in the world where there's no prion disease. And I've used it. They've checked very carefully. And I found that actually, if I give people enough of some of these glandular, I can actually avoid hydrocortisone and and not and not use it.

But I do use, adaptogenic herbs, Rhodiola, ginseng, you know, B vitamins. I'm, I'm using that pretty much. I'm using cortisol support literally in almost every patient when we're doing these protocols.

I think that's it's vital to do that. Yeah. So, so but I wanted to talk a bit about Rasputin. So religion, penetrates, tissue, about 50 times more than rifampin.

So there's a pharmacological turn, volume of distribution and 0.9l for rifampin, and it's 49l for, retribution. So one of the reasons I think we see such a stronger response to it is because it penetrates tissue.

But I also think that's the reason why there's a significant risk of uveitis as a first time reaction. And so one of my, smart, ophthalmology, ideological friends, called me up one day when I referred him this uveitis patient.

And so and he summarized it perfectly. So it's a Slimer in the eye. And I said, right. And we know that Bartonella, like Lyme, causes every itis every inflammatory part of the eye, right? That that's affected.

The recipient just happens to penetrate that tissue. But I've never seen it with rifampin. So now, before I personally approve of you and I have, a consultation, referral letter that I send.

I have the patient take my letter to the ophthalmologist to make sure they're fully plugged in, because it's a very treatable and manageable issue, but it has to.

They have to be plugged in to the ophthalmologist ahead of time, because they can't have any delay in getting seen and started on on Duracell and other treatments for uveitis.

And so you. Do have you added methylene blue to your protocols with rebuke nuclear retro? Have you have you tried mixing them to see what it does based on the Hopkins data?

No. Not regularly. No. Okay. I mean, I'm having good success with it. The problem I'm facing, and I don't really know the the variability of what I'm seeing, I'll just say in general, if people do a nine week oral DAP, some combination and finish with a six day high dose pulse and dump of quad zone and take two months off, and then every roughly every two months I treat with two weeks of antibiotics with six days of high dose tap zone.

But it's with rifampin, methylene blue, doxy, Jethro Max. And in some cases, I mean I'm using 5 or 6 drug combos with a lot of liver support, probiotics, you know, microbiome support, etc.

on the average I would say four pulses, which takes almost a year. It takes down the load sufficiently that many patients will go in remission for a long period.

But where I'm finding they need more pulses in this protocol, and at least for me, it's keeping down the number of days I have to leave my antibiotics.

Herbal support, pulses, and clinical endpoints

Is this with mold and the ones with long Covid? It it's taking a lot longer for these kind of patients. Their immune system suppressed. Right. Because of the T cells. B and the rest.

Those are the ones I'm finding because Bartonella already suppresses that. I'm finding that needing more pulses. But, you might eventually we can talk about this privately, you know, after this call.

But you might want to try looking at the methylene blue in combo, because I used it based on the Hopkins studies and culture and I did find that adding it to the protocols did seem like it did make a difference.

It's a great way to stain biofilms, by the way, so we know that at least gets into biofilms. Yeah. In one patient we even looked at his biofilms without staining the slide.

And sure enough, they were blue. So anyway, so that's, so I would say Rafi being, the way to give you honestly, Tribune was maybe 10 or 15% before the pandemic, and I now believe it's 20 to 30%. Wow.

So I, I'm more reluctant to use it. But we also had published that some teenagers with neuro and children with neuropsychiatric diseases seem to respond a lot better and faster when switched from rifampin to recipient.

Okay. You know, one of the questions that brings up, of course, is the way they do TB therapy, like the direct observe, therapy with Dottie is to use pulse therapy.

And instead of and I've done this for patients because as you refer, butin has a much longer half life than rifampin. So if they're on hormones therapy like thyroid, everything gets thrown off.

And you have to adjust the other hormones, not just cortisol. I found that if I pulse the rifampin once a week, by doubling the dose the way they did in TB, I do fine for some people.

Of course they get her cases and they get larger cases. But I do that for people on multiple, like if they're on thyroid meds and, you know, cortisol in the rest to keep down the effects based on what you were talking about, about the distribution in the body.

It may be that that's, you know, an alternative in some of these people that might be at risk for it, that may be using a higher pulse dose of rifampin might get thrown.

In, maybe. So on the other end of the spectrum, I had a patient who's a young guy who's going to college. He was relatively noncompliant. I had the trust of his family in him, and they just didn't want to give up on him.

So I just, I asked him if he would just simply just take a few things. I made it as easy as I could. I and I asked him to take, Allison, Garlic, garlic, garlic for those who are listening.

Right. And liposomal oregano and, curfew plaques from Zion and turmeric and so, and also vitamin D. So, he, he promised me he would just take those daily and he did.

And this was, January of, a couple of years ago. And, my next conversation with him was in October, and he told me that over, the course of that year, by springtime, he was feeling much better.

By summer he felt normal, first time normal. And, in years. And then we tested for Bartonella by fish. And it was negative, in October, you know. And that's fascinating because I used to use a lot of Allison years ago from Doctor Zang for traditional Chinese medicine with, like, Allison Copp to say, circulation.

I used to protocols the the problem, of course, is the smell that comes out of people's bodies using that. If he's in college, I'm surprised he didn't get, kind of poked a little bit by his friends because the that you can always smell the Allison coming out of your skin, but the other people around you can, but but it's interesting.

So for you, it was Allison. Curcumin, liposomal oregano oil and artemisia. Those were. That was the combo using, Nora to measure just, and with Allison, oregano and, turmeric.

And turmeric. Now, that's just one person, right? So we don't know if that's generalizable, but I kind of feel like the herbs have, significant antimicrobial role.

And again, now that we can actually look at the blood and see if it's, if it's working, I feel like again, we're at ground zero in terms of coming up with, you know, better protocols.

All right. That mix. That's why I hope to prove with this randomized double blind study. And the question will be when I discuss it with the statistician, whether we do multiple pulses after the initial round to actually account for Bartonella, that I'd like to do it.

I'm still waiting actually, to meet with Nicole, but I think in the next few days we're going to have a discussion actually on that exact topic. But very nice.

Yeah. So I think, you know, I think we're beginning to turn this whole business into more of a science, you know, now that we can actually see the microbes and see what we're doing, to them, I, I like you.

I noticed, how many, how the cascade of symptoms of infection creating inflammation and different system dysfunctions really requires that we use a functional medicine strategy to recover people.

Yeah, it's absolutely essential. And, and, and a couple other interesting things, you know, not really. Expecting this at all. But reflecting back on the last 15 years, I don't I haven't had one patient in my practice with a heart attack or stroke.

So that's kind of interesting, right. Because the the research model of cardiovascular disease. In other words, lowering vascular information from yes, we're going back to where we started with this conversation.

Exactly. You know, it's interesting you say that because the only person I can think of that has had it was an 80 year old with diabetes, with hyperlipidemia and of course, obesity and all the risk factors with insulin resistance.

But you're right. Come to think of it now, again, I don't have the same internal medicine population that I had when I started off years ago. But yeah, you're bringing up an interesting point because you're right.

I mean, I haven't had a patient with a heart attack and a stroke or otherwise, apart from this one patient. I'm thinking of you, right? I haven't had one literally in a long time, but I thought it was mostly because my population of patients had changed over time from a standard internal medicine practice.

But maybe it is. Maybe it is working on vascular. It's fascinating. But we know, you know, and that's Roma is a virus like in there have been plenty of papers finding different organisms in there, including each minority.

They found that in private parts, you know, 20, 30 years ago. Yeah. So I think, you know, I just I think it's exciting to, to just, appreciate everything that we can now look, see and test for and learn.

Because it's it's going to be a big game changer, you know, sooner or later. And, you know, and since you brought that up and it says. But just because you discussed functional medicine, you know that they're now finding microplastics and all of these, all of these clots and arteries, which is driving inflammation, bringing all these thousands of toxins in.

And it's a question they're finding much higher heart attack and stroke rates in the people with high, right. When they've got this high volume of plastics that's showing up in here.

And then the question becomes, based on our conversation, how much is Bartonella interfering or interacting, in fact, right, with these microplastics increasing the inflammatory response, in fact causing some of us.

Yeah. How does that change the biofilm.

Future research, microplastics, and closing remarks

We sometimes when we look at these blood smears, it's not part of our, protocol to, Yeah, we have strict, guidelines on what we report as a biofilm, but we sometimes see these, like, we Irish kind of synthetic looking, you know, they don't take up, dying very well. And, you know, it's been a bit of a puzzle, but maybe those are microplastics.

I just don't have a way to prove it. Yeah, yeah. Well, Bob, this was great. So quick question, because we're kind of getting to I knew this was I knew this was going to go close to an hour.

There was no question in my mind, how can people get in touch with you if they want to learn more about what you're doing, get in touch. What's the best email for them to get in touch with you?

Well, for the for the laboratory, it's info@md.com or the website. And on the medical practice side, it's, it's the, the website is HTML, md.com and, you know, we, we still take a limited number of new patients.

But I'm spending much more time on the lab side. So. Yep. Got it. Yeah. No, we're all at a point. That's why we're trying to train new doctors right through islands and the islands.

Because at this stage, you've been in clinical practice, as I have for, you know, quite a while, and you can only take so many. But fortunately, the science is advancing nicely.

And I think we both can agree that, you know, we can certainly give our patients a lot more hope than we could a decade ago based on where the science is progressing for lime and Bartonella and but on all these co-infections.

Yeah, it's changed dramatically. Again, just having visibility on to the problem. It's finally turning this more into, science. And and algorithm driven, based on objective test results.

And we'll continue to do that. My, my future is going to be to look for ways of proving causation, like looking to see if the immune system is, in fact, presenting fragments of peptides from these microbes to the immune system.

And then we can start getting into selective modulation of the immune response. And it's exactly where I started my my research at NIH. And almost like some people are doing with peptide therapy or shot therapy, trying to at this point modulate it that way.

Because the only molecules that we have to type in order to do a successful transplant. And I remember that's when I started my career, I was in the transplant immunology lab at Albany, and, my first mentor there, is now, doing this, and has developed some gene therapies for, rheumatoid arthritis.

So I think the there are the techniques, have finally matured enough where we can, potentially look at the peptides that the immune system is responding to and see if those peptides happen to be from, microbes.

Yeah. Good. Bob, thank you so much for taking the time today. This was great. I know people are going to get a lot listening to this conversation. So again, for those of you who have tuned in my name is Doctor Richard Horowitz.

I'm the co-host of the Healing Lyme Summit 2.0. We've been discussing Bartonella and also a bit of babka with doctor Bob mosey, Jani Bob, thank you again.

And by the way, for those of you again who don't know about T labs, please check it out. We're finding it to be a very effective laboratory also for finding tickborne infections and proving them in our patients.

So, Bob, again, I want to thank you for your time and effort today. I appreciate that. Thank you so much. Okay. Thank you. Thank you.