Introduction and Guest Background

Everyone. Again, thank you for coming. Being part of the multiple sclerosis and neuro immune Summit. I am here with my very good friend, Doctor Ken Cherlin.

Now, Doctor Sean and I have known each other for many years. Really, a decade now. And, Ken is a board certified neurologist who's also trained in, functional medicine.

Integrative medicine. He he, practices functional medicine. Does research, is a cutting edge neurologist. And he is where I send my complex neurological patients.

And they really get superb care, with Ken and his team. So, Ken, I am so pleased that you here. Is there a thing that I left out in your intro that you'd like to stress?

No. Thank you so much. Appreciate it. Okay, so let's get right to this. You know, I know that people are, very, very excited about, some of the new DMT that are coming out there.

And one of them is the BTK inhibitors. I wonder if you could comment, briefly about, about that drug type and whether or not it's worth all of the excitement that is happening in the conventional world?

Yes. Absolutely. And I want to sort of broaden that if I can, and put it in context because, I think, you know, we will really help a lot with the discussion and where things may go over the next 40 or so minutes.

You know, I don't know if in this summit, your 3.0 summit, you're interviewing the folks from, the Octave Bioscience Science group, but I know that was in the 2.0.

And that particular test, is very important. I think even in looking at these BTK inhibitors. You know, when we're looking at, we're talking, first of all, broadly about what's called disease modifying therapy.

And you know, in the in the management of the disease mass, we broadly divide, the approach, the clinical approach into symptoms management. Maybe that's pain or spasticity or, you know, bladder function issues and things like that, and then drugs or treatments.

Many of the new treatments or biologic agents, they're not just the standard sort of chemical drugs, but, they are really aimed at attempting to and particular, slow what we call sustained disability progression.

There, you know, as you know, and you're a researcher, you do clinical trials, you have to define a primary outcome for a clinical trial. And, historically, if we go back to the very first drug that was introduced in the United States in about 1993, that was beta serine.

BTK Inhibitors and MS Treatment Landscape

And at the time, the study really just looked at relapse rates as the main outcome. But as time went on, it was realized that, well, we should be looking at relapse rates for people at least who are relapsing remitting Ms..

That there are other measures of primary outcome that may be, more important that we really have to put first in terms of determining if a treatment is effective.

And so there's been a shift, toward what's called sustained disable progression. And that's based on a gold standard in, in, in sort of a measuring stick of how a person is doing with the Ms.

called the ADSs or extended, Persky Disability Status scale. And it's largely a glorified physical examination in which different aspects of the exam are given points.

It's heavily weighted toward gait or gait dependance, meaning that. Are you walking independently? Can you walk 500m, unassisted? Or do you use a cane?

Do you use a walker or are you in a wheelchair? If you're in a wheelchair? Can you still transfer things like that? So those sorts of questions weigh heavily in the EDS score.

All right. So if we look at broadly all of the different agents and there's some like 25 or 26 agents currently there in several different broad categories, which we can certainly talk about.

But if we use this test, the Ms. disease activity tests from Active Bioscience, which really puts a microscope on mass at the cellular level. What's very different from just eyeballing an MRI and saying, how's that person doing?

There's a lot that can be missed there. What we see is that the people who are on what I call B-cell depletion therapy. So that's, sometimes it's rituximab, but that's not technically approved for multiple sclerosis in the United States.

But it's okra realism. Mab or oak reverse, also tumor Mab, which is key symptom. And then the newest sort of reiteration of over to mad a rather a ocrelizumab called bri. The.

So when we look at the M. S. disease activity score, I find and I have a lot of patients with Ms. in my practice and they all get this blood test when they come in.

And by the way, can't just go to your local quest station or lab LabCorp or talk to your primary care doctor and get this test. This is an exclusive relationship between the clinician, like myself and Octave Bioscience, to be able to perform this test.

So what we find is that the folks who are on those B-cell, depletion therapies, have by far, the lowest scores these drugs are in red. And we should clarify that low scores are what you want. Yes.

You don't want a high score. So the octave you want to have a low score. So, continue again. Yeah. So it is, it is, measured from 1 to 10. So essentially I don't think there's a zero but one to, you know, or is low 4.5 to 7 is moderate.

And then, 7.5 to 10 is high. And what's wonderful about this test is not only does it give you kind of a, a snapshot of your current level of disease activity from an immune perspective, how active is that immune system in attacking my linen nerve cells in the brain from a sort of a myelin breakdown perspective, and then also from a what they call near axonal integrity, which is the degenerative component of them that's independent from the destruction of myelin.

And so the people on these drugs have very, very, very low scores. And I think the folks who own the patent on, key symptoms are offered to Mab, which is, Novartis Corporation.

They can project out, expect, you know, relapse rates over a long period of time and that this drug can suppress relapse rates as long as 20 years. So, you know, these are very powerful treatments, but they do have some downsides in that we're talking about, attacking or really destroying, what are called B cells.

These are the immune cells that make antibodies. But these are the cells that come from your bone marrow. They circulate around in your bloodstream. They're they're among other things to protect you against viruses and bacteria.

They produce antibodies, but they engage in normally do engage what are called microglia in the brain. And that's thought to play a major role in Ms..

And by the way, these cells get infected with the Epstein-Barr virus, which is probably a major trigger. We know people who have EBV are about 30 times more likely to develop that mess.

So bottom line is these are very effective drugs. But they do have some downside insofar as, you know, in functional medicine we talk about like root causes or upstream versus downstream.

And when you inhibit those B cells, and not all B cells, just these auto reactive ones, what are called CD 20 positive B cells. And not surprisingly, there can be some consequences.

And that is, you know, more often you have infections, upper respiratory infections, bladder infections, things like that. And since Ms. is a central nervous system disorder, meaning the brain and the spinal cord, not your peripheral, nervous system, the nerves, like your immediate nerves, the carpal tunnel nerve, it doesn't have anything to do with that.

Question might be. Well, what if we can go even more downstream if we can really engage these B cells when they are in the central nervous system and stop them from interacting with the central nervous systems, own, immune cells, the microglia, rather than mess with them when they're out there circulating in the periphery.

So we have this, enzyme. We all do. It's in our in our, immune cells called a Bruton tyrosine kinase inhibitor. And if, if this, particular enzyme is blocked, essentially it, plays a major it appears in, you know, an animal models at least that we can inhibit the activity in the central nervous system but still protect the immune activity in the periphery outside of the central nervous system.

That's a great theory, right. So there were about five different compounds that have been or are being in different stages of testing and clinical trials.

And we've recently gotten some results which are very interesting and in some ways very exciting. But and, but with some surprises as well. So we've seen and or these are like early press releases, you know, doctor was by the time this, interview is aired, it will be past us.

But when we're now interviewing, there's a major European meeting on. And that happens every year called electrons and atoms is coming up in about a week.

And and these big, big meetings are platforms for the, the researchers to, sort of roll out to show their results, unveil their results. So we have two main, of the of the 4 or 5, we have two main BTK inhibitors, one is from Roche Pharmaceuticals or Roche Genentech, and the other one is from Sanofi, Sanofi Genzyme.

And actually we've been involved with researching both of those. There's there was another one from Serrano which I'll touch on in a minute. I believe Novartis has one.

And I think there's one other one. I don't know the status of the Novartis one. So what did we learn? Well, we learned that in it appears that in both the Roche, compound, which is called Stanhope Bruton Ed and the, Serrano, not Serrano Sanofi, compound, which is called tola ibrutinib, that the BTK inhibitors appear to have a very significant effect on people with progress of multiple sclerosis.

There's a caveat here. There are very few, proven or approved drugs that are effective in this, but we've called stage more than this. And, you know, typically, classically, we talk about masses having a relapsing remitting stage as, you know, where their attacks.

And then the person may be recovers. They may not recover entirely. So it's kind of like climbing steps where you're going up and your level and then up and your level and then up and then your level.

But in this case, these changes leave people with more and more disability, potentially over time. And there are many drugs approved for relapsing remitting Ms..

There are no clinical trials today that study new compounds for relapsing remitting Ms. that are placebo controlled. All of them have what are called active controls.

And I call this sort of the the like the who are we going to beat up on and the, the popular drug to test these new compounds against it is called a Baggio or turf, in which is a fairly low efficacy drug.

And what has happened especially, what we saw with the, Serrano one and then with, the Sanofi tola Brut nib. Is that it? This study, this large study failed to show that there was a difference in efficacy between a Baggio and these BTK inhibitors, even though their sort of close cousin predecessor, the B cell to leaders like Ocrelizumab, were highly effective, highly effective.

However, what both Roche and Sanofi have found, in different stages of clinical trial. Roche's in state phase two, is that these drugs are effective in shutting down new acquired lesions in people who have progressive mass.

So it's really important to understand that, I can tell you Serrano made a decision not to just shut down the drug. They they it was, you know, maybe it was effective from a, you know, compared to, if you look at the, a Baggio group that was equally effective.

So we couldn't show a difference. But if you're going to develop a new drug, that's a huge financial investment, and there's no point in and rolling out a treatment that's not any more effective than something that's already out there.

Probably a lot less expensive. So they just shut down their whole program now. The same thing happened with the Sanofi, Tola Bruin ab. No more effective than, turf Luna mild, but we have now, you know, taller ibrutinib and thinner Bruton AB that appear to really stand out as promising treatments for a progressive mouse, which, like I said, even if you look at Oak Liz Mab, that has an approval for progressive, then that's primary.

Progressive. The benefit is, is very, very marginal. So a stop for a because I really want to highlight this, because I have secondary progressive Ms..

In most people with relapsing remitting Ms., assuming that you can continue to live, you will eventually can convert to this progressive phase of the illness where we're not having relapses.

But the concern is this relentless progression of worsening disability. Yes.

Progressive MS, Age, and DMT Decisions

And so this is a big deal, that there are a couple of drugs, they aren't yet approved, yet. So you can't we don't want people to think that I've had these kind of questions.

They people, you know, everybody's on Google. Let's switch. Okay. That's great. Our patients are sort of more informed, although there's something to be said for the experience and the knowledge that we have to be able to interpret the data, correctly.

But, you can't get these drugs or anything, but they will be available. I would say, you know, just looking at timelines of other drugs. We were involved in bringing key symptoms to market.

We've been involved in bringing the new Alzheimer's drug to market called Donanemab from Eli Lilly. So from the time that the studies are finalized and, paperwork goes in to the regulatory agencies, the FDA, and then eventually, what's called CMS centers for Medicare and Medicaid Services, you're generally probably looking at about, I would say about six months or so.

It's what I've seen. So they undoubtedly will probably be. So they'll eventually be approved. And for people with the progressive phase of the illness, this would be a useful drug.

Now, you and I both have the perspective that while drugs may be, you know, FDA approved for your condition, it's

important to also go beyond the drug in terms of what is it that we can be doing if we have progressive Ms.

or relapsing remitting Ms., because maybe the listers will have relapsing remitting Ms. from your point of view, in addition to, you know, a highly effective drug for either relapsing remitting or for progressive Ms., what else is important to be doing?

Well, I think, you know, I want to I want to if I can very briefly circled back around is some observations that are indirect from these studies because it's really important.

Look what what have we learned, you know, about. And this besides just does it drug work or not work. And there is a wonderful present. I actually sent it yesterday.

There's a lovely, platform that doctors have access to, which is like watching a, academic lecture every day on different relevant topics, say, in this case and in this.

And it's called, view medi. But anyway, we look at nce over a lifetime. So we talk about the average age at diagnosis as being somewhere around 34 years old.

Certainly is pediatric. And that's that's a whole other issue. You know, there's a whole other set of issues to deal with when children get M. S. But they certainly do.

But what's very interesting, and I this is something I learned yesterday, and really never given much thought to it is if we look at just the population in the United States and we just break down the population by age, right, where we're the largest, you know, it was say, you know, what's the largest population like 25 to 35, 35 to 45, 45 to 55, 55 to 65 and so forth.

So what we talk about, the point is, while we talk about M. S as being a disease that affects younger people and in fact, the largest group of people affected by M. S, this growing, shifting population that's just getting older and older and older, it's not the young people, it's the older people.

Right. Because that's the demographic of the United States. And so we are dealing the the average age, the largest number of people in that in that demographic, age stratification, you know, strata is something like 48 years old.

And why that's really important is and this relates to your question is, as you know, whether I'm working with someone with Alzheimer's, whether I'm wearing some with Parkinson's, whether I'm working with some with ALS or M. S., these this is not these are not one disease, right.

There are common manifestations that I'll quote. Dale Bredesen, our friend, who says, you know, when you look at Alzheimer's disease, it's and think about all the causes.

It's like thinking about a roof with 36 leaky holes. And so in order to tackle that individual, you know, we're we're put that person with, say, Alzheimer's, mild cognitive impairment due to Alzheimer's.

We can't just target one thing. We can't think of Alzheimer's as if it's just one disease. We can't think of them as as if it's just one disease. So what is observed?

And this circles right back around to those B-cell depleting therapies. And the Bruton tyrosine kinase inhibitors is that earlier in the in younger people with M. S right.

Those 34 year olds even those children the pediatric cases that this disease is much more robustly expressed in the peripheral immune system that it is a very immune driven disease.

In fact, just this past week, so a couple of new folks for, my brain tumor program, my functional medicine program, very aligned with doctor walls. And, you know, these are very young people with very, very active disease, lots of

and lots of lesions, lots of enhancing lesions.

Fortunately, you know, from a from the disability perspective, they were they they were good. But that does not speak for a good prognosis. And we certainly need to jump in on that.

So if we think about oh, I didn't say that those older folks, those 48 year old plus. And by the way, doctor Walls, it led me to do some math. I hope it's okay. I know how old you are.

Yeah, that's. It's kind of. You want to say it publicly or whatever, but I went, okay, your your video that went viral and kind of puts you on the map.

The 2011 minding your mitochondria. And I said, how old was Terri Walls when she did that video? How old was Terry Walls when she described her personal transformation?

2005 2006 in that time frame of going from the tilt recline wheelchair to riding your bike, well, you were exactly that age. And what we learned, at least back then, back 25, what we've learned about these older individual is that the disease shifts from being more robustly peripheral immune.

There's an active B-cells crossing, you know, getting into the brain and that that blood brain barrier that's become leaky, like a leaky gut, to a disease that is more, into more compartmentalized, internal meaning the brain.

Right. More involving mitochondria binding your mitochondria, oxidative stress. Right. Micro glia. So now when we go back and look at these BTK inhibitors and say, why did they not work in relapsing remitting Ms..

But they worked in progressive mass. But the B cell type leaders, which are really kind of related to BTK inhibitors, but they're more a little more upstream, if you will, because they mess around with your peripheral immune system.

They're really effective, really, really effective in these younger people, right, with relapsing remitting M. S.. So I think that's that is so super important to understand that when we say what can we do from a functional medicine perspective, we're thinking about the big picture that everyone is unique and everyone is different.

And and age does make a difference. Yeah. H h Which then, you know, gets me to the next question. As we get older. Yeah. And people ask me this a lot.

I, you know, I'm getting older are the risks of the DMT that I'm taking now outweighing my benefit because I know that relapses decrease over the age of 45, particularly over the age of 55.

And at what age should I think about coming off? Yeah. And I think that's a complicated question. And I don't know that we have good research to guide.

And yeah, I tell people I want to do the functional medicine stuff to get you all fully tuned up. And my conventional neurology colleagues, we have some very interesting debates over what is the age where, and they aren't doing what you and I do with the functional versus stuff.

They just say, okay, well, we'll stop the drug because you're too old. What are your thoughts about stopping DMT and how does age and your functional medicine approach interplay with that?

Right. Yes. And I apologize because I didn't fully answer your last question. So we'll answer that now. And so, you know, as I've spoken for many years, for you on what we have learned about discontinuing disease modifying therapies, and since then there's been at least a couple of clinical trials.

There's one called Disc disco discs. It could be disc coms, but it's disco EMS, the Disco Trial, which was a multicenter, randomized, single blind phase for Non-Inferiority trial examining the risk of, new disease activity in multiple sclerosis patients who continue versus discontinue disease modifying therapy.

And, you know, for better or for worse, what the trial found is, is more or less what we've observed previously, which is that age does age is really the only major to terminate that we have.

And I would be for and we have to again treat everyone different. Right. You can have all the hope in the world and that's really important. But you also have to have objective data.

So we do want to circle, by the way back round to that octave test, which none of these included. Right. But some of the strongest data really looks at people who are older than 55 and even 65 or 60, just depending on the trial, where if that person has been clinically stable, there's been no changes on their MRI, their, you know, their EDS score is stable, no changes, no disability progression.

Discontinuing that drug really only leads to about a 10% likelihood at worst of relapse or or recurrent disease recurrence or progression. So age is really the main factor.

But if we flip it around and we say, okay, your newly diagnosed with M. S., how's it going to go? You know, what is the crystal ball? Tell us. Well, we know that, well, and this is more prevalent among women.

Men can do much worse with them, this or that, spinal cord lesions, if they have what we call posterior fossa lesions, meaning like cerebellum, brainstem, if they have just a, large, large lesion burden, you know, this the degree of their disability at the initial presentation.

So these are all major considerations when, when predicting how's it going to go for that person. And we really have to take that into account when we talk about functional medicine.

Because in my, you know, I have taken a bit of a shift in that, you know, I was a conventional neurologist and then I was like, wow, functional medicine.

This is what I want to do. And then I realized that both toolboxes have limitations, but together they are very, very powerful. And as long as we're approaching each individual person on that level that you are a unique story, you are a unique narrative.

You are. And this is not the same as doctor was a nurse or my or another, you know, person might have M. S. and we can say, I think it would be okay if that's what your motivation is to take this wall style approach and hold off on disease modifying therapy versus another person.

And I'm going to look at everything and say, I'm going to be honest with you, and I'm even going to look at your readiness for change, your willingness to make a commitment.

Have you been able to sustain that? Maybe you're in this disease activity score, right, because that's not even taken into account in any of these studies to say, look, you should always be following a wall style approach.

There is no reason that you shouldn't, you know. But for you, I'm also going to recommend disease modifying therapy. And then we're going to track that right.

We can track your disease activity, score your MRI, your physical exam, all those things over time, your 25ft walk, your peg hole test, all these things that we look at you know, and then we can say, okay, you've done really well.

Everything's stable. Where are you in your heart and in your mind in terms of stopping your drug? Because I can't tell you with 100% certainty where you're going to kick back into, you know, gear in terms of having active disease.

But I feel like you've done your homework and you've created a solid foundation for yourself. And if that's what you really want to do, I'm going to support you.

But we're still going to measure we're still going to track. That is so important. Faith is very important. Hope is very important. Belief narrative is very important, very important.

But we still have to be sort of transparent and honest with ourselves. And if the numbers are going in the wrong direction or the MRI is going in the wrong direction, you know, you need drug, you need drugs, but you should still be following the walls protocol.

And, to everyone who's listening, I want to reinforce that. Yes, for some people, you can do really great with a functional approach. No drugs, everything stays stable, and that's fine.

But others, your benefit so much from taking the disease modifying treatments. Can use them in his practice. I can't prescribe those drugs because I. But I send them to back to the neurologist with the strong encouragement that.

Yes, I think you should do both diet lifestyle plus DMT. And if you're in that circumstance, you might do perfectly fine without DMT. But everyone, everyone who's listening, please be sure you're getting your MRI's.

Senior neurologist making a very informed decision about how DMT is, play in your care. You mentioned, another concept.

Narrative, Functional Medicine, and Patient Readiness

The narrative. And you sort of, glaze over that pretty quickly. Could you expand a little bit more on what you meant by, a person's narrative on their illness?

So there's been a lot of work done in this concept of the illness narrative. The the sort of pioneer, of this work is a Harvard, psychiatrist named Walter Kleinman.

Arthur Kleinman. My, my, my fault. Arthur Kleinman, who wrote a book called The Illness Narrative. And then for me, there was a very transformative is actually a PhD thesis, done by a physician at Michigan State who, took Kleinman concepts and, wrote his thesis on what was called the diagnosis narrative.

You know, we have as long as human beings had been on the planet, and as long as there has been the spoken and not necessarily the written word, but the spoken word, we have told stories.

Story is actually the most powerful thing that we have. It's more powerful than any drug. It's more powerful. IT stories create every aspect of our reality.

My wife uses the word manifestation, right? Very important. And we we play out story in many, many, many different ways through faith, through formal religion, through our own personal belief systems.

What we tell ourselves, what we see, we're in the midst right now of an election time with two very different, you know, candidates. And the two sides can't really talk to each other because the narratives are so different and the people that support one can't.

You know, I don't get into politics here, but, you know, we all know what I'm talking about, right? They don't see what's going on on the other side because they're so close and they've cling so hard to the narrative of the candidate they prefer.

So this is the same thing with our lives, with their masks, with the symptoms that a person may present with. Right. I'm coming to the doctor. Right. A painful loss of vision in one eye.

I have numbness on one side of my body. And then do you have that mass? Right. That's often the referral that I get. This person may have M. S. there are formal diagnostic criteria, but what's very interesting, too, because much of our health and wellness and disease and illness is driven by a part of the brain called the limbic system that really receives information from all our senses sight, smell, touch, sound, everything, and then decides, is it safe?

One of my favorite lines from a movie called Marathon Man is it safe yet? And Or is it time to defend yourself, to be in fight or flight? And when you're in fight or flight, no matter what is driving that physical imbalances, emotional,

spiritual imbalances, the brain will send out signals you may have muscle cramps.

You may have tingling. You may become very sensitive to light. You may have pain all over. And in some ways, as individuals can look like they have Van ness or sound like they have.

And that's from the story they're telling. But when you do an MRI, when you go through the formal diagnostic criteria, they don't write. So alternatively, they might have Ms.

as you go through that evaluation, but you have to get that person to the point in their narrative and understanding what is going on. That's my role.

That's a doctor or a physician, some might argue a healer. What is a healer? A healer commands a narrative in a very powerful way. You know, the shamanism that is medicine.

And when the person then hears that narrative, and that's the gist of that Michigan State thesis, is that then defines who they are that determine that that's the pivot point where once you believe, if I tell you the narrative that that resonates with you, right, then you're ready for change.

But until you until I mean, if I use the wrong narrative and you could come to my office of the completely preconceived notion, I have Lyme disease, I don't have that mask. Right.

And until that says you know, you this is Lyme disease. I did the special tests and nobody else can do. And you have Lyme disease. You're not ready for change.

And, so, you know, I always say that even in functional medicine, our tests don't really diagnose things. And functional medicine. Not the way that we use conventional testing.

Our tests help us tell a story to our patients about who they are, what's going on with their body, and how ultimately, we can take a path toward healing.

So narrative is absolutely critical. And moving forward, narrative is critical because you can say, I love doctor walls, I love doctor walls, but, you know, and, you know, I can't tell you how many people will come to my office and they they know they're in what we call that sort of contemplation, stage where, yes, they they understand, sort of.

But in fact, the work is really hard, and they're not really ready for a change. Right? They're not really ready to do the diet and exercise and the mind body work and get their sleep dialed in and things like that.

Yeah. You know, I think telling the story back to the patients so they can understand what's happening and have it make sense to them and as I'm listening to you and sort of thinking, about my time in the VA when, I was very cognizant that as I'm helping people with their story and understanding, we had to have metaphors, a wide variety of metaphors that people could relate to as they would to try to understand, how they became ill and, over what processes they had control over.

So with, it's interesting, we developed farming metaphors, engineering metaphors, auto mechanic, metaphors, school metaphors to help people understand, and that it was most remarkable when people could tell the story back to me, tell their story back to me in a way that made sense to them and the folks who could do that were ready to really embrace the changes that were going to be very helpful to them.

If people couldn't tell me back a story, that made sense to them that I knew they it just was not going to work out because they weren't going to be able to make, any of the changes to improve their, nutrition, improve their self-care routine.

Does that match your experience? Can it does. And sometimes those narratives are very obvious, you know, and I I've worked with veterans, I trained in VA hospitals, and I love the veterans, I really do.

But they're a great example. And you'll see, I'll have a 80 year old guy walk in my office. He's still wearing his USS whatever hat. Right? I mean, he wasn't in the service.

He's not been in the service for 65 years or something like that. But he's still got is that's who he is, right? That's who he is. So veterans very much.

And, you know, I am so grateful for the service of veterans. So when I see those, I don't want it to be misunderstood because a lot of bad stuff happens to people in their time of service and deployed, whether it's burn pits and Agent Orange and getting injected with peanut butter vaccines.

Those of you who know what I'm talking about, but the reality is we also move through this thing called life. And so the person that comes in and they're, you know, experiencing symptoms and maybe they're obese and diabetic, they've been a smoker and they have dyslipidemia and all these other things, and lived a high stress life and been pretty sedentary.

But by golly, it was the Agent Orange that did it right. So really, it's it's really tough. It's, a challenge sometimes, too. You really have to listen.

And that's the part that unfortunately, too often has been laid to the wayside in, in a world of conventional medicine where volume is king and you have this, yes, many patients as you can and you don't have time to listen to those stories.

I do, because we, you know, I'm more of a functional medicine doctor, but but it doesn't happen often enough. And that's why we get stuck on pills that, you know, no one's checking on.

And to to your point, I mean, I see, I say to look, you know, you're on this cholesterol lowering pill and a blood pressure pill, and I'm not telling people to get off those drugs, by the way.

But if you were, I'd say my my narrative is, you know, if you have a clipboard and you're standing at the entrance to the emergency room and, you're, you're counting people coming in with chest pain and coronary syndrome or stroke, and you say, hey, by the way, you're diabetic and you're on medicine or you're hypertensive, you on medicine, you cholesterol, you're on medicine.

Yep yep yep yep yep. So if you're taking that pill and thinking that it's just going to fix everything and prevent the bad stuff from happening. Not happen, it's going to happen.

Right. So I see folks who are diabetic and they feel it. They're taking their medicine has permission to have it. Piece of cheesecake. You know that. You can't do that right.

And so it's a stepping stone. The drugs can be helpful. But but we have to adopt these therapeutic. Yeah. Supports. Yeah. You know those meds for the high cholesterol, high blood pressure.

They slow down the disease process. And so they're very helpful.

Remyelination, Exercise, and Practical Next Steps

But, unfortunately, people are not necessarily taking the time to reinforce just how critical what we do is, for managing all of those internal medicine type diseases that I take care of, that, drive all the terrible neurologic diseases that you take care of.

Yes. One one more thing I wanted to touch on, before I let you go, Ken, is I know many people have, this hope that there's going to be a pill that I can take, or maybe an infusion that's going to tell my brain to remind itself.

We might, you know, repair all that myelin. And I know there's that many, many drug trials that are out there. Do you can you give us, a quick update on.

What do you think? Is there going to be an infusion or a drug that will fix the reimagination problem that is happening? As part of aging, as part of, Alzheimer's and, of course, as part of, Ms..

And for everyone who's listening, we all want our brain to get re myelinated correctly. Of course we do. Yes we do. So there actually have been a few, investigational compounds, biotin, high dose biotin that's been looked at and compound called and I may not be pronouncing it right, Beck's, protein, which is a retinoic acid receptor gamma agonist and, and a monoclonal antibody that targets something called lingo one where when lingo one is activated, it prevents remote island nation.

But unfortunately, all of these clinical trials have failed, and their efficacy to promote brain re myelination. So even though it is certainly hopeful that we will soon, maybe or eventually have something that promotes rely on Asian from a drug perspective that does not exist.

However, don't be too disappointed because there are some very good things that you can do that promote re myelination. And probably the biggest one is exercise.

Exercise promotes regeneration nerve cell growth and re myelination. So if we're sitting in our easy chair, as we've kind of alluded to this whole time waiting for the drug to do something to us so we don't have to do it ourselves.

Well, do your walls exercise program because that makes a big difference. There are some other things that may be supportive, and I don't want people necessarily running out to the health food store and grabbing things off the shelf.

But it appears that CDP choline supports re myelination in women, particularly post-menopausal, progesterone. I I'm a firm believer in bioidentical hormone replacement therapy.

It appears that progesterone supports re myelination. The flavonoid compound quercetin may support remind the nation. So there are things out there that and again most importantly exercise.

Something's going to be fixed just with taking a supplement. But definitely exercise. We can do that today even without a drug, you know, as we age, if you become mobile, it really accelerates the aging process.

The atrophy, the demyelination that occurs with aging. And I know many people with that. Ms.. We, we're not exercising enough. We're feeling fatigued.

And so it becomes hard. People ask me like, you know, I'm already exhausted by ten in the morning. How could I possibly exercise? What do you say to that? Can.

I mean, I think that first of all, we have to meet people where they are. And we can start with simple things. Maybe some chair yoga, maybe the stem that I know you're, you know, very, very fond of.

But we have to get the body moving. It has to be a graduated exercise program. And then, on the other hand, you know, the people I ask them that exercise because a big component of what we do here at Shoreline Health Neurology is, neuro fitness or neuro fitness program.

And, you know, not everybody we have query giving them their whole neuro fitness plan and working with them. And do you go home and actually do it? Well, I walk, I walk, jog, I walk, run.

Okay. But is that your neuro fitness program? Are you challenging yourself? Is there enough of a stressor? A dose of a stressor that's a healthy dose that actually stimulates your body to grow and change.

If you do the same thing over and over again, you're only going to get the same outcome, right. You're not going to grow. You're not going to change. So that use it or lose it.

The other cliche that's very true, applies, you know, applies here. Okay. Well, it can, this is really been wonderful. I

always, always love our conversations.

And again, everyone who's listening, I want you to know, Ken's practice is where I send my, complex patients. And now that I'm not seeing patients directly myself, Ken's office is where I see.

I send all of, the people who are coming, asking, to come to my practice. So, Ken, the most important thing here is how do people find you? We have a website which is functional medicine.

Dot doctor spelled out doctor. And you can go on that website and sign up for a free telephone consultations. We can learn more about you and make sure that, your situation is a good fit and you'd be very happy.

But we see lots of folks, courtesy of Doctor Walls and have done so for many years. We have many patients who are able to, stabilize, even reverse the trend.

We have cases of, one case in particular of complete resolution of the MRI changes, which is astonishing to me. But, you know, it gives people hope that if they can emulate those changes and on an individualized level, they can have similar outcomes.

Thank you. Thank you so much. Thank