Introduction and Guest Welcome

Welcome to another edition of Mycotoxins and chronic illness. And chronic illness has always been my focus. And this today, I it's my pleasure to, interview and discuss long covid with doctor Mobeen Siad Saeed like pronunciations.

I apologize, I can always fine. And, he is, CEO of doctor B. Com, a wonderful resource for medical information and also, host of his podcast, which I think has been a wonderful source of information on what's real and what's the real science in Covid.

And I said I've listened many, many sources and I keep coming back to, doctor Sally as the man to listen to. So, with that, I want to welcome you. And, today we're going to talk about long Covid.

And, where would you like to start? So thank you very much, Eric, for having me. It's an honor to be here with you and discussing the chronic diseases.

I am a baby in the field of chronic diseases. You are a master here. So, thank you very much for giving me credit as well. But really, I am a beginner. The, for Covid, it actually become.

And I think we all became accidentally pushed into not having to learn about it. There was no other way for it. So for Covid, as I have been for the last two years presenting various studies researches, I have collected a number of studies and based on those, a number of possibilities for chronic Covid, a long Covid, both after the infection and after the vaccine.

And then what are the managements and what have been the successful managements. So I think that is the topic that you have. And that is what we will discuss today.

So once again, thank you for having me. Thank you. And again, thank you for being here and organizing the information. I think that's the most important thing.

People don't realize how much work you put into organizing the information for us. So thank you. Thank you very much. So, shall we start? Yes. Let us get going.

That is excellent. So so the way we will discuss today. And please, Eddie, discuss with me during this time as well. Add in your comments to, the way we will discuss this.

Number one, I would like to show some references to various pathologies that we have seen so far. Data is still evolving, information is still coming in, will know more, and the and the management will become more refined and more specific at this time.

Long COVID Research Overview

It is, I believe, refined enough, but not fully. There. Secondly, I would like to show a reference to FLQ site where the I recover protocol is present that I have led and many other doctors Doctor Paul, medic, Doctor Peter Cody, Doctor Tina Pierce, Doctor Bruce Anderson, doctor you.

They have all kind of worked together to pull this protocol together, which has been working fairly well. The disclaimer I want to make sure I have no financial or other, interests involved with any of the parties I will discuss here.

I'm going to discuss in cell. I will discuss the cell trends. I will discuss civil society. I have no interest at all other

than they are good people who are doing good work, and I use their work to share that information with others.

No financial interest, no strings attached. So that is the structure. So I'm going to share my screen very quickly and start from the references. So here if you see this is the flux site.

So you can write FLQ dot net. And that would redirect you to Covid 19 critical care.com. Or you can directly write critical care in here if you go to protocols.

And then if you go to I recover management for long haul Covid 19, you will see that this protocol is led by me. So the approach outlined below is a consensus protocol based on a collaboration led by movie and say it and a all.

So whenever you are looking for managing long Covid the audience here I'm expecting them to be medical professionals, medical students, nursing students.

This area, for example, we are updating it right now and maybe in another week this would be further updated with the first line. I'm going to discuss some part of that today, and then you can follow that here as well.

Now for some of the studies that have shown the pathogenesis, which we will discuss some of that today and then see what could be the clinical signs and symptoms and then how to manage them.

So the baseline studies at the following number one this is a study or a paper development of phase two antibodies after SARS-CoV-2 infection. So there is a professor here Professor William.

He has done this paper as well. There is another study that I'm presenting here. There was a, network hypothesis by an immunologist, last century where they had said that.

When we make when we encounter a pathogen, we make, of course, antibodies against that pathogen. Then what happens is that once the antibodies are produced that are now attacking the pathogen, for example, if you use Omicron or SARS-CoV-2 as an example, as it arrives in our body, we make antibodies against the spike protein and other parts of it.

Then what happens is we need these spike protein, these antibodies themselves to be cleared out and to clear them out. One of the homeostatic normal mechanism, normal response of our bodies to make antibodies against these antibodies, to clear them out.

This is a homeostatic function. This is a normal function. However, in some percentage of people those anti antibodies are also called anti Ido type antibodies.

They do not go away after being produced. And now they would continue to attack our own body because the original antigen now looks like these antibodies look like the original antigen.

So I will discuss this a little more later on. Yes.

Spike Protein, Monocytes, and Blood Cell Changes

The the result of that is we would end up with auto antibodies to Ace2 as a result of either vaccination or the infection. And so this is that network theory that I discussed or I showed you.

Then continuing with the references, there is a lot of work done by Doctor Bruce Patterson and Doctor U. And that work is about the monocytes and monocytes getting pieces of spike protein, especially S1, in them, for a long period of time.

This is the paper. This is the fresh paper here. 10th January 2022. Persistence of SARS-CoV-2 S1 protein in CD 16 plus monocyte in post-acute sequelae of Covid 19 up to 15 months post-infection.

And so that is a very, very important paper. And they have answered a lot of questions. But I wanted to talk about one important statement here, and that is that their observation is that up to 30% of the people infected could become long Covid.

That's a very high number. That is a pandemic in itself. Yes. So not only this is a disaster, but this also puts a lot of burden and responsibility on doctors who are managing chronic diseases to be aware of what is happening and how the pathologies are evolving, and then how to manage, because these patients are now part of a daily life and they are suffering and the misery just continues.

So this is a great paper to look at. Then there is this paper. This is the, long term changes to blood cells triggered by Covid 19 infection. So in this study, the researchers found that it is possible that the blood cells are RBCs because these cells can lose their morphological structure, become a little rigid, and most importantly, when RBCs go do that, then you know that RBC would get stuck in various blood vessels and the clotting would occur and and blood flow disruptions will occur.

So that is also an important thing to keep in mind. I also wanted to say from this one monocyte the outcome of this, if I could summarize it. So you have the seed planted in your mind that is monocyte carrying S1 and then patrolling the boundaries of the blood vessels will mean that there will be vascular inflammation.

And when that vascular information is towards the head and neck region, then there can be a lot of neurological disruptions. So now imagine inside the blood vessels we are getting issues with the clotting because of and the blood cell shape changes.

And outside of the blood vessel there are monocytes that are dysregulated. And now blood vessel is attacked from both sides and it is inflamed and the blood flow is disrupted and clotting is occurring.

That is what is a possible mechanism that is happening in long Covid. Then finally in nausea. So this is a study about who knows me knows me. It has been a very common, outcome as well during the long Covid.

Interestingly, some people's anosmia recovers by itself. Some people, when we give them therapies, the, you know, who knows recovers within a few days, two weeks.

And for some folks, the first year of or hypo hypoxemia continues for months. Yes. So these are the basic, studies that I wanted to first put in front of our audience.

So far, so good. Eric. Yes, I said each one of them. It makes me want to dive deep, but I'm going to let you keep going before I distracted take us off the path, because, I was going to get through these is.

These are just wonderful. Absolutely. So I think what we can do is this today we'll talk about management so that a more practical value is in front of the, doctors.

You can use it. And then, we can do a separate series where we can go individually for each one of these studies and discuss them in depth. Yeah. Because they, they, because they just to come again, they tie in they, they give us a window and more insight into what we see in chronic diseases of all types.

I mean, that's the beauty of this work. It's not it's doesn't stand alone. It's trying to gather information and data that we've been looking at and just giving us a ways of putting it into more practical, so we can really begin to use this to help patients.

So just perhaps a little we'll, we'll go with, what we can do today. Absolutely. So I'm going to go back and now I have a presentation as well. I am very, drawing oriented to.

So every so often I'll, I'll go to my drawing board and draw some of the concepts to. So be ready for some drawings. So I enjoy this question. Thank you.

So first of all medical disclaimer these are not advices we are talking appears. We are talking doctors and sharing information, sharing other people's researches and observations of doctors with the medicine.

Of course if this is a patient who is listening please talk with your doctor. If there is a doctor who is listening, please make sure that as you do all the time, case by case, the management may differ.

So let's start with the possible causes for the long Covid. There is a theory in immunology of virus hit and run where the virus arrives, does some disease, causes some pathology, and in that process ends up triggering the immune system.

This regulating it. And then the virus is wiped out, but the immune system behind is still dysregulated. It is still wobbly. It is still incorrectly functioning.

And that continues to go on. And that becomes the basis for a chronic disease. So that hit and run is possible. I think it is very much possible with Covid.

That is one the second pathology, and I have given the references before the S1 protein pieces sitting in the monocyte. And again you have a you may have a lot of question that why are they sitting in there?

How long would they continue to sit in there? Will they ever get eradicated? Would they ever be cleared? What is the outcome of the S1 sitting in the monocytes?

All of those questions are in the paper. And the answers to them. I just and just to specify for people this is a piece of the spike protein. We've been talking.

But this is correct. Absolutely. And so, if you would like, give me one second. So I hope you can see my drawing board now. So on the virus. So let's say this is SARS-CoV-2 on the virus.

The spike protein is divided for for its functional pieces. It has it is scepter binding domain or RBD. This is where it binds to Ace2. So that is one.

So this is. This blue part is the Ace2. This is the remaining cell. The red part is the receptor binding domain. Then for the remaining part of this spike protein there is an S1 unit.

And it is an S2 unit. And for completion see what happens is when the when the virus comes and docks with the with the S2 this is S2 the blue one. Right. And the virus docks with the Ace2.

Then the Tmprss2. That is a surface protein or not cells. It cleaves it part of the S1 from S2, which causes this whole S1 and S2 to become separated.

Now S1 stays stuck to the Is two, and it is actually recycled or downregulated and brought into the cell for digestion. However, the S2 and please don't mind, my cat is here with me and it is going to start meowing.

You just woke up. The S2 portion that is then made naked because S1 is removed. This has two portion, acts as a fusion protein and it connects with this cell membrane.

And then it allows the virus to fuze and send the other. This is the normal function we are talking about this part. The S1 part sitting in the monocytes.

So let's say this is a monocyte. This S1 part is somehow hanging out in monocyte for months and months. And some people. And that causes this monocyte to become dysregulated because it has a an antigen in it.

Neurological and Sensory Symptoms

And it would present that. And the other cell would trigger it. And the monocyte itself would continue to be triggered

and make cytokines, which causes it to cause continuous inflammation.

Now the monocytes are again they are in three states. I'm so sorry. I'm going on with all in this steps. You can stop whenever you like. This is this is fine.

Just one thing that's always interest me about this story is that the monocytes normally has a very short lifespan, I would imagine. Correct. And so there's something about this.

Once it's in there that kind of immortalize. Is it a bit correct. So then discuss that in this paper as well. There are a couple of possibilities. So let me complete, I the part and then I'm going to address that question.

That's actually a beautiful question. And it is asked very often. And that was my curiosity as well. And we still only have conjectures about it. So let's say this is a blood vessel and outside is a monocyte monocytes usually patrol the boundaries of our interfaces, our tissue interfaces.

Blood vessels are interfaces. There are other interfaces as well in our tissues. So monocytes are patrolling there. And when needed they can convert into macrophages.

They can convert from classical to non-classical and intermediate. However if they are irritated by this antigen presenting them then they continue to create inflammatory state around the blood vessels which causes the blood vessels to become inflamed.

And we see a lot of pathology related to that. Yeah. So having said that, the question, monocytes are supposed to be either short lived or convert into macrophages and B long lived.

How do they start surviving? So in this paper, Bruce Peterson and your doctors, they discussed that it is possible that the continuous irritation of the monocyte makes it not become cleared out, or it is possible that the one monocyte is eaten up by another as part of the regular homeostatic mechanism, whereas the senescent cell is removed by phagocytosis but from another cell, and when it is eaten up by another cell, then this spike protein just transfers to the next one.

So it is now going from cell to cell and sticking in there and keeping the dysregulation continue. There may be more mechanisms, but these are some that they have done.

Now if I go back here to the discussion. So S1 proteins sticking out in the monocyte and continuing around the blood vessels and inflammatory state is a very important, possibility for long Covid.

Then as I showed you the reference before, there is a possibility of the blood cell morphology change. We especially the RBC shape change can cause issues, as we can all suspect.

It is that from changes in the, the membrane lipids or so it is they say that there is a change in the membrane lipids, which makes the cell a little more rigid.

Then maybe microtubules changes as well. But more importantly, they have observed the shape changes. Exact pathology is still not clear. Right. Yeah. Well and again I some day we'll talk about this.

And I think I can relate this to mitochondrial but RBCs don't have mitochondria right. So okay okay. So there is something going on with the RBCs and the other blood cells as well.

And the end result is they become a little more rigid and their shapes become dysmorphic. And so they're not easy to walk around in that blood vessels or fly around in the blood vessels and they get stuck in because issues.

And that would then start the propensity towards clotting. That's what then the autoantibodies to Ace2. This is the mechanism that I was discussing before.

I'm going to quickly draw this, to see if I can. Make sense of or I'm able to present it. So let's say here is this spike protein. And this spike protein again.

Now you know the pieces of it RBD this is RBD receptor binding domain. This is S1. This is S2. Now we know that we can make spike antibodies against the RBD.

So let's say we make an antibody that binds here to the RBD. This is the antibodies binding site. So this is the other binding site. We know that there is a light chain and a there is a heavy chain.

I'm just simplifying my illustration. These are not normal illustrations that we see. So here is the binding domain. And this is the function of this antibody.

So this antibody is going to now bind with RBD and try to clear the virus encode the virus and do all the biological functions that we are aware of. Now imagine our body decides to make another antibody and we're going to make it green, which is against this antibody.

And so that antibody learns to bind with this antibody. Right. So this is the binding domain of this antibody. This was the binding domain or the binding region of the other antibody.

So you can now see look at the red antibody first the red antibody is connecting with the RBD. So if there is anything that is going to connect with our red antibody then that thing has to look like RBD right.

Otherwise the binding will not occur. So when our body in all its wisdom to try it, it's a homeostatic normal mechanism which is called network hypothesis.

When our body makes antibody not your typical antibody. So this green antibody is called anti your typical antibody because it is against another antibody.

Right. And it is against the in your typical part of the other antibody. But here is a delicate point here. This antibody the green one. When that is formed it will have an appearance of the spike proteins RBD.

Only then it can bind with the red one. And now because this green one has the appearance of the RBD, this green antibody can start binding with the Ace2 receptors.

Okay. Because it looks like this antibodies binding region looks like this spike proteins binding region. So when it starts binding to the Ace2 receptor, that is where the dysregulation starts coming in.

Now the interior. So the paper that I showed you this saw that the antibodies, auto antibodies in hospitalized patients, 93% of them had in the outpatient, about 40% had them.

And they saw that most of them got cleared out within a couple of months. But in some people, these just persisted and stayed on. Will they eventually get cleared out?

I think so, but at least by the time they were doing the research, they found that in some people they were still there. Yeah. Now those auto antibodies are going to stimulate Ace2 and they're going to bind with these two and do the similar regulation as spike.

This. Right. And you know, so we started having problems with you know, all the, all the functions of Ace2. But you think that this would go away when the, when the danger signal, if we could get the dangerous signals to go away, usually the autoantibodies should dissipate.

That is correct. So this is a homeostatic mechanism. Right. Ideally. So pathogen comes in. Antibodies to the pathogen are made then auto antibodies to the antibodies and antibodies are made.

Then all of the system pathogen goes first then the antibodies to the pathogen go. Then the autoantibodies go back

to the sequence. But in some people the sequence fears and the auto antibodies just hang out there right.

And that they cause dysregulation. And in case of. So you could ask this question that why in case of Covid it ends up with the long Covid with all variety of symptoms and all those issues.

And the reason is that the binding region for the auto antibody is very important. It is binding to Ace2. Ace2 has a hugely important function in our body to manage inflammation.

So as soon as we disrupt esters, we put body in a chronic inflammatory state. And that is the underlying problems with the chronic diseases. There is a chronic inflammatory state either triggered by the virus or bacteria or whatever, or by our immune system just doing it by itself.

So here we have an autoantibody continuously disrupting is tumors disrupting the inflammatory systems, balancing system and causing an increased inflammation in the body.

Tendency of the inflammation okay. Continuing then you would see in many Covid patients there is tinnitus. Actually I have Covid right now and I have some tinnitus which comes and goes some patients.

It's really horrible for them. Yeah. So tinnitus can develop. Then there could be a balance problem that can develop or hearing loss that can develop.

And the reason for that is vestibular cochlear dysfunction, in which one thought so far is that because the blood vessels to this area are very delicate and small, these a capillaries, they can become easily clogged by the other mechanisms that are discussed.

And that can cause. And one second, if I back up, we also know that Covid causes clotting tendency to. So if we put that all together, it is a possibility that vestibular cochlear systems, especially hair cells, are not getting their oxygen correctly and nutrition correctly in the waste products are not cleared out correctly, and that causes the disruption in their function and tinnitus occurs.

If that is happening to the hair cells in the semicircular canals, then the balance issues would occur and if this damage is permanent then the hearing loss would occur.

This is a problem for, you know, Covid as well. The signs of it starts within the Covid, but then they start persisting. Yeah. And again, just we see this in in again in many chronic illnesses when the red blood cells start to stiffen a little bit and you have a little bit more fibrin fibrinogen floating around, it doesn't take much to make that very difficult for that red blood cell to get across out of the blood vessel.

It's absolutely, absolutely. And then one of the outcome, it would create outcomes in all parts of your body. But there are some outcomes which become very, very prominent for us.

And we start noticing them. And that's why the more narrow the area, the harder it is. Yeah. Right.

Management Principles and Early Treatment

And then, the before I go to the GT, another there is one, bullet point that is missing here and that is in those Mia. It knows Mia or hypo knows Mia or hypoxemia are very common complaints as well, where the patient stops or has reduced smell, or they even have phantom smells.

They say all of a sudden I smell coffee, or I smell a bad smell, and that is because they've developed hypoxemia. So I had shared a, research there before in the early part of the discussion, there is the researchers had shown that the epithelium of the olfactory bulb, not the the logical pieces or the olfactory nerve, but the epithelium of the February bulb and the supporting cells.

They become infected. And the inflammation, local inflammation presses on the olfactory neurons and dysfunctions

them or causes dysfunction of them just because it is an inflammation of the epithelium.

And if there is damage to the epithelium, that damages the whole system's function. So, it knows Mia is also another important thing to keep an eye on.

So if I'm going to know. Continue. Long Covid is a set of symptoms syndromes which can be clustered in various parts. And in some patients they can be combinations of them.

All of them. Some of them long Covid could be just neurological, GT, respiratory, musculoskeletal, cardiovascular, autonomic or a combination of them.

I have seen mostly combinations of them, and the most disturbing for the patients are neurological. Where there is brain fog, there is lack of, concentration, lack of the ability to process, cognitive decline.

Possibly in those Mia tinnitus and so on. So there are lot of neurological issues that patients find really bad and they cannot perform their functions correctly while the tragedy at this time is that they are not recognized very well, and so others think that they're just making it up or this is just psychological and that makes it even worse.

Now the disease is courses, hills and valleys. Patient feels better. Patient feels back. They feels better. They feel worse and so on. They just keeps going through that process.

And one thing I forgot about new logical Bell's palsy and its recurrence, yes, is also a very important part of neurological disruptions. Yarbrough is mostly acute, but Bell's can actually be fluctuating in post Covid for a long time.

And even with the vaccine for a long time. So hills and valleys, persistent low intensity, persistent high intensity, then temporary improvement with intervention, then permanent improvement.

So this is very important that sometimes as we discuss the protocols you would see that patient has improvement. And then they have a relapse. And that means you have to try a different avenue.

There are multiple pathologies. And so there is. But all of them give rise to inflammation. So when you control the inflammation you would see a good response.

But now what is the underlying pathology that needs to be addressed. So you may have to do some, research some exploration by trying various possibilities, management possibilities to see which way works.

And just, you know, this is a beautiful slide because, you know, I think this is what you see when you try to treat, you know, chronic Lyme disease or chronic fatigue. But, it is this laid it out just so, so simply and just made it so obvious because when you're dancing with the immune system and, you know, it's like, it's like, miss, you know, just the way miss was considered a psychological disease till we got an MRI and, you know, till we could really see that these people had something wrong with them.

Or we could see the, the, the abnormal proteins in the CSF. But, you know, all these diseases have been delegated to the psychiatrist. And much to the chagrin of the patients.

I mean, I think that's what causes even more of the, well, it increases the, debilitation is when you're just not respected by anybody. Absolutely. Yeah. I have a very young, patient and not my patient, but family members.

She's a patient of long Covid, young woman, very dynamic, very energetic, looking forward to her life. And all of a sudden, she got post vaccine injury.

And one big part of that was the and is still persisting, but thankfully has almost 90% gone was brain fog and concentration, difficulties and ability to work and cognition, cognitive decline.

And she would tell me that when she would say this at here to her superiors, a workplace that I'm having these difficulties, her peers would laugh. I mean, they're her age as well.

So they'll just make fun of her and say, you're just making it up. And the her, management would not even consider. They would say, talk to the doctor.

And doctor would say, this seems like a psychological issue. Yeah. So this is how bad it is. Yes, yes, and I suspect that. So you are working with the chronic diseases.

I suspect this is similar everywhere. This is everywhere. This is and it's gotten actually has improved tremendously over the last 20 years. I mean, if that's if you can imagine it being that's bad now, it was it was impossible.

Well, in the past it was just totally patients were generally just ignored. You know, they lost their families, their friends because, you know, nobody would believe them.

But but what's really so important here is to understand that when things when, when symptoms wax and wane a lot, just remember it is your immune system is dancing with you.

You know it's not a fix. People have this concept of the body as a fixed thing, you know? And our medical pictures are broken legs and bullet wounds and heart attacks that happen in the moment, and not the waxing and waning of your immune system.

How it dances with the world. You have a good day of a bad day. You're in a good mood. You're in a bad mood. I mean, it's life. And anyway, it's just so important that people remember that when you're dealing with these diseases, you just seen a reflection of what it is to be human, that there is change every day.

Absolutely. So, with this management consideration. So of course, like any chronic disease, the considerations, there are prophylaxis that should be done within the disease itself so that the chances to become long Covid are less.

And what I've seen is that ivermectin has been very useful, treatment with steroids. As soon as the viral phase is over, it's also very important to start controlling the immune system from going totally dysregulated.

And then other, prophylaxis, for example, vitamin D and other such substances should be in the correct amount. That is important. So that is one consideration.

This consideration is more than for the chronic for the current chronic disease doctors, this is mostly for acute disease management, managing physicians because they have to take care of it.

That patient doesn't become or has less chances of becoming, long Covid. Then the logical component, sometimes is not just very easily handled with the management.

And so one has to look into various techniques, including lymph flow massages, which some people think it is crazy science. But this is actually important because it helps move the lymph a little faster.

And that helps clear out some of the debris that is collecting a little faster and move things a little more. Yes. When we talk to the like, you know, one of the difficulties is when people first get ill, they're often not very sick.

And it's, you know, and so it's very hard to say, well, you know, try the ivermectin and the lupus I know you don't need it today though. I think the FBI director and I've seen turn people around overnight when they're moderately ill sometimes, you know, doesn't work all the time, but it's worth trying.

But it's people understanding that, trying that you don't want to wait until you are, in the second week and very sick to start doing things, to try just simple, safe things first, then be willing to, you know, know that you're not that sick.

It does feel like a call, but this is a call that can get a lot that can get a lot worse fast. Correct? Absolutely correct. Yeah. So then in some patients, pulse therapies on a monthly basis may be needed.

So I've seen that for the management sometimes continuous management helps. But in some patients continuous management does not help. And you've got to give a break and then resume and then break and then resume.

And that slowly helps them to come out of this situation. And so sometimes pulse therapies are more important than continuous. And then disease usually goes in remission within a week or two.

Then the patient goes and bounces around and does their thing and be happy. And then they come back with a relapse. And then it seems like a couple of months are needed for a continuous therapy.

And what happens is, one of my friend doctors were saying that usually the benefit starts appearing in six seventh week, but patient doesn't wait for six seven weeks.

They just want it for their medicines. Today I should feel better. So there is a lot of, close consultation with the patient to have them go through this journey.

Yes. Patience. Patience, absolutely. And just one of the thing is, what we see all the time in chronic illness is toxicity is what's keeping a lot of people stuck in chronic inflammation.

And just as you're saying, the lymph flow, the chronic, the TMJ, the chronic neck tension, that is, you know, we we spend our lives sitting at desks with our arms up, typing, that is the things that, that we don't usually attend to.

Because if you have a cold, it doesn't matter. But when you have chronic inflammation detox is critical and absolute and attention to structure, you know, through chiropractic, osteopathic, all these different ways of of dealing with, with the structure will help.

Again, it's not the magic bullet for most people, but it's important component to getting these medicines to get people where they want to be with these therapies.

Absolutely. And now that you're seeing it, I'm looking at my shoulders. I'm sitting like this because my my chair, elevated. So I'll try again. One of these days we have to talk about your ergonomics.

Yes. Yes, absolutely. So okay. These are some considerations. And the second set of disabling. So the most disabling and disturbing things for the patient are neurological symptoms.

And then the cardiac symptoms. So focus on those and help them through those. So now let's look at a general idea for how to management the basic approaches.

And this approach changes. But I want to put my thought out. And of course folks who are in the chronic disease management, they know better than me. And that is that I'm not so sure you are to two modest, but okay, there is a propensity for inflammation that is causing damage.

And what I've seen is that many patients, let's say that there is inflammation of the cranial nerve and the swollen cranial nerve is now slowly getting damaged.

If we don't control the inflammation, then it is possible that the damage becomes permanent and then we can do whatever. It's not going to be reversed.

So there is an immediate need to keep the tissue damage from happening, or to reduce that as much as possible while you figure out what is the basic pathology to address.

So in the beginning, steroid therapy is important. I have seen many, protocols which deviate from steroids, and they want to use more specific chemokine blockers.

That is interesting as well. What I have seen is that there is a side effects and their, specificity has its own issues. Generally, low dose steroid for a couple of months is something to keep patient on while you're figuring out what else is wrong, and to fix it.

This reduces their damage while you're treating them. So now labs, there are a couple of, companies who whose labs have been very much used by the chronic, long Covid or post vaccine injury patients.

Testing, Immune Dysregulation, and Protocols

One is the cell trend. It is not in the US. It is a, I believe, European company. But they do these auto antibodies for Ace2 and many other parts there.

They have a beautiful profile of auto antibodies that they do. Oh yeah. And that is a big thank you that this is very interesting. Yes. So you can go to I believe Denmark sell trendy T and if I remember it correctly, let's actually very quickly see it so that we can see.

And again I have no financial well trend dot dot dot slash it. I don't know if it is cell trends or cell trend. So my apologies if I come up with some weird site here.

Okay. So we are on the correct site. It looks right. Yes yes yes. So they have a great profile of auto antibodies that they do, which are not available here.

So you have to work through their site with them. But I am seeing so many patients I, I have interviewed the patient Sean, he has been working with them as well and got his profile.

Okay. I know that would be very interesting because I'm, I'm very interested in, future use of plasmapheresis for people who just aren't responding. This would be a yes to help.

Okay. Correct. So that is one. The other one is the insult. Once again, I have no financial interest of any sort. Yeah. Incidents have created their own, set of tests.

And with that they create a long Covid index and they save the indexes here. Then you need management this way and so on. So they have that as well. I think it is 360, $70.

I do not know if the insurance covers it, but again somebody who is in trouble might want to. It's well worth that. I recommend that I'm actually doing the on almost all my patients who I'm stuck on, whether they have long Covid or just, you know, chronic fatigue or chronic Lyme or whatever, I think it's useful.

I've done lots of cytokines tests over the years and, stopped, you know. Right. Like I do I do it for a few months, and then I realize I'm not doing anything with the information.

And I stopped doing it. And then it looks exciting again because we love numbers. But I have to admit, the in-cell the panel has been helpful. Yeah, yeah. So once again, no.

Yeah. Advertising. You know, we know no kickbacks and no commissions. Okay. So in addition to those one can look for interleukin six, interleukin 12 fecal Covid test for any remnants of the Covid which yeah, I wasn't that's another test I wasn't aware of.

Yeah. So test so if possible. So again it is not available everywhere. But if possible fecal testing for remnants. Because there are studies which show that debris of the virus can continue to stick in the GI cells for up to 59 days after the symptoms have subsided.

And now there are diverse studies from there. Some studies says that the presence of those broken out in feces inside the cells help build the immune system strength against the future exposure by affinity maturation.

And we all know that that is a process with the antigen is exposed to our, to follicular dendritic cell to our B and T cells, and they become more and more mature in attacking this antigen in the in the future.

And some studies say that this continuous presence of messenger as pieces of RNA are actually disrupting the local immune system and causing local inflammation, which then creates a lot of gi t related long Covid symptoms.

So far, the studies have not shown viable virus, but they have shown, in viable pieces of virus which may be immune circulating. This also could be a problem with the microbiome as well.

And that restoration would be useful as well as well. Yeah. So general approach to the Covid is basically two branches. One is to see if our immune system is dysregulated, and the other one is to see if the virus pieces are sitting somewhere, or a third one will be a combination of both.

And so whatever is the basic pathology, it's going to fall in these categories. Either the virus pieces are there or the immune system is dysregulated, or the both of these are present together.

And then based on that there are a few management approaches. For example. If immune system is dysregulated then that that dysregulation could be of multiple types and outcomes.

For example mast cell activation syndrome could be unmasked by this dysregulation. So a patient who actually has mixed mast cell activation syndrome and is not aware of it, they all of a sudden might have start having them CAS.

And that is an unmasking of the NCS. In some patient. There is even a triggering of the CAS as well. And the patient of M CAS were already the most patient.

They may get flare ups, so that is one possibility. Second, possibilities, as I discussed before, that may not be here in this, diagram is the inflammatory state of the monocytes and the blood clotting issues, which are also because of the auto antibodies.

And this is why I believe that this discourse of saying the spike protein goes around in the body causes an issue is not possible because spike proteins are picked up by the immune system.

It is the auto antibodies that are freely allowed to circulate in the body. And if they are in your typical antibodies, anti type antibodies, then they would act like spike proteins.

And they could behave as a moniker for spike protein like spike. So if it is an mix like signs and symptoms, then mix management should start. And the FCC protocol I would show you there the management approach is present.

If it is not that, if you suspect it is not mixed like behavior, then steroid and ivermectin with low dose naltrexone should become the first line. I have seen this to be repeatedly the gem of the prescription, the first line of prescription.

And think about it that steroids would keep the inflammation in control. The damage in control. I give it a meeting. There is a study. I can probably, look it up right now in front of you, and you might, find me googling it, but it's interesting.

In vitro study, ivermectin binds with spike protein and is to. I believe this is the one. So ivermectin dogs to the SARS-CoV-2 spike receptor binding domain attached to Ace2.

This is an in vitro study. And here they have these beautiful diagrams. So if you see here this is the ivermectin molecule. This is I believe ivermectin molecule docked with the SARS-CoV-2 spike receptor binding domain bound with Ace2.

So here this triangle is the viral spike protein. This arrow is the ivermectin this little thing. And the remaining this molecule is Ace2. So ivermectin disrupts the action of Ace2.

And this spike protein at least from this in vitro model. And if that is the case imagine if we have auto antibodies and you give ivermectin it is going to bind with that auto antibody in the same way and disrupted just like your disrupted spike protein.

This is such a beauty. Yeah. No that is beautiful. I mean that is because we see it work clinically. And it's so nice when you have a story that makes sense.

Correct? Correct. So, Eric, one of the Eric Read requirements I have from myself is that when I present something, I present that based on some mechanism, some, some known science.

All right. So that we can think about it. Yes. And of course, there are, doctors there are researchers who can hypothesize and then go find the solutions for that.

I am actually using someone's hypotheses and solution and presenting them. Yes. So steroid would keep inflammation under control. Ivermectin would help with if the spike protein is hanging around there would help with that.

If the antidote type, antibody or network hypothesis mechanism is occurring, ivermectin would help with that. We also know that ivermectin helps it with the nuclear factor K beta kappa B, and it's like changing beta disruption as well.

And then helps with the inflammation. So that would be there as well. And so and then finally I have written fluvoxamine here since then we have changed it to naltrexone.

So low dose naltrexone not naltrexone with low dose naltrexone has been working like magic. So what you do is you start with these three and then three, four weeks later you start tapering them.

Take steroid out first, then take ivermectin, then leave low dose naproxen for some time and remove that to yeah worst case we that we have people unload those naltrexone for years and with with excellent results.

So yeah. So this has really been great. It's whoever started this the naltrexone is used. Kudos to them. But that has really helped. Now if there is a possibility of some virions of broken pieces still hanging out somewhere, then ivermectin once again will help because it would keep binding with this spike proteins and keep disrupting the spike protein from, interfering with the other immune systems.

And then, of course, we've talked about vaccines as well. I think you and I talked about to vaccine anyone who's at risk, although nowadays the efficacy of vaccine has become an issue with Omicron.

Fortunately, Omicron itself is becoming milder too. But please speak with your doctor to figure out if vaccines are an important part. Now management, I'm just going to recap.

This is the first line on the I. T cover. This would be updated soon. So I am kind of, taking that part and presenting it here before it has become updated.

In a few days it will be. So the first line ready solid and low dose, 10 to 15 milligram. And you continue for two, three weeks. And then you taper it by two, three days of five milligram reduction.

Then another two three days or five milligram reduction. I usually try one week each. So starting with 15 for two three weeks, then one week for ten, then one week for five and then stop.

But if you have a tapering mechanism that you prefer and like, there is no hard science on the tapering. So you do what you feel comfortable. In addition to that ivermectin 0.2mg/kg body weight.

So it's not the ivermectin need for higher concentration. And because we are really not attacking the virus itself, it's just the binding with those disrupting autoantibodies or with the spike protein, if that is present somewhere.

And then low dose naltrexone begin with one milligram and then it can be escalated just to be very, very be in close contact with the patient to make sure that their side effects and this situation is managed.

This is the first line. Now before I go to the rest of this, what I will do is this. So there is a lot more here. What I will do is this for the remaining part of the discussion, I would then go here to say after that first line, it is possible to try fluvoxamine for neurological situations, and I have seen that in some people fluvoxamine can actually increase the neurological issues.

And if that is the case, we stop. Yeah. And we also know that fluvoxamine is not being given for psychological or psychiatric condition here. Instead, this is the sigma one antagonism that reduces inflammation in the brain.

You can think of flu work as a partner to ivermectin. Ivermectin cannot cross blood brain barrier. So that stays in the body. And fluvoxamine can go there and help with the inflammation.

Yeah. Then very interested in the dosing because I you know like for the treatment of acute Covid.

Treatment Options for Refractory Cases

They've been talking about 100mg twice a day which again many people can't tolerate. And I've often start people at 50mg. And I thought even 25 is enough for some.

And so it really looks like from the data there that the dosage for what we're trying to accomplish here can be much lower than that's what's used for treating depression.

Absolutely. Even a half of 25 milligram. Yeah. Even 12.5mg seems to be fine as well. Yeah. That that is that's clinic I've gone that way because people just couldn't tolerate it.

And I figured that it was just they picked the 100mg twice a day dose just because that's what is approved. Correct? Correct. We are correct. Yeah. So there are therapies here.

Then if you want you to see that macrophage activation syndrome. And how do we manage that. So here it is vitamin C omega 312 a certain melatonin. And then if you wanted to see how to help with the mix then that doctor Tina Pierce has been very good in putting together this part of the protocol that is here as well.

So my point is, after that first light therapy for the remaining part, you can actually see the management protocol here. You can refer it and you can help further.

Right right right right right. Yeah. So there's a lot of this will be updated in a few days with the first line showing up here. The first line that I just presented. Right.

Yes. I'm noticing I didn't see them forever up there. Okay. So Maravilla okay. But overall is something that, in my opinion that is in the extreme cases.

Okay. Number one, number two, doc has a larger set of side effects and so needs very good care from the physician. Number three Mira Mira could be expensive as well.

Oh yes it is. Yeah I think so. But overall is an option. Doctor use group doctor Bruce Patterson's group this whereby it that. But overall in all refractory long covid it works like magic for them.

But I haven't used it I haven't had a need to use this. But then I do not have a refractory long Covid person as well. Most of them recover with the remaining protocol.

So Morabito is a is an option. But I don't have it here and I think I will keep it as a side note that in refractory refractory. No, it does it just because of my practice.

What I'm on, you know, part of the referral list, but because, you know the nature of my practice, people look at the website, they see we treat chronic disease.

We tend to get the ones who are failing and and but overall it works well. Even then it works, but not quite as miraculously as advertised. It does work, but it takes a long time.

You know, we haven't. Yeah, I that's the thing. I yeah, I think one balancing. Thing that should be done. Is that what I see sometimes in these protocol discussion is to say no steroids, just Milagro.

And I think that is not entirely the right approach. Steroids has a broader, implications for the immune system. And because there is so much dysregulation.

But I alone is a very specific part. So overall help with steroids is necessary. Yeah. I think one of the things we run into is that, again, when you start getting into the very sensitive patients, there are many of them who can't tolerate even tiny doses of steroids, even one milligram that happens absolutely happen.

They just can't sleep. They get so agitate it. And so then we have to do other things. I'm also noticing while we're talking here is the, is that the atorvastatin?

You know, because, Doctor Patterson's group is in love with, pravastatin. And and we would be updating this as well. So Trevor Stratton is fine as well. Or the strategies.

No, no, I use both. I mean, some people feel a tour of a statin because it gets into the brain might be more useful. Yeah, it's just interesting. And just the dosing between 10 and 40mg, I mean, again, but yeah.

So, so, melatonin is very interesting as well. And once again, on the ramping up part, I would suggest that if the patient is refractory before moreover drug, there may be a an attempt for pulse dosing, stop things and then start again in a couple of weeks and see if that works.

If there does not, then of course Bravo is one more option. But this is the management approach. Yeah, yeah. No, no, I forgot about my apologies. I forgot about anosmia.

0.3mg/kg body weight, ivermectin for three days. In majority of the patients in those because, removed is gone. That's that's just a beautiful thing. I've seen it so many times now my own mother in law had in Austria, and she had it for weeks.

And I said, all right, take this. And within three days she was fine, Yeah. That, that that that is, you know, I, I love the things that you've seen work, especially in people with long term cases, because one of the things that was so difficult in the beginning, or still is, is that it is so many people who get acute Covid, you know, do so well that so many of the treatments you oh, I mean, it's only when I saw people who were really sick respond within a day or 2 to 2 ivermectin that I really, you know, truly believed it.

How well it worked, you know, because it's when you have self-limiting illnesses, it's, you know, many things look like they work. So it's always exciting to see that.

Yeah. This is this is I a little of a wonderful tour de force of like, how to approach long Covid and, you know, and I don't think we mentioned but, maybe you did, but in my experience.

Oh, I've treated pretty much vaccine injury vaccine reactions the same way as long Covid because I really do think it tends to be that spike protein activating the immune system.

And it's actually the same management. Yeah. Yeah, yeah. And I, I just wish that we could get, the powers that be to, to, acknowledge it and just so people so we could treat it so it wouldn't have to be as much of a disaster for people who have the problems.

You know, I think that it's, you know, we I said we need the vaccines for all people for certain. I mean, like, the death rates are just so dramatically different.

I mean, I don't think anybody I mean, it makes me crazy when people tell me that it didn't do any, didn't make a difference. If, you know, people in Israel, in New York, I mean, just the it's just so different, you know, but you're very correct.

And, one tragedy is that on one side, the good news is that long Covid is acknowledged and insurance can cover it. Vaccine toxicity or injury or whatever way you want to put it that is not covered.

No. And it's very difficult. The there's the the state the various database works really well if you had an allergic reaction, but if you had anything other than that, it's a very difficult to navigate and had to report.

And so people are getting vaccine injured and then they're having to one go through that miserable, journey of getting back to normal. And second, they have to pay out of their pocket to get all this.

Oh yeah. No, no, it's it's but that is that well, that's another story. Well, the economics of chronic illness is not nice. I, I am responsible for a lot of that because most of what we do is not covered by insurance.

You know, I mean, those of us who are in this field a long time, we often start off taking insurance, but along the way we start because, oh, we get massacred, and threatened by the insurance companies and Medicare, you know, because you, you, you, you do things that they don't approve us.

Right? So it it's a very it's yeah. Medicine is complex in America. The, the politics of it is another is a whole other world. But, but I just want to thank you.

This was an amazing, journey through, you know, just treating long Covid and understanding the etiology. I think that's where I would love to come back again and just, you know, go deep on these things because they illuminate the immune system.

And the more we treat chronic diseases, it is all about the immune response. And, how people hold their one of the, one of the things that we talk about on this program a lot is also the psychological component, and not in the sense that, you're sick because you're depressed and you think is one of the worst things we do to people is make them victim.

But the illness causes the chronic inflammation in the brain will flare, depression will cause, obsessive compulsive disorder and will cause anxiety to get worse.

I mean, all they're all self-protective mechanisms, you know, the body's trying to protect itself. And when you're constantly inflamed, you set off these, you know, just like the histamine response rate.

Self-protective mechanism. Correct. You'd be transient. I mean, I absolutely I think at some point you should come join me in my life show as well. And we talk a little about chronic diseases.

It's very, very useful for people to hear how to manage and approach it and what to expect. It will be a pleasure. But, you know, and I, I, I'm a great proponent of I said, well, I've, I've been doing this for a long time, but I learned to think differently about it after reading, you know, Dutch Navy and working with your Navy, but especially just reading his early work, about mitochondria and their response to, the to danger, because that seems to be, you know, so much is when the body perceives danger, it, it it hardens its defenses, you know, those shoulders come up, you know, mean and absolutely.

And we we we get into it, we get into trouble. Those cell membranes, begin to, get stiff. And it's just what happens, and it's a good protective mode move, but not one to stay in.

That is a problem. So I just want to again, thank you so much. This was an amazingly deep, and informative

lecture. And, you know, for all the patients out there, you know, it's just important to remember that if you can educate your physicians and, you know, let them listen to this, watch doctor, doctor, you know, movies, series.

I mean, you can I can't tell you. I mean, like, I don't get to watch enough of that. But every time I do, it's like I go, oh my God, so much more immunology details than I thought I knew.

And it just a whole other pieces that open up and that is what is missing because, you know, no matter how well you were taught immunology, if it was more than five years ago, you are already so out of date, and not understanding.

And I think that's why, you know, physicians, if there's one thing they should be constantly going back to is learning, is relearning immunology, because it gives you the tools to think absolutely about what you're doing.

And, and your, your, your programs, you know, there for the layperson. But I find them amazingly educational. So I just want to thank you so much. Thank you very much.

Thank you very much for having me. Thank you for discussing this. I think we are, let's have a series of going deeper into various etiologies and pathologies and, meet again, I we will.

Okay. Thank you so much. Thank you. Let's. Bye bye for now.