

Leo Galland, MD: Treating PASC (Post-Acute Sequelae of COVID): How to Address Long-Hauler's Syndrome Effectively

Interview by Dick Benson

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Leo Galland, MD, a board-certified internist, Dr. Galland was educated at Harvard University and the New York University School of Medicine and trained in internal medicine at the New York University-Bellevue Medical Center. He has received international recognition for developing innovative nutritional therapies to treat autoimmune, inflammatory, allergic, infectious and gastrointestinal disorders and has described his work in numerous scientific articles and textbook chapters.

Today we want to talk today about the presentation you are going to give at the Environmental Healthcare Symposium (EHS).

Dr Galland: Yes, my presentation will focus on COVID, from the perspective of not only treatment, but prevention because my view is that the prevention of long COVID in people who are exposed to the virus should be a major public health priority.

Integrative Medicine: A Clinician's Journal (IMCJ): Is that because of the issues that people have before they are exposed to COVID or is it because of what COVID does?

Dr Galland: The reason I think it should be a priority is that there are reports that people who are hospitalized with COVID, 50% to 70% of them, take months to recover. And those people who are not hospitalized, in about 30% of them it takes months to get better. There are some patients who are pretty sick and disabled a year, a year and a half later. This is a huge number of people on whom the late effects of COVID had an impact, and there's been really very little research on not only the treatment and reversal of COVID, but prevention at the time of acute infection.

My own experience with the protocol I had worked out for treating COVID is that fewer than 2% of the patients that I see have gone on to have what we'll call long COVID. I think we know enough about the biology or the physiology of long COVID that it's possible to have rational treatments, preventive measures as well as treatments, for the disease once it occurs.

IMCJ: Is the impact of long COVID consistent across patient types?

Dr Galland: There's a great deal of variability and different organ systems are involved; there are different combinations. There's not a clear relationship between how sick a person is and the likelihood of long COVID. So that's one of the things that I'll be addressing in the presentation; it'll probably be the focus of my presentation.

Here's what's known about biological changes in the bodies of people who get COVID-19 when they're recovering. These changes can be identified even in people who are not very sick. They fall into a few categories.

Number one are disturbances in circulatory function, which can be understood based upon the destruction of the enzyme that is the cellular receptor for the virus. The virus enters cells by attaching to an enzyme, ACE2, on the outside of the cell membrane. In the course of viral attachment and viral cell entry, ACE2 is destroyed, and ACE2 plays a vitally important role in circulatory function. It also has anti-inflammatory effects, antithrombotic effects. Almost all the acute complications of COVID-19 can be traced to an ACE2 deficiency that results from the viral infection. Now, there's evidence that this kind of deficiency persists into the recovery stage—and I'll review some of the studies, which found that even healthy young people who are recovering from COVID-19 have disturbances in circulatory function—which are totally consistent with the effects of ACE2 deficiency.

The second thing that we know about the pathophysiology of recovery from COVID is that there is an ongoing state of inflammation in the body. The immune system is not returned to its pre-COVID state, and the inflammation is actually fairly complex. It involves abnormalities in T cell function. There is also a high rate of autoantibody formation. That's related to B cells, and the auto antibodies that develop are usually functional. That is, they have physiologic effects that are measurable. They're not just there as a lab finding. Included in those autoantibodies are autoantibodies directed against ACE2, which were recently demonstrated by a group of researchers at the University of Arkansas. Those autoantibodies are also functional and were shown to have an impact on ACE2 function.

There are 2 mechanisms by which ACE2 is impaired. One is a direct attack on ACE2 by the virus and the other is the formation of anti-ACE2 autoantibodies. That's a delayed effect, and it correlates with the development of antibodies against the spike protein.

The third thing that we know is that there's mitochondrial distress that's probably dysfunction, but

definitely distress, that can be measured in people who are recovering from COVID.

The fourth, or maybe the first actually, is that there are disturbances in the oral and gut microbiomes, and these changes have mostly been measured in people who have been hospitalized, so we can't be certain that they're not in part a result of hospitalization. But the changes involve a loss of diversity and anti-inflammatory organisms and an increase in the levels of inflammation-inducing microbes. A recent study showed that probiotics actually can decrease the likelihood of the development of long COVID.

We've got those 4 components, and I think those 4 components are probably to some extent universal. That is part of what happens after COVID-19, and it may be severe enough to contribute to the long COVID syndrome.

In addition to that, damage to organs occurs, the brain and the lungs being the most important of these. Damage in the brain actually involves the loss of gray matter, which is quite common and is associated with impairment in higher cognitive functions even in people who haven't been very sick. The other thing that may occur is damage to the autonomic nervous system. That's another neurologic complication producing a syndrome called POTS—postural orthostatic tachycardia syndrome—which is now pretty well known because of COVID-19.

The damage to the lungs most of the time is not that obvious. If people get pneumonia, there'll be evidence of COVID pneumonia. It'll usually resolve. The long-term changes in the lungs involve 2 things. One is the thickening of the lining of the alveoli, of the air sacs, which interferes or slows down oxygen transport. This is called interstitial lung disease. This will produce shortness of breath and usually a drop in oxygen saturation with activity.

The other is the loss of blood vessels. So again, we're back to the importance of the circulation. There is a loss of blood vessels in the lungs. There is a group in Boston that's developed a computer program for analyzing CT scans to demonstrate this. Just looking at a CT scan, it may be read as normal, but using their computer algorithm, if the scan used thin enough sections, you'll be able to determine that there's just a loss of pulmonary circulation. Sometimes there are other things. There's metabolic damage that can occur. Diabetes and high blood pressure occur as a complication of the post-COVID syndrome at a rate that ... In other words, people who have had COVID are 2 to 3 times as likely to develop diabetes or high blood pressure over the subsequent 6 months as people who didn't have COVID. Kidney damage may occur. These are not the most common complications, but they're just examples of the tremendous variety of types of damage and dysfunction that this virus can leave in a person's body.

IMCJ: How do you treat the condition?

Dr Galland: My approach to the treatment of acute COVID and the prevention of the complications of acute COVID

dovetails really very tightly with the prevention and even the treatment of long COVID. Although, depending on the type of damage somebody has, you may need to perform other measures to treat long COVID. But it's an increasingly systematic approach that I take, which starts with recognizing the 4 things that I said are together kind of universal: the oral and gut dysbiosis, preventing, reversing and treating that with some antimicrobial natural products, some probiotics and prebiotics; restoring ACE2 activity, primarily through diet, a high polyphenol diet, which also shapes the gut microbiome and specific supplements, of which the major ones are vitamin D, Resveratrol and curcumin. Those have been part of my treatment for acute COVID since February 2020. I strongly suspect that that has something to do with the very low incidence of long COVID in the patients who I've treated.

The third component would be restoring mitochondrial function. For that, I look for evidence of mitochondrial dysfunction, and there are several things that can be helpful for that. Dietary supplements, which include Coenzyme Q10, the most studied supplement for mitochondrial restoration, and sometimes niacin and NAC to support mitochondrial complex 1 specifically. Then, as far as rebalancing inflammation, I may add some specific nutrients or herbs to help up with that. Omega-3s, I think are very important, possibly combined with alpha-lipoic acid for their anti-inflammatory and neuroprotective effects. And Andrographis, which is a traditional Chinese medicine herb, for its immune modulating effects.

The bottom line is that it is possible to have a comprehensive, systematic approach to treating and reversing long COVID. To me, it makes the most sense to start by preventing it with measures that conserve or restore ACE2 function, balance the inflammatory response at the beginning when someone is actually sick with COVID, and definitely preventing or reversing the oral and gut dysbiosis, which seems to be very common with this disease.

We also know that vaccination may have no impact on the risk of long COVID. Or if it does, there may be a 50% reduction in its effects. Well, going from 30% to 15%, we'll leave millions and millions of people with long COVID. I have also seen a study indicating a 50% reduction and another study indicating no reduction, no impact with the vaccines.

The best case scenario with the Omicron strain would be that it's highly infectious but has very low virulence. That would be like a natural vaccine. If there's very low virulence, but high infectivity, maybe this will spread throughout the world and induce immunity at a very low cost. That, of course, is a dream. That's the dream solution that this virus somehow evolves into essentially its own vaccine. But there's no way of knowing whether that's really happening at this time.

IMCJ: Thank you very much for your time. I am sure you will have many updates between now and your presentation at EHS.