Commentary

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Post COVID Symptoms Inducing Postural Tachycardia Syndrome and Related Autonomic Conditions

Paul Lelorier*

Department of Medicine, Section of Cardiology, Louisiana State University Health Sciences Center, New Orleans School of Medicine, New Orleans, USA

***Corresponding author:** Paul Lelorier, Department of Medicine, Section of Cardiology, Louisiana State University Health Sciences Center, NewOrleans School of Medicine, New Orleans, USA; E-mail: plelor@lsuhsc.edu

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Description

Although the phenomenon described as "long-COVID" is well recognized, due to variable case definitions, lack of standardized diagnostic criteria and lack of objective findings, the incidence and prevalence is unknown. However, many patients complain of symptoms of dysautonomia, although the term is thrown around loosely and can include everything from an actual autonomic neuropathy as demonstrated by formal, standardized testing, to vague, multisystem complaints, without any demonstrable findings of dysautonomia, and has also included post-COVID Postural Tachycardia Syndrome (POTS), although the latter is not always found to be neuropathic in nature.

In the study by Aditi-Verma et al., a new insight into the potential mechanism for post-COVID autonomic symptoms is described. Louisiana State University, through its autonomic laboratory at University Medical Center New Orleans, is a regional referral center for dysautonomias, syncope and postural tachycardia syndrome. As such, it performs a substantial number of autonomic function tests, many for POTS. These labs are still unabundant around the United States. After the association of COVID with autonomic complaints and the previous observation of an association between some patients with POTS and the presence of small fiber neuropathies, the authors decided to perform an analysis of patients in their database who had undergone autonomic testing in 2019, including Quantitative Sudomotor Axon Reflex Test (QSART), which has been demonstrated to be a useful tool for screening for small fiber neuropathies. This database was cross-indexed with patients with a confirmed SARS-CoV-2 infection and who reported worsening autonomic symptoms post-infection. In addition, patients with SARS-CoV-2 infection without prior testing but with autonomic complaints were also approached. All patients needed to have negative nerve conduction and electromyography studies for inclusion. Also, no other cause of dysautonomia could be present.

Six participants were enrolled and divided into two groups. The first group had 4 volunteers reporting worsened autonomic symptoms and had pre- and post-COVID autonomic testing performed. The second group of two reported new autonomic symptoms post COVID and were tested one year after their infection. All participants were female, white and between 21 and 37 years of age. Importantly, although all complained of brain fog, cognitive testing was normal.

Abnormal QSART was demonstrated in all patients. Amongst the patients that had pre-COVID testing, QSART had worsened in 3 out of 4. In all 3 patients in whom skin biopsies were performed, there was decreased intraepidermal small fiber density consistent with a small fiber neuropathy.

Neurocardiogenic syncope was elicited during the tilt testing portion of the autonomic test in one patient with worsened symptoms and blood pressure responses to Valsalva (usually in terms of decreased pulse pressure during phase II) were elicited in 2 out of 4 patients in this group. Parasympathetic indices were largely unaffected.

Although this study is small, it does provide a "before and after" look at changes in autonomic testing in a population that had sufficient dysautonomia symptoms to justify having undergone autonomic testing in 2019. Although this is not necessarily reflective of the general population, it does provide the advantage that every patient served as their own control. This study is important because it does highlight the involvement of small fiber neuropathy in many patients with post COVID autonomic symptoms. This appears to be the first such study showing changes in autonomic function from a known baseline post COVID. This has been seen as vindication for this large group of patients who have been frustrated by a lack of explanation for their symptoms as there are few other ways of objectively diagnosing small fiber neuropathy.

The mechanism by which SAR-CoV-2 causes small fiber neuropathy remains unclear. The development of small fiber neuropathy does not appear to be correlated with severity of infection. Viral entry into peripheral neurons *via* the angiotensin converting enzyme II receptors is one proposed mechanism as well as a clinical variant of Guillain-Barre syndrome with more autonomic than motor manifestations. Additionally, cytokine-mediated small fiber damage has been described.

In our study, 50% of patients had hyperadrenergic responses to tilt as evidenced by norepinephrine levels in addition to

abnormal QSARTs suggesting a mixed-type mechanism for Postural Tachycardia syndrome. The possible explanations for this are legion and beyond the scope of this article. Suffice it to say that since various mechanisms are at play, it is likely that there may not be a single "silver bullet" solution to the problem. Since the condition is heterogenous, this may explain the often-conflicting results in the limited clinical trials available as well as the frustrating lack of universal clinical response to any one treatment modality. One such example is the varied response of long-COVID POTS to intravenous gamma globulin between trials and between patients. While such an approach may work very well for mechanisms involving anti-receptor antibodies, it may be less effective if the mechanism involves a small fiber neuropathy.

Where do we go from here? Standard definitions of what long-COVID is and is not, are required. Inclusion into many post-COVID POTS trial requires little other than clinical criteria for POTS and antibodies to COVID. Clearly, this is a setup for heterogenous study populations. Just like screws, all COVID long-haulers are not the same. Sometimes a flat head screwdriver just won't do the trick.