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## CLINICAL VIGNETTE

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# POTS Secondary to COVID-19

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### Introduction

The COVID-19 pandemic caused by SARS-CoV-2 has produced significant death and devastation. While we first focused on treating acute illness, we now know that patients may experience ongoing symptoms for months after initial infection. These “long- COVID” symptoms can include fatigue, dyspnea, joint pain, or chest pain.<sup>1-3</sup> Reported here is a case of postural tachycardia syndrome (POTS) secondary to COVID-19. Other cases of post-COVID POTS have been reported in the literature in recent months.<sup>4-7</sup> This case report adds to our growing understanding of this emerging virus and its potential sequelae.

### Case

A 25-year-old healthy female with epilepsy returned from travel to Europe in March 2020. Five days later, she developed cough, fever, and flu-like symptoms and tested positive for COVID-19. Her initial symptoms lasted for 2.5 weeks and included persistently high fever, cough, and shortness of breath.

Around three weeks after symptom onset, she presented to the emergency room with ongoing cough, shortness of breath, and tachycardia to 150s-160s associated with any activity. Lab work including CBC, BMP, troponin, lactate, and procalcitonin as well as EKG and CXR were unremarkable. Her oxygen saturation was 99% at rest and 96% with ambulation. She was discharged home.

One week later, the patient was admitted to the hospital for left-sided chest pain, tachycardia, and prolonged fevers thought to be secondary to COVID-19. Repeat labs and CXR were unremarkable. D-dimer was 371, but bilateral lower extremity dopplers were negative for DVT, CTA chest was negative for PE, and TTE was normal. Her COVID-19 PCR remained positive. She was monitored on telemetry with no arrhythmias and discharged home after two days. No other new cause of her fevers or symptoms were found.

By late April 2020, five weeks after symptom onset, she continued to have dyspnea on exertion, tachycardia, and lightheadedness with standing, increased heart rate to the 150s with walking, ongoing chest pain, and fatigue. She was seen in primary care and vitals were notable for negative orthostatic blood pressures but a rise in heart rate from 81 lying down to 90 sitting and 133 standing. Her exam was otherwise normal. Labs including CBC, BMP, LFT, ESR, CRP, D-dimer, BNP, and troponin were within normal limits. Her EKG showed

atrial tachycardia with a heart rate of 134 bpm. She had a cardiac monitor showing sinus rhythm with rare supraventricular and ventricular ectopic beats but no sustained arrhythmias. She was diagnosed with POTS disease secondary to COVID-19.

During the summer of 2020, the patient continued to have tachycardia with heart rates of 130-140 during activity. She was started on metoprolol with gradual increase in her dose. She had physical therapy and worked on increasing her exercise tolerance at home. She had ongoing fatigue, chest pain, and lightheadedness, but these slowly improved. At six months after her initial illness, she had residual but much improved symptoms. Her heart rate remained in the normal range while standing but increased with activity. Soon after, she was able to return to work.

### Discussion

POTS is defined by an exaggerated heart rate increase in response to postural change. The criteria for diagnosis are a sustained heart rate increase greater than 30 beats per minute on standing with no orthostatic hypotension. Typical symptoms include lightheadedness, orthostatic intolerance, palpitations, headache, poor concentration, and fatigue while standing. POTS is most common in young women and is often co-morbid with migraines, Irritable Bowel Disease, Ehler’s-Danlos, chronic fatigue syndrome, and fibromyalgia. It is often a sequelae of viral illness, with 28-41% of patients reporting onset after a viral prodrome, though the pathogenesis of this is unclear.<sup>8,9</sup>

This patient’s presentation was consistent with post-COVID POTS given her symptoms and the objective findings of a rise in her heart rate to the 130s with standing, an increase of 50 bpm from lying down, and negative orthostatics in the setting of recent COVID-19 infection. Other cases of post-COVID POTS with similar symptoms have been reported.<sup>4-7</sup>

The mechanism by which COVID-19 causes POTS disease remains unknown, though chronic inflammation or autoimmunity may play a role.<sup>5</sup> Various mechanisms of pathogenesis have been proposed and further research in the area is needed.<sup>7,10</sup>

The treatment for POTS is typically exercise or physical counter-maneuvers, increased oral fluid intake, and a high salt

diet. Medications like fludrocortisone and midodrine can be used to raise blood pressure and improve orthostatic symptoms. Beta-blockers can also be used to help heart rate.

The prognosis for non-COVID POTS varies as it may be a self-limited or relapsing-remitting course. Most patients are symptomatically improved at one year and at that point 30% no longer meet diagnostic criteria.<sup>9</sup> This patient's gradual improvement over several months suggests that post-COVID POTS may follow a similar course and eventually resolve. Other cases have similarly involved persistent symptoms, with improvement at six to eight months.<sup>7</sup>

## Conclusion

This case provides an example of post-COVID POTS and shows that POTS and other autonomic disorders can be complications of COVID-19 infection in previously healthy, non-hospitalized patients. As we see more patients affected by post-COVID symptoms, particularly after the recent winter surge, it is important for us to recognize potential complications from the virus so that we can treat our patients appropriately.

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