

CLINICAL VIGNETTE

Postural Orthostatic Tachycardia Syndrome in a Young Female

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A 21-year-old woman with schizoaffective disorder was taken to the Emergency Department by paramedics. That morning, she became dizzy and lost consciousness when she stood up from a seated position prompting the 911 call. She reported prior episodes of lightheadedness for months, as well as palpitations however denied chest pain, shortness of breath, seizure activity, focal neurological deficits, or infectious symptoms. She was taking lamotrigine 100mg in the morning and quetiapine 400mg twice daily. She reported her lightheaded episodes started before she started the medication.

Physical exam was pertinent for resting tachycardia ranging from 120 to 160 beats per minute (BPM), orthostatic hypotension, and a normal cardiovascular exam, without evidence of structural heart disease. The patient's laboratory results included hemoglobin of 13.4 g/dL and a white blood count of 11,700 cells/uL, which normalized without antibiotics. The patient's electrolytes, troponin, procalcitonin, thyroid stimulating hormone, morning cortisol, erythrocyte sedimentation rate, and C-reactive protein were within normal limits. Electrocardiography demonstrated sinus tachycardia, normal axis, a shortened PR interval of 115 milliseconds, and normal morphology, without signs of ischemia.

During her first two days of hospitalization, she received six liters of intravenous fluids. Despite resolution of her orthostatic hypotension, her resting heart rate remained in the 120/min, increasing to 140 to 150 BPM with standing, associated with lightheadedness. Echocardiogram was normal. Review of records, revealed tachycardia in the mid-120 BPM during prior clinic appointments six months before the hospitalization. With postural intolerance that was refractory to fluid resuscitation, she was diagnosed with postural orthostatic tachycardia syndrome (POTS). Orthostatic tachycardia caused by quetiapine was also entertained. Cardiology agreed with the diagnosis of POTS. Although quetiapine is a cause of tachycardia and orthostatic hypotension, this patient was tachycardic prior to starting quetiapine, supporting the diagnosis of POTS. The patient was discharged home with instructions to increase fluid and salt intake, wear compression stockings, and make slow transitions when standing. The patient was also prescribed fludrocortisone 0.1 mg daily, and suggested to discuss the option of decreasing her daily quetiapine dosage with her psychiatrist. She followed up with the Cardiac Arrhythmia Center for Autonomic Testing. She has normal sweat responses in the forearm, proximal and distal leg and foot with no abnormalities

identified in sudomotor axon flex. Heart rate response to deep breathing had a mean difference of 7.9 beats/min, which is below age-normative limits. Tilt table test found a normal maximal systolic blood pressure drop of 16 mmHg (normal < 30mmHg), with tachycardia response of 50.6 beats/min, which indicates orthostatic tachycardia. The patient also reported lightheadedness towards the end if the upright tilt. Her Valsalva ratio was 1.766, which is within age-normative values. Adrenergic sensitivity analysis revealed no significant impairments.

Her composite autonomic symptom score (CASS) was as follows:

Cardiovagal: 1
Adrenergic: 0
Sudomotor: 0

A CASS score of 0 is normal, 1-3 is mild generalized autonomic impairment (GAI), 4-6 is moderate GAI, and 7-10 is severe GAI. The conclusion was that the patient has mild GAI within the cardiovagal system. The tachycardia response was in excess of the blood pressure drop, consistent with POTS.

Cardiovascular deconditioning is observed in all patients with POTS.¹ It is hypothesized that many pathophysiologic mechanisms can contribute to susceptibility to POTS, which include: dysregulation of norepinephrine release, excessive sympathetic discharge, abnormal mast cell activation, and problems in the renin-angiotensin-aldosterone system.^{2,3} The criteria for a POTS diagnosis are: a heart rate increase of ≥ 30 BPM within 10 minutes of standing and absence of other causes of orthostatic symptoms.¹ The current treatment of POTS includes specialized exercise programs, which begin with activities in horizontal positions with progression to upright exercise, e.g., recumbent biking, rowing, and/or swimming laps or kicking laps with a kickboard to address underlying cardiovascular deconditioning.¹ More research is needed to determine the optimal management of patients with POTS because results from efficacy studies are heterogeneous.⁴ Patients that are diagnosed with POTS are advised to increase salt and fluid intake. Use mineralocorticoid and adrenoceptor agonists can also be considered.

Competing Interests

The authors have no competing interests to declare.

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