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A YOUNG FEMALE WHO DEVELOPS TACHYCARDIA AND ORTHOSTATIC INTOLERANCE FOLLOWING A RECENT INFECTION

Michael A. Valentino, MD, PhD

Case Presentation

The patient is a 20-year-old female with a history of asthma and anxiety with panic attacks who presented with palpitations and lightheadedness/pre-syncope. The morning of admission, she was attending a seminar and experienced an acute onset of palpitations. The palpitations started while she was seated and worsened upon standing. They were associated with lightheadedness, shortness of breath, and chest tightness. A nurse attending the seminar recommended that she go to the emergency room.

On presentation to the emergency department (ED) her vital signs were: Temperature: 99.2°F, Blood pressure (BP): 140/98; Heart rate (HR): 140; Respiratory rate (RR): 16; Oxygen saturation: 100% on room air. She reported that she had experienced prior panic attacks but that this episode was persistent and significantly more intense in comparison. She also reported that a few days prior to presentation she had completed a prolonged course of antibiotics for tonsillitis. Her only medications were oral contraceptive pills (OCPs) and dextroamphetamine/amphetamine (Adderall®). She reported taking her Adderall® as prescribed, most recently on the day prior to presentation. She denied other stimulant or illicit drug abuse. Her family history was significant for anti-phospholipid antibody syndrome (APS) in her mother.

Investigations

On physical exam, she was in no acute distress. She had moist mucous membranes and no conjunctival pallor. Her cardiac exam was remarkable only for tachycardia. The rest of her exam was unremarkable. Orthostatic vitals were checked revealing a BP of 130/75 and HR of 90-110 beats per minute (bpm) while sitting and a BP of 145/97 and HR of 130-150 bpm while standing, with associated lightheadedness and shortness of breath.

Differential Diagnosis

The differential diagnosis included cardiac tachyarrhythmia (e.g. supraventricular tachycardia, atrial flutter/fibrillation), dehydration, anemia, hyperthyroidism, hypoglycemia, pharmacologic stimulant abuse, infection, and pulmonary embolism. An electrocardiogram (ECG) revealed sinus tachycardia (Figure 1) with no significant ST-T wave changes. Labs drawn on admission revealed normal blood counts and chemistries, and her thyroid panel revealed an elevated thyroid stimulating hormone but normal free T3 and free T4 levels. A urine drug screen was positive for amphetamines, consistent with her reported use of Adderall® the day prior to presentation. Given the acuity of onset of her symptoms, her family history of APS, and her OCP use there was a high suspicion for pulmonary

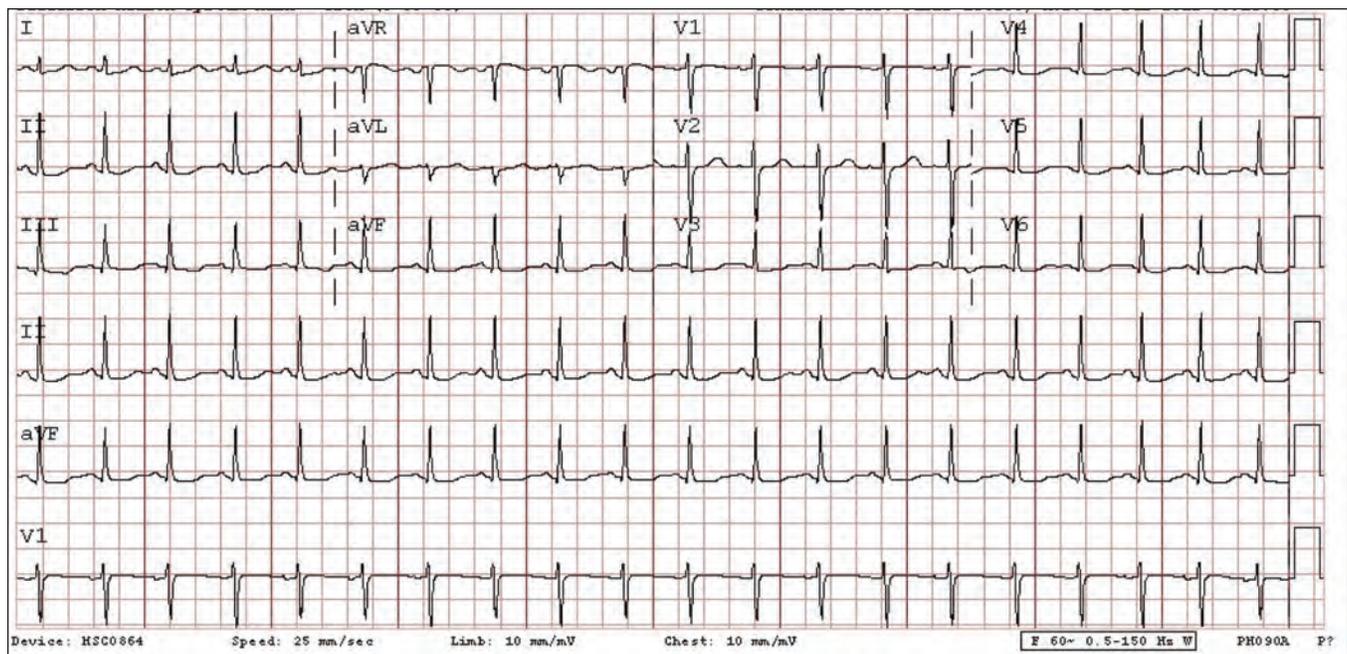


Figure 1. Patient's admission ECG

embolism, which was subsequently ruled out with a ventilation/perfusion scan. She received two 1-liter saline boluses in the ED for suspected dehydration, and she was admitted to the general medicine service.

The next morning the patient continued to exhibit orthostatic intolerance with significant tachycardia upon standing and ambulating, associated with shortness of breath and lightheadedness/dizziness. Since the initial workup was negative and her orthostatic tachycardia continued, the Electrophysiology service was consulted. Their differential diagnosis included: amphetamine-induced tachycardia, other exogenous stimulants, and inappropriate sinus tachycardia. However, given the patient's orthostatic tachycardia with normal BP, her age, gender, and recent infection, the most likely diagnosis was postural orthostatic tachycardia syndrome (POTS).

Outcome and Follow-up

On the second day of the patient's admission, her tachycardia improved. Her HR while ambulating peaked in the 110s associated with only mild shortness of breath. The decision was made not to treat with beta-blockers or calcium channel blockers since she remained hemodynamically stable and only minimally symptomatic. She was instructed to discontinue the Adderall® and avoid any other stimulants, including caffeine. She was to follow-up with Electrophysiology at Thomas Jefferson University Hospital and to schedule an outpatient tilt table test.

Discussion

Orthostatic intolerance is defined as cerebral hypoperfusion or sympathetic activation while standing, relieved by recumbency. POTS is a clinical syndrome characterized by orthostatic intolerance without orthostatic hypotension.¹ The accepted definition of POTS is orthostatic intolerance with a rise in HR of >30 bpm within 10 minutes of head-up tilt or standing, or a HR of >120 bpm while upright, without orthostatic hypotension.²

The majority of patients with POTS are young females, with an average age of 30 years.³ The most common symptoms are those related to cerebral hypoperfusion and sympathetic activation.¹ Fatigue is also a common symptom, and many patients have poor exercise tolerance.¹ While most patients cannot identify an inciting event, some patients recall an antecedent viral illness, while others associate the onset of symptoms with recent surgery or trauma.^{1,4}

Several pathophysiologic mechanisms have been proposed to underlie POTS. About 50% of patients with POTS have regional autonomic denervation, typically in the lower limbs. Sympathetic denervation of the lower extremities, as demonstrated by a quantitative sudomotor axon reflex test (QSART), thermoregulatory sweat test, and impaired norepinephrine spillover in the lower extremities in response to various stimuli, can cause loss of

vasomotor tone and pooling of blood in the legs.^{3,5} This can cause a decrease in preload upon standing and a reflexive increase in sympathetic stimulation of the heart in order to maintain cardiac output. The cause of this peripheral sympathetic denervation is still unclear, but ganglionic ($\alpha 3$) acetylcholine receptor (AChR) antibodies have been demonstrated in ~15% of patients with POTS indicating a possible autoimmune mechanism.³

Another subset of POTS is characterized by a hyperadrenergic state with elevated plasma norepinephrine levels (>600 pg/mL) and a rise in BP on standing.⁴ In a recent study, 29% of patients with POTS had elevated plasma levels of norepinephrine upon standing, and these patients benefited the most from β -blockade.³

In some patients, hypovolemia may also contribute to the manifestation of POTS. Dysfunction of the renin-angiotensin-aldosterone system has been implicated as a cause of the hypovolemia observed in some patients with POTS, as these patients lack a compensatory increase in circulating renin and aldosterone despite low plasma volume.^{6,7}

Several investigations should be made to establish the diagnosis of POTS including: orthostatic vitals (head-up tilt or standing), QSART response, thermoregulatory sweat test, supine and standing norepinephrine measurements, and 24-hour urine sodium excretion (surrogate marker for plasma volume).³ Pharmacologic treatments for POTS are aimed at increasing plasma volume (fludrocortisone, erythropoietin), enhancing venous return to the heart (midodrine, octreotide), or counteracting a hyperadrenergic state (β -blockers, clonidine, pyridostigmine, disopyramide).^{3,4} Nonpharmacologic treatments for POTS include high salt intake, water repletion, small frequent meals, regular exercise, and compression stockings. Typically, treatment is tailored toward the suspected underlying mechanism determined from autonomic testing.

Key Points

POTS is a disorder that is most common in young females and characterized by orthostatic intolerance without a decline in blood pressure. The syndrome may have several etiologies, which include peripheral denervation with lower extremity pooling of blood, a hyperadrenergic state, and low plasma volume. In a significant number of patients, the symptoms of POTS are manifested shortly after an infection, as occurred in our patient's case, or following other stressful events such as surgery or trauma. Initial work-up includes ruling out common etiologies for tachycardia, including: cardiac tachyarrhythmia, anemia, stimulant abuse, dehydration, pulmonary embolism, and hyperthyroidism. The patient should have orthostatic vitals measured or undergo a formal tilt table test. An increase in HR of >30 bpm or a HR of >120 bpm upon standing, without orthostatic hypotension, correlates with a diagnosis of POTS. The patient should then undergo autonomic testing as outlined above to determine the etiology of the patient's orthostatic intolerance and determine the most appropriate therapy.

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