Case report

Postural orthostatic tachycardia syndrome in primary care: diagnosis, treatment and a case of African-American man presenting with POTS

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SUMMARY

Postural orthostatic tachycardia syndrome (POTS) is a heterogeneous autonomic disorder characterised by orthostatic intolerance and a rise in heart rate by at least 30 bpm or an absolute heart rate value of at least 120 bpm within 10 min of standing or during a tilt table test. Overwhelmingly, POTS affects young Caucasian women, which can lead physicians to miss the diagnosis in men or non-white patients. We describe a case of 29-year-old African-American man who developed lightheadedness, generalised weakness, tachycardia and palpitations and was subsequently diagnosed with POTS. We review its clinical features, differential diagnosis, pathophysiology and treatment options. We also emphasise that POTS should be considered as a differential diagnosis in any patient presenting with typical clinical features, who may not be in the usual demographics of the disorder.

BACKGROUND

The autonomic nervous system (ANS) is vital for regulating physiologic processes, including blood pressure, heart rate, digestion, cerebral blood flow and maintaining cardiovascular function during an upright posture.

Under normal circumstances, approximately 500–1000 mL of blood pools in the lower extremities and splanchnic circulation, which in healthy individuals results, via an increase in sympathetic outflow and other compensatory mechanisms, in heart rate elevation by 10–20 bpm, along with a small drop in systolic blood pressure (5–10 mm Hg), and a rise in diastolic blood pressure (5–10 mm Hg)—all necessary for maintaining cardiac and cerebral perfusion.^{1 2}

Postural orthostatic tachycardia syndrome (POTS) is a heterogeneous autonomic disorder characterised by orthostatic intolerance and a rise in heart rate by at least 30 bpm or an absolute heart rate value of at least 120 bpm within 10 min of standing or during a tilt table test. As POTS is a disorder of the orthostatic intolerance, the normal compensatory mechanisms involved in maintaining an upright posture are compromised, which results in symptoms of presyncope, syncope, postural tachycardia, dizziness, exercise intolerance and fatigue.

This article reviews pathophysiology, clinical features, diagnosis and treatment of POTS and discusses a case of an African American man

who presented with clinical features and objective evidence of POTS, but whose diagnosis was questioned and delayed because he was viewed as outside of the typical demographics of patients with POTS.

CASE PRESENTATION

A 29-year-old African-American male medical student with a medical history of mild hypertension developed lightheadedness, generalised weakness, tachycardia and palpitations. He also noted persistent headaches, fatigue and transient chest pain over the past 2 months, which he attributed to his intense schedule at school and becoming a new father. On physical exam in the emergency room, his supine blood pressure was 137/93 mm Hg, with a heart rate of 102 bpm, but the orthostatic vital signs were not obtained. The diagnostic investigation included EKG that confirmed sinus tachycardia, unremarkable chest X-ray and routine blood tests and drug screen that were all within normal limits. Given the unremarkable workup, a decision was made to discharge the patient home with a prescription for lisinopril–hydrochlorothiazide 10–12.5 mg daily and a follow-up with a cardiologist on outpatient basis.

The patient attempted to take lisinopril–hydrochlorothiazide, which only resulted in worsening of his symptoms. Subsequently, he became bedridden with fatigue, palpitations and lightheadedness resulting in significant impairment even in the most basic activities of daily living, such as going to the restroom, brushing teeth or bathing. As his cardiologist could not determine the cause of his significant physical disability, he was referred to psychiatry for a suspected generalised anxiety or conversion disorder, which was not present, according to the psychiatrist. Subsequently, following a presyncopal episode, his blood pressure medication was discontinued, and his orthostatic vital signs without blood pressure medication at that time revealed a supine blood pressure of 70/55 mm Hg with a heart rate of 55 bpm and an upright blood pressure 140/80 mm Hg with heart rate of 95 bpm after standing up for 3 min.

Further diagnostic workup, which is summarised in [table 1](#), consisted of ruling out anaemia, thyroid disease, cardiac arrhythmia, pheochromocytoma, Addison's disease and acute Lyme infection. A 30-day cardiac monitor demonstrated episodic

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Table 1 Diagnostic investigations

Test	Result
Blood and urine tests	
Complete blood cell count	–
Comprehensive metabolic panel	–
Thyroid function tests	–
ANA, ESR, CRP	–
Vitamin B ₁₂	–
Lyme western blot IgM/IgG	–
Ganglionic AchR antibody	–
Morning cortisol	–
Urine toxicology screen	–
24-hour urine catecholamines	–
24-hour urine metanephrines	–
Cardiac tests	
Orthostatic vital signs	+ (Postural tachycardia, orthostatic hypertension)
EKG	+ (Sinus tachycardia)
30-day cardiac event monitor	+ (Episodic sinus tachycardia)
2D-cardiac echo	–
Electrophysiology study	–
Ultrasound of renal arteries	–
Autonomic function tests	
Tilt table test	+ (Postural tachycardia)
Valsalva manoeuvre	–
Deep breathing	–
QSART	–
Neurologic tests	
MRI of the brain without contrast	–

+ indicates abnormal results ; – indicates negative or normal results.

ANA, antinuclear antibody; CRP, C reactive protein; ESR, erythrocyte sedimentation rate; QSART, Quantitative Sudomotor Axon Reflex Test.

sinus tachycardia in the range of 110–160 bpm. An electrophysiology study was negative for supraventricular tachycardia and cardiac re-entry rhythm. 2D echocardiography showed no evidence of valvular disease, aortic root enlargement or cardiomyopathy, and Doppler ultrasound of the renal arteries showed no evidence of renal artery stenosis. A tilt table test revealed a supine heart rate of 70 bpm, which increased up to 110 bpm within 10 min of tilt, accompanied by stable blood pressure and symptoms of presyncope. Despite the tilt table test results being consistent with POTS, his medical team questioned the diagnosis as POTS is traditionally viewed as a disorder of young Caucasian women. The patient sought out a second and third opinion from clinicians specialising in POTS, at which time, a second tilt table test was again consistent with POTS. At that time, treatment was initiated with increased salt and fluid intake, a low dose of a beta-blocker and avoidance of triggers such as caffeine consumption, prolonged standing, heat, sleep deprivation and dehydration.

Pathophysiology

The pathophysiologic mechanisms of POTS are variable and include neuropathic changes, hypovolaemia, impaired vasoconstriction, exaggerated hyperadrenergic response and autoimmune aetiology. Exaggerated sympathetic activity is a common

pathophysiologic finding in many patients, and volume depletion is another common mechanism resulting in symptoms of hypovolaemia.^{3 4} This likely occurs due to the impairment in the renin-angiotensin-aldosterone system.^{1 2 5} Attenuated cardio-vagal baroreflex sensitivity has also been seen within the patient population.^{6–8} As a result, labile blood pressure, postprandial hypotension and orthostatic hypertension can also occur in patients with POTS. The orthostatic hypertension was noted in our patient during one of his stand tests when his blood pressure increased from supine 70/55 mm Hg with a heart rate of 55 bpm to an upright blood pressure of 140/80 mm Hg with a heart rate of 95 bpm after standing for 3 min.

More recently, it has been suggested that POTS may be an autoimmune disorder as a result of the identification of the following antibodies in the serum of patients: ganglionic acetylcholine receptor antibodies, alpha 1 adrenergic receptor antibodies, beta 1/beta 2 adrenergic receptors antibodies and M1/M2 muscarinic receptors antibodies.^{9–11} There is also evidence that patients with POTS have a higher prevalence of positive antinuclear antibodies and comorbid autoimmune disorders than the general population.¹² In a cohort of 100 patients with POTS, Hashimoto's thyroiditis was the most prevalent comorbid autoimmune disorder, followed by rheumatoid arthritis, lupus and celiac disease.¹²

INVESTIGATIONS

Clinical features

Clinical features of patients with POTS usually fall into orthostatic, such as postural tachycardia, lightheadedness, dizziness and presyncope, and non-orthostatic symptoms, such as fatigue, exercise intolerance, headache, cognitive symptoms, gastrointestinal dysfunction and sleep disturbance.¹³ Symptoms can vary in severity and can result in significant impairment of all activities of daily living, including eating and bathing.¹⁴ At least 25% of patients are unable to work or attend school, and the disability from POTS can be comparable to that of congestive heart failure, chronic obstructive pulmonary disease or chronic kidney failure.^{15 16}

DIFFERENTIAL DIAGNOSIS

Diagnosis

POTS is defined as an increase in heart rate of at least 30 bpm or an absolute heart rate of at least 120 bpm within 10 min of a tilt table test or standing up, without significant change in blood pressure.¹⁷ Another key feature is that a rise in heart rate must be accompanied by symptoms of presyncope and orthostatic intolerance. The testing can be done via a tilt table test or during a simple stand test performed at a doctor's office. Postural tachycardia alone is insufficient for the diagnosis and may occur in some healthy individuals: symptoms of presyncope and/or orthostatic intolerance must be present during the tilt table test and must last for at least 3 months as part of the diagnostic criteria of POTS.¹⁸ In teens 19 years and younger, a 40 bpm heart rate elevation within 10 min of a tilt table test or standing is required for the diagnosis of POTS.¹⁹

Differential diagnosis of POTS includes all other conditions that can cause tachycardia and lightheadedness and must be ruled out. These typically include cardiac arrhythmia,²⁰ thyroid disorders,²¹ dehydration,²² medication side effects, autonomic neuropathy,²³ diabetes,²⁴ anaemia,²⁵ connective tissue disorders,²⁶ panic disorder²⁷ and generalised anxiety disorder.²⁸ However, in some patients, POTS can be comorbid with any of the above-listed disorders. The key to diagnosis is the patient's

history and confirmation via a tilt table test or orthostatic blood pressure and heart rate measurement.

TREATMENT

The therapeutic approach to POTS consists of non-pharmacologic and pharmacologic treatment options, which are used with the goal of reducing symptom severity and functional impairment. Pharmacotherapy includes first-line medications such as fludrocortisone, a mineralocorticoid that augments retention of water and sodium and expands plasma volume²⁹; beta-blockers, which decrease resting and postural tachycardia by reducing sympathetic overactivity²⁹; midodrine, which is an alpha-1 agonist that causes vasoconstriction and increased peripheral resistance, and other commonly used medications for the orthostatic intolerance, tachycardia and fatigue, such as pyridostigmine, ivabradine, clonidine, modafinil, methylphenidate or selective serotonin reuptake inhibitors (SSRIs).²⁹ Intravenous immunoglobulin therapy can be effective in patients with autoimmune dysautonomia and treatment-refractory POTS with serologic evidence of autoimmunity.³⁰

Non-pharmacologic treatment consists of increased fluid intake of at least 2L/day and sodium chloride consumption via diet and/or supplemental sodium chloride of at least 5 mg daily.¹⁵ Avoiding dehydration, prolonged sitting or standing, and medications that can exacerbate orthostatic symptoms are advised. Although POTS is not caused by deconditioning,³¹ it can be exacerbated by a lack of physical activity due to chronic symptoms and significant exercise intolerance, which is present in most patients with POTS. In several studies, a structured exercise programme has been shown to improve the quality of life in patients with POTS,^{32–34} but more studies are needed to design individualised exercise programmes that the patients are capable of completing. The patients appear to tolerate exercise that minimises the impact of gravity better than the exercise in an upright position, such as exercise via recumbent bike, rowing machine or swimming. In selected patients with treatment-refractory POTS, intermittent intravenous saline infusions can improve symptoms and quality of life.³⁵

OUTCOME AND FOLLOW-UP

Over the following 12–15 months, the patient's symptoms improved with the use of non-pharmacologic treatment options, such as increased fluid and sodium intake, and a structured exercise programme. His functional status and energy level have also improved, but he continues to struggle with fatigue. A trial of metoprolol tartrate 12.5 mg daily was beneficial for reduction of postural tachycardia but resulted in worsening of his fatigue, so the patient discontinued it.

DISCUSSION

In the current case study, the patient presented with typical clinical features of POTS associated with a heart rate increase of >30 bpm on physical examination and on a tilt table test, confirming the clinically suspected diagnosis. As expected, his blood pressure medication likely exacerbated his POTS symptoms and once discontinued, his symptoms improved. Medications known to exacerbate the symptoms of POTS include angiotensin-converting enzymes inhibitors, alpha-blockers, calcium channel blockers, diuretics, monoamine oxidase inhibitors, tricyclic antidepressants and phenothiazine.¹⁴ If the use of these medications is absolutely necessary, they should be used with caution in patients with POTS.

As it commonly occurs in many patients with POTS, our patient was initially misdiagnosed with an anxiety disorder and was referred for psychiatric evaluation. The symptoms of anxiety disorders often mimic the symptoms of POTS resulting in misdiagnosis with many psychiatric conditions, including generalised anxiety disorder, panic disorder, obsessive-compulsive disorder, conversion disorder, factitious disorders, malinger and depression. Although some of the physical symptoms of POTS mimic anxiety, such as palpitations, tachycardia, chest pain and dyspnoea, the underlying pathophysiology is different. A number of studies suggested that the prevalence of anxiety disorders in patients with POTS is not higher than in general population, but physical anxiety can be a symptom of POTS, similar to anxiety being a symptom of hyperthyroidism.³⁶ Complicating the issue further, patients with a chronic illness that results in significant functional impairment are at a higher risk of developing anxiety, depression and suicidal ideations than the general population.³⁷ When present, both POTS and anxiety or depressive disorders need to be treated concurrently in order to decrease symptoms and improve the functional impairment caused by both disorders.

Our patient was an African-American man who falls outside of the typical demographics for POTS, 80% of whom are women, aged 15 to 50 and least 90% of whom are Caucasian patients. Although he presented with typical clinical features of POTS and objective evidence of POTS on a tilt table test which was done twice, his diagnosis of POTS was still questioned and delayed mainly due to his race and gender. Although there have been no formal studies on the prevalence of POTS in non-white population, two large studies noted that their POTS population was 93%–94% Caucasian, about 3% Hispanics, 1% Asian-American and 1% African-American.^{15 38} Although the exact reasons for the racial disparity are unknown, the possibilities may include a higher genetic predisposition for certain autoimmune disorders in people of European descent and variability in renal sodium absorption via a renin–angiotensin–aldosterone pathway between African-American and Caucasian population.^{39–42}

CONCLUSION

In summary, POTS is a disorder of the ANS that is both frequently misdiagnosed with other disorders, such as dehydration, anxiety, anaemia, a viral illness or an eating disorder, and underdiagnosed due to a lack of awareness and knowledge by the healthcare providers. African-American patients with POTS are particularly vulnerable to misdiagnoses or a lack of diagnosis since POTS is commonly viewed as a disorder of young Caucasian women. As in the case of our patient, we suspect that other non-white patients with POTS are likely undiagnosed or misdiagnosed with other medical or psychiatric conditions.

Patient's perspective

I, the second author of this case report, developed POTS as a medical student and have had difficulty obtaining the correct diagnosis, despite being evaluated by several physicians. I was eventually diagnosed with POTS, but the diagnosis came with some objection and doubt because of my race and gender. Once my POTS diagnosis was confirmed, I was able to start the proper treatment for it and come to terms with my diagnosis. I am much improved now and hope to start residency training in Emergency Medicine in 2020.

Learning points

- Postural orthostatic tachycardia syndrome (POTS) is diagnosed based on the typical clinical features of orthostatic intolerance, dizziness, fatigue and other symptoms of presyncope and objective evidence of heart rate elevation by at least 30 bpm from supine to standing position, either via a tilt table test or a stand test.
- Patients with POTS are often misdiagnosed with anxiety disorders and other psychiatric conditions because symptoms may appear similar, but the underlying pathophysiology, diagnosis and treatment are different.
- Although Caucasian women of reproductive age are more likely to have POTS, it should be considered in the differential diagnosis in patients of any gender, race or ethnicity who present with typical clinical features and objective evidence of POTS on a tilt table test or stand test.
- Prompt diagnosis and early interventions lead to the best outcomes for patients who experience significant functional impairment as a result of POTS.

As POTS affects millions of people worldwide, physicians need to be aware of its clinical features, diagnostic criteria and various treatment options. Increased awareness, proper diagnosis and reduced diagnostic delay will ultimately result in improved patient care and reduced disability in patients affected by POTS.

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