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Paroxysmal orthostatic tachycardia syndrome (POTS); "standing up" to a common cause of syncope

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Abstract

Paroxysmal orthostatic tachycardia syndrome (POTS) is characterized by a combination of symptoms including lightheadedness, palpitations, weakness and fatigue which are associated with positional change. This entity is fairly ignored in the differential diagnosis of syncope & pre-syncope in comparison to its more famous counterpart orthostatic hypotension despite being extensively studied and having a higher incidence. We report a case of a 62 year old man who was admitted to the hospital for pre-syncope and was diagnosed with POTS. We aim to increase awareness about this syndrome to help prevent unnecessary diagnostic testing which in-turn will decrease health care costs.

Keywords

paroxysmal orthostatic tachycardia; hypotension; pre-syncope; syncope; tachycardia

Introduction

POT is characterized by a constellation of symptoms including (1) lightheadedness, palpitations, weakness, visual disturbances, tremors and fatigue associated with the upright position; (2) elevation of heart rate(HR) ≥30 when changing from the supine to the upright position; and (3) the absence of orthostatic hypotension [1,5]. Pre-syncope and syncope are the most common presentations of this cardio-neurogenic entity [3,4]. POT is much more common than it is diagnosed with an annual incidence of nearly 500,000 cases per year in the US alone and it is more prevalent than its more famous counterpart orthostatic hypotension [1,2,5].

Case Presentation

A 62-year-old patient with a history of well controlled hypertension& diabetes mellitus (DM)presented to the Emergency department (ED), for a pre-syncopal episode and complaints of generalized fatigue and weakness that had been going on for several years. Initial vitals in the ED were blood pressure 145/85 mmHg, heart rate (HR) 110, respiratory rate 17, temperature 37.8C and saturations of 98% on room air. Physical examination was only significant for a very anxious looking gentleman with complaints of fatigue that kept insisting that he was only here because his newly appointed care taker wanted him to come. Bedside neurological exam was unremarkable. Additional ED tests including electrolytes, blood sugars, EKG, urinalysis, thyroid function and CXR were all

unimpressive. Patient was admitted for further extensive workup to rule out neurologic, cardiac and pulmonary sources of symptoms. Testing included, but was not limited to, EKG, CTA, ECHO, carotid and lower extremity doppler & orthostatic's. All diagnostic modalities were negative. Throughout his stay in the hospital, the patient was persistently in sinus tachycardia ranging in 110-120, and at certain points of examination elevating to 130s. No arrhythmias were noted on cardiac monitoring and atrial tachycardias were ruled out. At this point a re-visitation of his history was made and was significant for certain standout clues; patient repeated many times that he has always had a fast HR since he was a child, and was unable to involve himself in strenuous activities as a result of this, combined with his persistent feelings of fatigue and intermittent episodes of pre-syncope . He always felt anxious and hypervigilant. After a review of literature a provisional diagnosis of POT was made and a simple standing test was performed, revealing an increase of HR>30 from the supine without changes in systolic/diastolic blood pressure. Close monitoring of patients HR also revealed a diurnal variation ≥30 beats per minute, with a higher resting HR in the morning as compared to in the evening. Patient was treated with low dose beta blockers (BB) and with physical therapy to address the physical deconditioning. At regular follow-ups patient reported a noticeable improvement in overall symptoms.

Discussion

POTS is essentially an autonomic dysregulation that creates a clinical picture of orthostatic lability characterized mainly by light headedness, fatigue, anxiety, palpitations, exercise intolerance and near syncope in the upright posture. Usually these symptoms are alleviated by a taking up a supine posture. These symptoms result in limitations in activities of daily life [1,2].

POTS maybe divided into primary and secondary subtypes based on etiology and association with other diseases. Primary POTS is idiopathic and is further sub-divided into “partial dysautonomic” (PD) and “hyperadregenic” variants. PD variant is characterized by impaired vascular response to orthostatic variations, with progressive weakening of compensatory mechanisms and increased intensity of symptoms. It maybe developmental, with onset of symptoms at adolescence or abruptly after a viral illness, sepsis or pregnancy alluding to a presumed immune driven pathogenesis. Hyperadregenic variant is signified by an insidious onset and the presence of orthostatic hypertension in addition to orthostatic tachycardia. It is also characterized by elevated levels of catecholamines and a familial history of the disease. Secondary POTS is associated mostly with diseases that result in peripheral autonomic neuropathy, most commonly prolonged DM, Sarcoidosis, lupus and alcoholism to name a few. Management is focused on physical reconditioning with the employment of pharmacotherapy to aid in this primary goal. Most medications used, are off label and include fludrocortisone, midodrine, BB's, selective serotonin re-uptake or norepinephrine re-uptake inhibitors. All therapies are directed at augmenting volume resuscitation and improving vascular resistance, in addition to addressing underlying disorders. Functional limitations associated with untreated POTS have been shown to be comparable to those experienced by patients with congestive heart disease and chronic obstructive pulmonary disease[7].

POTS, is a well-known disease entity, similar to orthostatic hypotension, but 5-10 times more common [2,6]. From limited studies available the sex based prevalence appears to show a preponderance in females as compared to males (female-to-male ratio of 5:1). As such it presents a common confounder