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Case Report

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GI POTS: the unnoticed

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Abstract

GI manifestation of dysautonomia is common clinical syndrome which is underestimated by clinicians. Postural tachycardia syndrome (POTS) is one of the most common causes of orthostatic intolerance and is being increasingly recognized in clinical practice. Gastrointestinal (GI) symptoms are reported in majority of patients with POTS Because of pathophysiologic heterogeneity, diversity of clinical presentation, and unpredictability of clinical response, POTS patients pose a considerable management challenge and, optimally, require a multidisciplinary approach to both evaluation and management. It's important for practising gastroenterologists to be aware of this complex syndrome and its GI co-morbidities. The available clinical evidence does not prove causal association between GI symptoms and POTS. Currently treatment of GI symptoms in patients with POTS remains largely non pharmacological and mostly symptom oriented. General measures to treat POTS may lead to improvement in both GI and non-GI symptoms in majority of patients. We present a Case series with literature review on POTS and its association with GI symptoms, evaluation and management.

Keywords: GI POTS

Introduction

Postural Orthostatic Tachycardia Syndrome (POTS) is a form of dysautonomia characterized by an exaggerated increase in heart rate upon assuming an upright posture, in the absence of orthostatic hypotension. It represents one of the most common yet underrecognized causes of chronic orthostatic intolerance, affecting predominantly young women. Although traditionally considered a cardiovascular disorder, accumulating evidence highlights the frequent coexistence of gastrointestinal (GI) symptoms such as bloating, nausea, vomiting, and altered bowel habits in POTS patients.

The interplay between the autonomic nervous system and gastrointestinal function is intricate. Disruption of this regulatory mechanism can lead to gastrointestinal dysmotility, impaired accommodation, and variable transit times, all of which contribute to symptom burden and impaired quality of life. Despite these associations, the GI manifestations of POTS are often overlooked or misattributed to functional gastrointestinal disorders, leading to delays in diagnosis and management.

Understanding the gastrointestinal aspects of POTS is crucial for clinicians, particularly gastroenterologists, as early recognition can guide appropriate multidisciplinary care and improve outcomes. This paper presents a case series highlighting the diverse GI manifestations of POTS, discusses underlying mechanisms, diagnostic approaches, and outlines current therapeutic strategies with a focus on integrated, patient-centered management.

Case Report

Case 1: 27-year-old female, student, presented with bloating of abdomen of 8 years duration. Symptoms were intermittent and last for only few hours. These events didn't follow a pattern and no specific aggravating factors. History of allergic rhinitis and hypothyroidism and were under control. She was evaluated by a physician and cardiologist for palpitations, underwent Holter study and was diagnosed to have sinus tachycardia. Her basic blood investigations were normal. Her supine to standing heart rate was significant to diagnose POTS. She was advised on non-pharmacological therapy and she is better on follow up.

Case 2: 21-year-old female, student from US, perennial recurrent episodes of nausea and vomiting for more than 1 yr. She was vomiting mucoid, non-bilious secretions with mild abdomen discomfort during the episodes. She had a history of hypothyroidism and PCOD. She recollected some self-resolving fainting episodes and incidentally noted to have heart rate ranging from 60 to 188. Her supine heart rate was 60 and on standing

after 5 minutes it reached 100. She was diagnosed to have sinus tachycardia and treated with beta-blockers. Diagnosis of POTS was made and she was advised with non-pharmacological and symptomatic treatment.

Case 3: 31-year-old male, returned back from Australia and had symptoms of episodes of giddiness, brain fog on and off for 1 yr. Evaluated by ENT surgeon, physician, cardiologist and underwent routine panel of tests. Neurological evaluation with MRI brain and EEG was normal. Meanwhile patient experienced intermittent episodes of breathing difficulty, his glucose tolerance test was abnormal. Clinical diagnosis of reactive hypoglycaemia was made by endocrinologist and was treated with complex carbohydrates and acarbose. He was referred to GI clinic for bloating and gas problems. Heart rate varied from 50 to 140 /min. He experienced severe symptoms impairing quality of life and was on antidepressants. His symptoms analysis and postural heart rate testing was positive for POTS. He was started on fludocortisone and midodrine with complex carbohydrates and achieved good symptom improvement on follow up.

Case 4: 31-year-old male, having giddiness and palpitations symptoms for more than 1 yr and was evaluated by cardiologist for his symptoms. ECG, echocardiogram, Holter, CT chest, and coronary angiography were normal. Visited GI clinic for his troublesome GI symptoms like altered bowel habits, intermittent bloating, chest pain and belching. Underwent blood investigations were normal. Celiac screen, food allergy test, Contrast CT abdomen and upper GI scopy with segmental biopsy were unremarkable. Postural change in heart rate confirmed POTS. He was started on fludrocortisone and midodrine with symptomatic improvement.

Case 5: 43-year-old female presented with 3 yrs history of palpitations, giddiness and found to hypoglycemic episodes more after an hour of food intake. Extensively evaluated with blood investigations and imaging and found to be normal. Visited GI clinic for troublesome bloating, hot flushes and dizziness which was attributed to acidity, she was on acarbose. Her evaluation was suggestive of POTS. She was advised on exercises, complex carbohydrates, beta blockers and treated as late dumping.

Discussion

Postural orthostatic tachycardia syndrome (POTS) is a form of dysautonomia seen in young adults which is associated with gastrointestinal symptoms including nausea, bloating and abnormal bowel movements. Underlying dysautonomia involving the enteric nervous system may account for gastric arrhythmia and dysmotility, but this is a simple description of the pathophysiologic mechanisms [1]. POTS term was coined by Schondorf and Low from Mayo clinic in 1993. Since mid-19th century these complex symptoms are referred as Da Costa syndrome, Soldiers heart, Irritable heart or Vasoregulatory asthenia.

POTS is considered the most common cause of chronic orthostatic intolerance. Orthostatic intolerance describes symptoms that develop in response to upright posture and, classically, resolve upon recumbency. Orthostatic hemodynamic dysregulation occurs when the normal physiologic gravitational regulatory mechanism is impaired. Prime hemodynamic feature in POTS is an excessive heart rate increase without a corresponding decrease in blood pressure. POTS is more common among young women with a female: male ratio of at least 4.5:1, an age range between 15 and 50 years and estimated prevalence of 170/100,000 people [1, 6]. A family history of orthostatic intolerance has been reported in 13% of these patients [2]. Delay in onset of symptoms to reaching diagnosis varies from 4 to 8 years in various reports. Common presenting symptoms are caused by cerebral hypoperfusion and / or sympathetic over activity which includes light headedness, blurred vision, difficult cognition, lower extremity or generalized weakness, syncope, palpitations, chest pain, dyspnoea, tremors and paresthesias. Classically, symptoms of orthostatic intolerance improve with recumbency, but resolution may be incomplete or delayed. Non-orthostatic symptoms commonly seen in POTS include dry eyes or mouth, and a variety of urinary and gastrointestinal (GI) complaints.

Core GI symptoms include nausea (86%), irregular bowels (71%), abdominal pain (70%), constipation (70%), heartburn (64%), cramps (61%), bloating (59%), Less common symptoms include diarrhoea, regurgitation, dysphagia, vomiting and gagging. Few of our patients presented with features of dumping syndrome with alteration in blood glucose levels documented by GTT. Symptoms related to late dumping is noted in two of our patients. Both orthostatic and non-orthostatic symptoms can be severe and contribute considerably to diminished quality of life. POTS patients has co-morbid conditions like chronic fatigue syndrome, fibromyalgia, interstitial cystitis, migraine headaches, hyper mobile form of Ehlers Danlos syndrome, mast cell activation disorder and median arcuate ligament syndrome.

While the underlying etiology is not established, multiple pathophysiologic pathways contribute to clinical syndrome of POTS with a general consensus of excessive tachycardia in a setting of cardiovascular deconditioning as a common pathway. [A] Neuropathic POTS: autonomic neuropathy characterized by predominantly lower limb sympathetic denervation leading to reduced venoconstriction and venous pooling in lower limbs. Thus, an excessive cardiovascular response is necessary to maintain adequate mean arterial pressures [1,6]. [B] Hyperadrenergic POTS: 30-70% falls in this subtype with elevated standing plasma norepinephrine levels more than 600pg/ml. [C]Hypovolemic POTS more than 70 % patient exhibit low plasma volume with a paradoxically low renin and aldosterone levels. [D]Autoimmune POTS: post viral onset, elevated auto immune markers, female preponderance and presence of antibody ganglionic AChR and various nonspecific autoantibodies proposes an autoimmune process. [E] Deconditioning: Physical and cardiovascular deconditioning is often evident in patients with POTS, although its presence as either a cause or effect is not clear [9]. Often POTS is associated with somatic hypervigilance, behavioural amplification, anxiety and depression [4].

In normal individual autonomic neural mechanism controls GI tract motility, secretion, and blood flow to enable digestion, absorption and excretion. Enteric nervous system (ENS) includes myenteric and submucosal plexus of the gastrointestinal tract; GI motility is primarily under the control of the myenteric plexus between the longitudinal and circular muscles. The submucosal plexus, located between circular muscles and mucosa, regulates fluid absorption, secretion, and blood flow, and responds to exogenous stimuli to maintain GI function [3]. Central nervous system sends neural inputs to modulate and control GI functions,

predominantly to oesophagus and stomach where as small intestine and colon are more independent. Extrinsic innervation is through parasympathetic and sympathetic nervous afferent and efferent nerves, sympathetic nervous system negatively regulates motor and secretory function, the absence of sympathetic inhibitory innervation causes excessive uncoordinated GI motility. Meal's rich in carbohydrate increases splanchnic sequestration of blood and may be resulting in relative hypovolemia and can explain post prandial increase of POTS symptoms.

Evaluation includes detail history and examination in looking for primary cardiovascular, neurological or GI problems. Diagnosis is by consensus criteria [5]. An active stand test consists of having the patient lying supine for 10 minutes with measurement of supine baseline blood pressure and heart rate; the patient then stands and with re-measurement of BP and HR at timed intervals (1, 3, 5, and 10 minutes). The head-up tilt table (HUT) with non-invasive hemodynamic monitoring is the accepted gold standard for assessing orthostatic intolerance in POTS. Laboratory investigations include evaluation for anaemia, electrolytes, thyroid test, serum cortisol, serum and urinary catecholamines, upright plasma norepinephrine levels more than 600 pg/ml. 24-hour ECG & Ambulatory B.P monitoring, and Echocardiography. Assessment of autonomic functions includes quantitative sudomotor sweat test and thermoregulatory sweat test. GI related tests include routine investigations for dyspepsia, gluten sensitivity, food allergy tests and upper GI scopy with biopsies to rule out other common organic cause. Gastric emptying studies have shown delayed gastric emptying and impaired accommodation, in a recent study 11 % had delay in gastric emptying and 48% had abnormal rapid gastric emptying [7].

Table 1: Consensus criteria for POTS [5]

• Heart rate increase ≥30bpm within 10min of upright posture in adults (≥40bpm in adolescents 12–19 years)
• Absence of orthostatic hypotension (sustained drop-in blood pressure ≥20/10mmHg within minute of upright posture)
• Orthostatic intolerance symptoms for ≥6 months
• Absence of other causes such as dehydration, other medical conditions, medications, and dietary infuences

First important step in management is a right diagnosis; core impact is in explaining the patient about the disease. Understanding its benign nature brings confidence to the patients and reflects on more good clinical outcomes. Non pharmacological treatment includes avoidance of prolonged recumbency and gradual rising from supine and sitting, avoiding prolonged standing and high temperature and humidity. Most important is to identify a trigger which differs for different individuals. Whole body head end elevation during sleep increases reconditioning in many patients. Mornings are tough for many hence scheduling works later in day may help, it's important to hydrate well before getting up from bed. Physical counter measures like leg crossing, muscle tensing, forward bending; squatting during standing and expected prodromal phase should be encouraged. Various exercise programs are available a regular, structured, graduated, supervised reconditioning exercise programs are preferable. Dietary modifications in GI POTS include increasing complex carbohydrates, proteins and reduction in fat and fibre.

More blenderised meals and decreasing size of meals and increasing the frequency was found to be helpful. Increased salt (> 10 gm) and fluid intake (2.5 L) promotes volume expansion in hypovolemic POTS. Caffeine and alcohol exacerbate symptoms hence to be avoided. Compressive stocking and garments may prevent peripheral pooling and improve symptoms.

Beta blockers like propranolol and metoprolol helps in controlling heart rate in hyper adrenergic variants though may increase hypotension. Ivabradine helps in patients with palpitations and low blood pressure. Other volume expanding medications used are clonidine, midodrine, fludrocortisone and pyridostigmine. We have tried beta blockers, fludrocortisone and midodrine in our patients with good symptom improvement. Symptomatic treatment of bloating includes prokinetic agents like erythromycin, metoclopramide, domperidone and ondansetron. Promethazine and lorazepam also found to be useful. Tricyclic antidepressants have been tried in refractory nausea. SSRI, SNRI (Venlafaxine) help with variable results[10].

Natural history of POTS remains unclear studies have shown in improvement of symptoms in more than 80% without any increased risk of mortality [8]. Those who had antecedent viral illness and acute onset symptoms had a better outcome than those with spontaneous onset and with a positive family history. Overall data suggest cure is uncommon but many patients improve over time [5].

Our patient's symptoms were predominantly bloating, belching, palpitations and abdomen discomfort. Non-GI symptoms like palpitations, tiredness, irritability, most of them have seen cardiologist, neurologist for their symptoms, and were started on beta blockers for sinus tachycardia. Two other patients had symptoms of late dumping with hypoglycaemia documented by glucose tolerance test and treated by endocrinologist with acarbose. All had postural test positive for POTS, 3 patients had gastric emptying studies and found to have rapid gastric emptying. Three of them were started on fludrocortisone and midodrine, along with symptomatic treatment for bloating and other GI symptoms. All of them required SSRI. Explaining the disease process brought a huge difference in their symptoms.

Conclusion

POTS is a complex syndrome that is underestimated and ignored by many health professionals, causing perplexity in both patients and doctors. It's a distress in disguise to the patients, unmasking them by early diagnosis improves the quality of life in them. GI symptoms are common in patients with POTS. As this syndrome don't follow a definite clinical pattern of illness pattern in a patient, the POTS is often overlooked and misdiagnosed as functional disorders, Idiopathic arrhythmia, reactive hypoglycaemia and chronic fatique syndrome. Patients have poor quality of life with undiagnosed and under treated symptoms. They predominantly end up with neurologist, cardiologist and gastroenterologist for symptomatic relief. Currently most patient are left in dark and treated symptomatically with anxiolytics and antidepressants. Widespread dissemination of knowledge will help in learning more about the syndrome, early diagnosis and

exploring treatment options for the emerging epidemic known as GI POTS.

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