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POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME (POTS): CLINICAL PRESENTATION, DIAGNOSIS, PATHOPHYSIOLOGY AND TREATMENT OPTIONS – A LITERATURE REVIEW

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ABSTRACT

Introduction: Postural orthostatic tachycardia syndrome (POTS) is a chronic disorder of the autonomic nervous system characterised by orthostatic intolerance and an excessive increase in heart rate (≥ 30 beats per minute) upon standing, in the absence of orthostatic hypotension. Patients often experience additional symptoms such as fatigue, dizziness and palpitations. The disorder predominantly affects young women. Although the aetiology remains unclear, several mechanisms have been proposed. Due to its complex clinical presentation and limited awareness among healthcare professionals, POTS is frequently misdiagnosed.

Aim: The aim of this review is to summarise the current knowledge regarding the aetiology, clinical presentation, diagnosis and treatment of postural orthostatic tachycardia syndrome.

Materials and methods: This review is based on literature available in the PubMed database. Articles were identified using keywords including postural orthostatic tachycardia syndrome, POTS, orthostatic intolerance and tachycardia, and the most relevant publications were analysed.

Results: POTS remains a moderately understood disorder. The prevalence is estimated at approximately 0.2% of the general population, with women of reproductive age accounting for up to 90% of cases. The pathophysiology is multifactorial and includes three main mechanisms: partial autonomic neuropathy, hypovolemia and a hyperadrenergic state. Management includes both non-pharmacological interventions, such as increased fluid intake and lifestyle modification, and pharmacological therapies including beta-blockers and ivabradine.

Conclusions: POTS remains a diagnostic and therapeutic challenge. Increased awareness and further research are essential to improve diagnosis and patient outcomes.

KEYWORDS

Postural Orthostatic Tachycardia Syndrome, Orthostatic Intolerance, Autonomic Disorders, Tachycardia

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Introduction

Postural orthostatic tachycardia syndrome (POTS) is a chronic disorder characterised by orthostatic intolerance and an excessive increase in heart rate without a significant decrease in blood pressure after assuming an upright position. Patients with POTS frequently experience additional symptoms associated with autonomic nervous system dysfunction, including dizziness, generalised weakness and palpitations (Lau et al., 2026; Vernino et al., 2021). The pathophysiology of POTS is not yet fully understood; however, significant progress in research has been made in recent years. Hypovolemia, vascular dysfunction and dysregulation of the autonomic nervous system are considered among the most probable contributors to the pathophysiology of POTS (Wei et al., 2025). Women are far more likely to be affected by POTS than men (approximately a 9:1 ratio), particularly women of reproductive age (Lau et al., 2026). The onset of POTS may be triggered by numerous factors, most commonly infections, hormonal changes (e.g. pregnancy or menopause), or severe physical trauma (Lau et al., 2026; Shaw et al., 2019). The disorder has a substantial impact on patients' lives, significantly reducing quality of life and often limiting the ability to work or maintain social interactions. Because the symptoms of POTS can be nonspecific and diagnostically challenging, many patients experience misdiagnosis or report that their symptoms are not taken seriously by healthcare professionals (Walsh et al., 2025). Further research and improved education regarding POTS are necessary to ensure timely diagnosis and appropriate management of this condition.

Epidemiology

Epidemiological studies estimate the prevalence of POTS to be approximately 0.2% in the general population (Wei et al., 2025). However, this estimate may be inaccurate due to limited awareness and knowledge of the disorder among healthcare professionals. Misdiagnosis or diagnostic delay is not uncommon and may affect epidemiological data. Women are disproportionately affected by POTS compared with men, with some studies indicating that up to 90% of patients are female (Lau et al., 2026; Mar & Raj, 2020). The disorder typically begins during adolescence or early adulthood; therefore, POTS is also observed in the paediatric population. The most common age of onset is approximately 14 years (Vernino et al., 2021).

Pathophysiology

The exact cause of POTS remains unclear; however, it is widely accepted that its aetiology is multifactorial. Understanding POTS requires a holistic approach that considers not only the cardiovascular system but the body as a whole.

The literature describes three main pathophysiological mechanisms:

1. partial autonomic neuropathy,
2. hypovolemia, and
3. a hyperadrenergic state.

Based on these mechanisms, three corresponding phenotypes have been proposed (neuropathic, hypovolemic and hyperadrenergic POTS) (Angeli et al., 2024; Mar & Raj, 2020; Vernino et al., 2021; Yeh et al., 2024). These phenotypes are not mutually exclusive and may coexist in individual patients (Angeli et al., 2024; Mar & Raj, 2020). Identifying the predominant phenotype may help guide appropriate treatment and management strategies (Angeli et al., 2024).

Partial autonomic neuropathy results from dysregulation between the sympathetic and parasympathetic divisions of the autonomic nervous system in the vascular beds. In the lower half of the body, impaired sympathetic activity leads to excessive vasodilation and blood pooling in the lower extremities and pelvic circulation. This pooling results in relative hypovolemia, as a significant portion of blood remains in the lower body rather than returning to the heart and brain, ultimately leading to cerebral hypoperfusion. As a compensatory mechanism, tachycardia develops in order to maintain adequate cerebral blood flow (Mar & Raj, 2020; Wei et al., 2025).

Hypovolemia also plays a significant role in the pathophysiology of POTS. Patients with POTS have been shown to have 13–22% lower blood volume compared with healthy individuals (Mar & Raj, 2020). Insufficient fluid intake or excessive fluid loss may contribute to this condition. In addition, studies have demonstrated that patients with POTS exhibit the so-called “renin–aldosterone paradox”. Under normal conditions of hypovolemia, renin and aldosterone activity should increase to promote fluid retention. However, in patients with POTS, these hormone levels are paradoxically lower than those observed in healthy individuals. This mechanism may contribute to the development or exacerbation of hypovolemia (Arnold et al., 2018; Raj et al., 2005).

Another proposed pathophysiological mechanism of POTS is a hyperadrenergic state. This condition is characterised by elevated catecholamine levels and increased sympathetic nervous system activity. Upon assuming an upright position, plasma norepinephrine levels normally increase compared with the supine position. In patients with POTS, however, these levels may increase more than threefold, resulting in excessive tachycardia (Mar & Raj, 2020). Among patients with this phenotype, studies have identified a subgroup with mutations affecting the norepinephrine transporter gene. Impaired function of this transporter results in increased norepinephrine concentrations within the synaptic cleft, leading to excessive sympathetic activation (Fedorowski, 2019; Shannon et al., 2000).

POTS shares several clinical and epidemiological similarities with autoimmune diseases; therefore, an autoimmune aetiology has been widely discussed. The onset of POTS is often subacute and may be preceded by viral infection (e.g. COVID-19), pregnancy, surgery or physical trauma. Patients with POTS are predominantly young women, and up to 20% are diagnosed with an autoimmune disease or have a family history of such conditions. Various autoantibodies have been detected in patients with POTS, including Sjögren’s syndrome-related autoantibodies, antinuclear antibodies and antiphospholipid antibodies, which supports the hypothesis of autoimmune involvement (Aboseif et al., 2023; Fedorowski, 2019). Another group of autoantibodies associated with POTS targets β 1- and β 2-adrenergic receptors. Compared with healthy controls, higher levels of these antibodies have been detected in patients with POTS. Acting as receptor agonists, these antibodies may stimulate the sympathetic nervous system and contribute to the development of tachycardia (Fedorowski, 2019; Mar & Raj, 2020).

Clinical presentation

The typical patient with POTS is a young woman (approximately 25 years of age) who first experiences symptoms during adolescence. The main symptoms of POTS include orthostatic intolerance, tachycardia, lightheadedness and chronic fatigue. Patients frequently report palpitations, exercise intolerance and chest pain (Fedorowski, 2019). Certain symptoms, including headache, migraine, dizziness and “brain fog”, are believed to be associated with decreased cerebral perfusion (Wei et al., 2025). Additionally, syncope occurs in approximately 30–50% of patients (Fedorowski, 2019). Gastrointestinal symptoms are also common, with frequently reported complaints including nausea, diarrhoea, constipation and abdominal pain (Angeli et al., 2024; Lau et al., 2026; Mar & Raj, 2020). Patients often report sleep disturbances and excessive daytime sleepiness, which further contribute to a reduced quality of life (Bagai et al., 2011).

Diagnostic approach

POTS is defined as orthostatic intolerance accompanied by an excessive increase in heart rate upon assuming an upright position while blood pressure remains relatively stable. Patients typically present with additional symptoms associated with orthostatic intolerance. To standardise the diagnostic process, medical societies have established the following criteria:

1. Increase in heart rate of ≥ 30 beats per minute within 10 minutes of standing or during a head-up tilt test (≥ 40 beats per minute in patients aged 12–19 years);
2. Absence of sustained systolic blood pressure decrease greater than 20 mmHg;
3. Presence of symptoms of orthostatic intolerance during standing that improve upon returning to a supine position (e.g. dizziness, lightheadedness, fatigue, palpitations);
4. Duration of symptoms for at least three months;
5. Exclusion of other causes of sinus tachycardia such as medical conditions (e.g. anaemia, dehydration, hyperthyroidism), medications and substances (e.g. sympathomimetics, anticholinergics, alcohol, caffeine), or physiological conditions (e.g. fever, infection, pain or panic attacks) (Arnold et al., 2018; Raj et al., 2022; Vernino et al., 2021).

Diagnostic delays are considerable, ranging from 5 to 7 years, and are significantly longer in women than in men (7 years vs. 3.8 years) (Lau et al., 2026; Seeley et al., 2025). Women also experience misdiagnosis more frequently, often due to the attribution of their symptoms to psychogenic causes. Interestingly, despite the higher prevalence of POTS in women and the absence of major differences in reported symptoms, the diagnostic process in women remains longer (Seeley et al., 2025).

Treatment and management

POTS is a chronic disorder with a multifactorial and still incompletely understood aetiology. As no treatment directly addresses the underlying cause, management focuses primarily on symptom control. (Fedorowski, 2019) Both non-pharmacological and pharmacological approaches are used, with non-pharmacological strategies recommended as the initial step in treatment (Lau et al., 2026).

Non-pharmacological management primarily focuses on patient education and lifestyle modification. Recommended interventions include increasing fluid intake to approximately 3 liters per day and increasing salt intake to approximately 10 g per day. These strategies are particularly beneficial for patients with the hypovolemic phenotype of POTS, as they increase fluid retention and overall blood volume (Garland et al., 2021; Lau et al., 2026; Williams et al., 2022). Reducing caffeine and alcohol consumption may also be beneficial (Lau et al., 2026). Compression stockings and abdominal binders can help reduce venous pooling in the lower extremities and abdominal circulation and are particularly recommended for patients with neuropathic or hypovolemic POTS (Fedorowski, 2019; Lau et al., 2026; Mar & Raj, 2020). Certain medications may exacerbate sinus tachycardia and should therefore be avoided, including diuretics, selective serotonin reuptake inhibitors (SSRIs) and serotonin–norepinephrine reuptake inhibitors (SNRIs). Additional triggers may include prolonged standing, hot and humid environments and large meals (Fedorowski, 2019; Lau et al., 2026). Patient education plays a crucial role in enabling individuals to effectively manage their condition (Fedorowski, 2019).

Pharmacological therapy is often necessary. Although several medications are used in clinical practice, many are prescribed off-label. Identification of the dominant pathophysiological mechanism may help guide the selection of appropriate pharmacological treatment (Lau et al., 2026; Mar & Raj, 2020). Midodrine is an $\alpha 1$ -adrenergic receptor agonist that causes vasoconstriction and is commonly used in neuropathic and hypovolemic POTS. It is particularly effective in patients with low supine blood pressure and helps alleviate

symptoms of orthostatic intolerance (Lau et al., 2026; Raj et al., 2022). Blood-volume expanders such as fludrocortisone and desmopressin are also used. Fludrocortisone promotes renal sodium reabsorption, resulting in water retention, whereas desmopressin, a synthetic analogue of vasopressin, has an antidiuretic effect and increases circulating blood volume. Because both drugs affect electrolyte balance, regular monitoring is required. These medications are particularly useful in patients with hypovolemic POTS (Mar & Raj, 2020; Raj et al., 2022).

In hyperadrenergic POTS, treatment aims primarily to reduce heart rate. Beta-blockers such as propranolol, metoprolol and bisoprolol reduce sympathetic nervous system activity and help control tachycardia. Pyridostigmine may also reduce heart rate through a different mechanism, as it enhances parasympathetic activity. As an acetylcholinesterase inhibitor, it prevents the breakdown of acetylcholine and enhances activation of nicotinic and muscarinic receptors (Fedorowski, 2019; Lau et al., 2026; Mar & Raj, 2020; Raj et al., 2022). However, gastrointestinal adverse effects such as diarrhoea, abdominal cramping and nausea are relatively common (Mar & Raj, 2020). Ivabradine is a newer treatment option that selectively blocks If sodium channels in the sinoatrial node, thereby reducing heart rate. Some studies also suggest that ivabradine may indirectly reduce sympathetic nervous system activity. Unlike beta-blockers, ivabradine typically does not cause hypotension (Lau et al., 2026; Mar & Raj, 2020; Raj & Sheldon, 2021; Taub et al., 2021).

Quality of life in POTS

Patients with POTS experience a wide range of symptoms, including palpitations, tachycardia, dizziness and gastrointestinal disturbances. As a result, their quality of life is often severely impaired. The impact of the disorder extends far beyond physical discomfort. Patients frequently experience difficulties performing everyday tasks, and the condition may significantly affect their professional and social lives (Seeley et al., 2023). Sleep disturbances, chronic fatigue, “brain fog” and cognitive impairment may reduce the ability to concentrate and maintain employment. Some patients become unable to work and may require disability benefits due to loss of income. According to one study, more than 70% of patients reported losing their employment because of POTS symptoms (Bourne et al., 2021). POTS may also negatively affect mental health. Anxiety and depression are relatively common among affected individuals, and many patients withdraw from social activities (Seeley et al., 2023). In many cases, patients experience significant diagnostic delays, which may lead to feelings of frustration, dismissal or lack of understanding by healthcare professionals (Walsh et al., 2025). Earlier diagnosis and improved education of medical professionals may help reduce the negative impact of POTS on patients’ quality of life.

Conclusions

POTS is a chronic disorder characterised by orthostatic intolerance and tachycardia without a significant decrease in blood pressure upon standing. Patients experience a wide range of symptoms, including dizziness, lightheadedness, fatigue, palpitations, “brain fog”, chest pain, nausea and exercise intolerance. The disorder predominantly affects young women, with the age of onset typically occurring between 12 and 50 years and often following triggers such as viral infection (Lau et al., 2026). Diagnosis requires fulfilment of several criteria, including an increase in heart rate of at least 30 beats per minute upon standing without orthostatic hypotension, the presence of symptoms of orthostatic intolerance, and symptom duration of at least three months. Other potential causes of sinus tachycardia must also be excluded (Vernino et al., 2021). Although the aetiology of POTS remains unclear, several mechanisms have been proposed, including a hyperadrenergic state, hypovolemia, autonomic neuropathy and autoimmune processes. Identifying the dominant pathophysiological mechanism may help guide appropriate treatment. Non-pharmacological interventions and patient education form the foundation of management; however, many patients require additional pharmacological therapy (Mar & Raj, 2020). POTS is a chronic and often debilitating disorder that significantly reduces quality of life. It interferes with everyday functioning, disrupts professional careers and may result in financial difficulties and social isolation. Patients with POTS are also at increased risk of mental health disorders. The prolonged search for an explanation of their symptoms often involves years of diagnostic testing and misdiagnoses, which may exacerbate anxiety and feelings of being dismissed (Bourne et al., 2021; Walsh et al., 2025). Further research into the pathophysiology and treatment of POTS is essential. Improved understanding of the disorder, increased awareness among healthcare professionals and the development of more effective therapies may ultimately improve the quality of life of patients living with POTS.

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