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POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME (POTS) IN PRIMARY CARE: DIAGNOSIS AND MANAGEMENT

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ABSTRACT

This article sets out a practical approach to recognising and managing postural orthostatic tachycardia syndrome (POTS) in primary care, drawing on current epidemiological, pathophysiological and therapeutic evidence. POTS is defined as a chronic syndrome of orthostatic intolerance with an excessive rise in heart rate on standing, without a meaningful fall in blood pressure, leading to a marked reduction in quality of life, particularly in young women. The article reviews the complex and heterogeneous causes of POTS, including a neuropathic component, hypovolaemia, a hyperadrenergic phenotype and a possible role for autoimmune mechanisms. It describes the typical clinical picture, the symptoms of orthostatic intolerance, and the wide range of coexisting conditions that together substantially limit patients' functioning. A diagnostic scheme is proposed that fits the resources of a family doctor's office, based on a focused history, physical examination, a simplified orthostatic test, basic laboratory work, an ECG, and clear criteria for specialist referral. On treatment, the article stresses the central role of non-pharmacological measures, including increased fluid and salt intake, compression garments, lifestyle changes and a gradual exercise programme, and discusses the supporting role of individually tailored drug treatment. The choice of treatment should reflect the mechanisms that dominate the clinical picture. The aim is to raise awareness of POTS among primary care physicians and to bring it into the differential diagnosis of patients reporting dizziness, palpitations, presyncope and chronic fatigue, so as to shorten the time to diagnosis and improve care for patients with chronic orthostatic intolerance.

KEYWORDS

POTS, Postural Orthostatic Tachycardia Syndrome, Orthostatic Intolerance, Non-pharmacological Management, Primary Care

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Introduction

Postural orthostatic tachycardia syndrome (POTS) is a disorder of the autonomic nervous system. It is marked by an excessive rise in heart rate on standing, together with symptoms of orthostatic intolerance, but without the fall in blood pressure typical of other conditions [1]. Diagnosing POTS requires a full clinical assessment and careful separation from the common causes of palpitations and dizziness seen in primary care, including dehydration, drug side effects and other arrhythmias [2]. POTS is linked to a substantial loss of quality of life, comparable to that seen in other chronic diseases. For this reason, familiarity with the syndrome among family doctors is essential to avoid misdiagnoses such as anxiety disorder, and to allow early recognition and appropriate management of the patient [3].

Definition

Postural orthostatic tachycardia syndrome (POTS) is a disorder of circulatory regulation, marked by chronic orthostatic intolerance and an excessive rise in heart rate on standing (an increase of at least 30 beats per minute in adults and at least 40 beats per minute in adolescents) without an accompanying meaningful fall in blood pressure (that is, a drop in systolic blood pressure of more than 20 mm Hg). Clinically, the syndrome presents with complaints that worsen on standing and ease after lying down. These include, among others, dizziness, palpitations, a sense of trembling, general weakness, blurred vision, exercise intolerance and fatigue. Diagnosis also requires the absence of other obvious causes of sinus tachycardia, such as acute physiological stimuli, dietary factors, and other illnesses or medications [2].

Epidemiology

POTS is estimated to affect about 0.2% of the population, and the true prevalence may be higher because the condition is under-recognised and its symptoms are often attributed to psychological causes [2, 4, 5]. Although the syndrome can occur in men and women of any age, it affects young women far more often. It is most commonly diagnosed between the ages of 15 and 25 [2, 6]. POTS is now regarded as one of the more frequent causes of chronic orthostatic intolerance in young adults. Since the COVID-19 pandemic, an increase has also been observed in the number of diagnoses of POTS and other forms of orthostatic intolerance, particularly in young women [6, 7]. The onset of symptoms is sometimes preceded by a viral infection, including SARS-CoV-2, or by other stressful events such as trauma, surgery or periods of hormonal change [4, 8].

Aetiology and Pathogenesis

The causes of postural orthostatic tachycardia syndrome are multifactorial and varied. Rather than a single cause, several mechanisms can be identified, which may dominate to differing degrees in individual patients. One important factor is a partial autonomic neuropathy with impaired constriction of the veins in the lower limbs, which encourages blood to pool on standing and leads to a secondary, reflex rise in heart rate [9]. A second commonly described element is hypovolaemia and disordered regulation of plasma volume, sometimes with abnormal activity of the renin-angiotensin-aldosterone system, which further worsens tachycardia and orthostatic intolerance [10, 11]. In some patients a hyperadrenergic phenotype predominates, with raised standing noradrenaline levels and signs of excessive sympathetic activation such as tremor, anxiety or excessive sweating [1, 2, 10]. A growing body of evidence also points to a role for autoimmune mechanisms. Antibodies against adrenergic and muscarinic receptors have been demonstrated, alongside frequent coexistence with other autoimmune diseases [12].

Clinical Presentation

The clinical picture of POTS is varied, but the profile of a young woman with chronic orthostatic intolerance is strikingly typical [4, 13]. Patients most often report dizziness, a feeling of “drifting away”, palpitations and pronounced weakness that builds on standing and eases after lying down [14]. A large proportion describe “brain fog” – difficulty concentrating, slowed thinking and problems with short-term memory [9]. In survey studies, the most frequent complaints are lightheadedness, tachycardia, presyncope, headaches and difficulty concentrating [3]. POTS may also be accompanied by gastrointestinal symptoms such as bloating, diarrhoea and abdominal pain [13]. Patients further report musculoskeletal pain, sleep disturbance, a sensation of cold, and bluish discoloration of the lower limbs, which reflects venous pooling [9]. Some patients experience syncope, though presyncope is far more common. POTS is associated with a substantial, multidimensional reduction in quality of life, especially in young women. The conditions that most often coexist with POTS are disorders within the dysautonomia spectrum and connective tissue diseases, in particular migraine and Ehlers-Danlos syndrome, as well as irritable bowel syndrome and chronic fatigue. Coexistence with autoimmune diseases such as Hashimoto’s disease and systemic lupus erythematosus is also seen [15]. In addition, some patients are diagnosed with anxiety disorders, which may either coexist or initially be regarded as the main cause of symptoms [4]. Orthostatic complaints, chronic fatigue, pain, sleep disturbance and brain fog all restrict patients’ working, educational and social lives. Because these symptoms appear at the peak of life’s activity, they are especially disruptive to the psychosocial functioning of this group [16].

Diagnosis

In the primary care office, the diagnosis of POTS should be kept as simple as possible and built on a few key steps: the history, the physical examination, basic measurements, and a decision about whether to refer for specialist testing [4, 14]. In the medical history, the doctor should systematically assess how the complaints relate to body position (standing, sitting, lying), how long they take to build after standing, and the typical triggers, such as heat, dehydration, exertion, stress or large meals [4, 13]. It is important to gather information about coexisting conditions (including autoimmune diseases, connective tissue diseases, and anxiety and depressive disorders) and about any medications that might worsen tachycardia or low blood pressure [4, 8]. During the physical examination, beyond a general assessment of the cardiovascular and respiratory systems, attention should be paid to signs of joint hypermobility, features of neuropathy, and signs

of nutritional deficiency [4, 14]. When time is limited, the family doctor can perform a simplified orthostatic test: measuring pulse and blood pressure after a few minutes of lying down, and then, ideally several times, over 10 minutes of standing. A sustained rise in heart rate of at least 30 beats per minute in adults (at least 40 in adolescents), with no meaningful fall in blood pressure and with typical orthostatic symptoms lasting at least 3 to 6 months, should raise the suspicion of POTS [2, 6, 13, 14]. At the same time, a basic panel of laboratory tests is recommended to rule out secondary causes of tachycardia, including a full blood count with assessment of iron status, measurement of TSH and thyroid hormones, and electrolytes. A resting ECG is also advisable to exclude other arrhythmias. If the diagnosis remains in doubt, Holter ECG monitoring and echocardiography can additionally be considered. In patients with suspected POTS, further specialist assessment is recommended after the initial evaluation in the family doctor's office, in order to confirm the diagnosis. The key confirmatory investigation is the tilt table test, with continuous monitoring of haemodynamic parameters [6, 8].

Non-pharmacological Treatment

Management of POTS should begin with non-pharmacological measures, which form the foundation of treatment and are recommended as the first step in the major expert consensus statements [2, 8, 10]. The aim is to improve venous return, increase circulating blood volume, and reduce the factors that worsen orthostatic intolerance. Patients are advised to increase their fluid intake (usually to 2 to 3 litres per day) and their salt intake, often to 8 to 10 g of sodium chloride per day, provided there are no cardiac or renal contraindications. These measures are intended to increase plasma volume [10, 17]. Compression garments are recommended – stockings or tights, along with abdominal binders – to reduce blood pooling in the lower limbs [17, 18]. An important part of treatment is a gradual, well-planned exercise rehabilitation programme. Exercise programmes that begin with training in non-upright positions (for example a recumbent bike, a rowing ergometer or swimming) and gradually introduce upright activity have been shown to improve exercise tolerance, reduce orthostatic tachycardia and improve quality of life [17, 19]. Other non-pharmacological recommendations include avoiding prolonged standing, sudden changes in position, hot baths and overheating, large meals and alcohol. It is also important to adjust or, where possible, stop medications that worsen tachycardia or low blood pressure. A further important element of patient education is teaching counter-manoeuvres against fainting (tensing the muscles of the lower limbs, crossing the legs) in situations where syncope is likely [2, 4].

Pharmacological Treatment

Drug treatment of postural orthostatic tachycardia syndrome rests on limited and inconsistent clinical evidence, and most recommendations come from observational studies and small interventional trials [18, 20, 21]. Current systematic reviews show that there is no single universal drug treatment, and that the effectiveness of therapy varies [18, 21]. The most commonly used drugs remain beta-blockers and ivabradine [18, 20]. In recent years, a growing body of evidence has also supported the effectiveness of midodrine [20]. Given the considerable heterogeneity of the clinical picture and of the underlying mechanisms in POTS, treatment should be individualised and directed at the dominant disturbances, such as excessive sympathetic activation, hypovolaemia or vascular dysfunction [4, 8]. The main aim of drug treatment is to reduce orthostatic tachycardia and improve clinical symptoms while preserving exercise tolerance [2, 8]. Drug treatment should complement non-pharmacological measures, including increased fluid and sodium intake, external compression and physical training [2, 11]. Systematic reviews suggest that ivabradine can lower standing heart rate and ease symptoms, although the quality of the available evidence remains moderate [18, 20]. Beta-blockers such as propranolol show the greatest effectiveness in reducing heart rate, particularly in the hyperadrenergic subtype, but they can lower blood pressure and worsen fatigue [4, 18]. In such cases ivabradine offers an alternative. Midodrine may be considered, especially in patients with marked vascular dysfunction and orthostatic hypotension [20]. The choice of treatment should be based on a thorough clinical assessment and on identifying the mechanisms that predominate in the individual patient [8, 11].

Vulnerable Populations

POTS most often begins in adolescence, with a typical age of onset around the early-to-mid teenage years, so the syndrome deserves particular attention in young patients [5, 6]. Because healthy adolescents normally show larger orthostatic rises in heart rate than adults, the diagnostic threshold in this age group is set higher: an increase of at least 40 beats per minute, rather than the 30 used in adults [2, 13]. This higher cut-off is intended to avoid over-diagnosis, although some authors note that the boundary remains debated and that adolescents with smaller rises may report a similar symptom burden [5]. Reassuringly, the course in younger patients is often more favourable, and a meaningful proportion improve as they reach adulthood [4]. The strong predominance of POTS in women of reproductive age points to a contribution from female sex hormones, and many patients describe fluctuations in symptom severity across the menstrual cycle [9, 13]. Hormonal and physiological transitions are also relevant: the onset of POTS is sometimes precipitated by pregnancy, and pre-existing symptoms may change during gestation in either direction [4]. These considerations matter to the family doctor, who should adjust the diagnostic criteria to the patient's age, keep POTS in mind in adolescents with unexplained orthostatic complaints, and counsel women of childbearing age that symptoms may vary with hormonal changes [4, 13].

Prognosis and Natural History

For both the patient and the family doctor, one of the most pressing questions concerns the long-term outlook, yet the natural history of POTS remains poorly explored and the available data are limited [4, 11]. The course is variable and difficult to predict in the individual case. In a frequently cited estimate, around half of patients recover spontaneously within one to three years of onset, particularly younger patients whose symptoms began after a viral infection [4]. This relatively optimistic figure should, however, be set against evidence from long-term follow-up. In a large cohort of patients diagnosed in childhood and adolescence, the mean duration of symptoms reached almost ten years, and the syndrome rarely resolved completely; the great majority of patients continued to require non-pharmacological measures to control their symptoms over the long term [5]. POTS should therefore be regarded as a chronic, often relapsing disorder, in which periods of relative stability may be interrupted by flares triggered by infection, heat, hormonal change or physical deconditioning [4, 13]. An important prognostic factor appears to be the time from symptom onset to the start of treatment: a longer delay before diagnosis and management is associated with poorer long-term outcomes, which underlines the value of early recognition in primary care [5, 11]. Sustained adherence to non-pharmacological treatment, especially a graded exercise programme that counteracts deconditioning, is linked to better functional recovery [4]. Because the family doctor usually accompanies the patient over many years, primary care is well placed to monitor the course of the illness, reinforce self-management, identify relapses early, and provide continuity of support. Setting realistic expectations from the outset - explaining that meaningful improvement is common but often gradual, and that complete remission cannot be guaranteed - can itself reduce anxiety and strengthen engagement with treatment [5].

Conclusion

Postural orthostatic tachycardia syndrome is a common, though still under-recognised, cause of chronic orthostatic intolerance. It typically affects young adults, especially women, leading to a marked reduction in quality of life and significant psychosocial consequences. In primary care practice, POTS should be considered in the differential diagnosis of recurrent dizziness, palpitations, presyncope and chronic fatigue, particularly when the symptoms clearly depend on body position and worsen on standing. Simple measurements of blood pressure and heart rate while lying and standing, supported by a basic laboratory panel and an ECG, allow a reasonable suspicion of POTS that warrants further specialist assessment. At the same time, it is essential to recognise coexisting conditions, including anxiety disorders, autoimmune diseases and connective tissue disorders. Treatment should always begin with comprehensive non-pharmacological measures: increased fluid and sodium intake, compression garments and exercise training. Drug treatment, based on limited but growing evidence, should be chosen individually with regard to the dominant pathophysiological mechanisms. Beta-blockers, ivabradine and drugs that increase vascular tone can bring a substantial reduction in symptoms. Awareness of POTS among family doctors, the ability to recognise the typical picture early, and timely referral to specialist centres are all key to shortening the time to diagnosis and starting treatment, which may translate into a better quality of life for the young patients affected by this syndrome.

Conflicts of Interest: No conflicts of interest to declare.

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