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Review article

Current look on postural tachycardia syndrome



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ARTICLE INFO

Article history:

Received 26 May 2015

Received in revised form

20 August 2015

Accepted 25 August 2015

Available online 26 September 2015

Keywords:

POTS

Orthostatic intolerance

Pathophysiology

Head-up tilt test

Syncope

Diagnostics

Management

Treatment

ABSTRACT

Introduction: Postural tachycardia syndrome (POTS) is one of the common causes of orthostatic intolerance. It is a pathophysiologically heterogeneous disorder characterized by orthostatic intolerance and an increase of heart rate by 30 bpm in the first 10 min after standing up or during head-up tilt test (HUTT) without orthostatic hypotension.

Clinical features: Symptoms accompanying this syndrome are those due to cerebral hyperperfusion and due to sympathetic hyperactivity and consist of orthostatic, nonorthostatic and diffuse associated symptoms.

Classification: For this heterogeneous group of disorders, many different classifications have been proposed. The most practical classification consistent with current medical evidence and in terms of clinical usefulness seems to be the classification based on POTS phenotypes: partial dysautonomic (neuropathic) form of POTS, hyperadrenergic POTS, POTS associated with poor conditioning, and POTS and volume dysregulation.

Diagnosis and examinations: Systematic and practical approach is essential to properly manage POTS. The patient should undergo a thorough cardiac and neurologic examination including EKG Holter monitoring and ambulant blood pressure monitoring. Screening HUTT has been shown to be helpful in the evaluation of patients with syncope of unknown cause, including POTS.

Differential diagnosis: The clinical manifestation of POTS can be similar to pheochromocytoma, vasovagal syncope, inappropriate sinus tachycardia and other supraventricular tachyarrhythmias. In all patients with signs of autonomic neuropathy, an underlying cause of neurological deficit, deficit should be diagnosed, compensated and ruled out as a primary cause.

Treatment: The treatment of POTS is mainly non-pharmacological. All patients need to be educated and need to understand POTS. Structured exercise program can have huge benefits. Pharmacological treatment is supportive and aims for volume expansion, sympatholysis, vasoconstrictor and increasing vagal tone.

Conclusion: Although POTS was described in the 1990s, many aspects of this multisystemic orthostatic disorder still remain unclear.

This syndrome significantly affects the patient's quality of life. It is necessary to broaden the general knowledge of first-line doctors in private and specialized practices to better manage the patients suffering from POTS and to refer them to specialized centers with available comprehensive diagnostic options and care.

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Contents

Introduction	e427
Pathophysiology	e427
Clinical features	e427
Classification	e427
Diagnosis and examinations	e428
Differential diagnosis	e428
Treatment	e428
Non-pharmacological	e428
Pharmacological treatment	e429
Conclusion	e429
Conflict of interest	e429
Ethical statement	e429
Funding body	e429
References	e429

Introduction

Postural tachycardia syndrome (POTS) is one of the common causes of orthostatic intolerance. It is a pathophysiologically heterogeneous disorder characterized by orthostatic intolerance and an increase of heart rate by 30 bpm in the first 10 min after standing up or during head-up tilt test (HUTT) without orthostatic hypotension [1,2]. Most commonly, patients between 15 and 25 years are affected. POTS is more frequent in women than men (female:male ratio, 4.5:1). Up to 50% of patients had a previous viral illness, and 25% have a family history of similar complaints [3–5]. The exact prevalence of POTS is unknown and it is most likely underdiagnosed. In clinical practice, it is probably 5–10 times more frequent than orthostatic hypotension. One estimate is that the prevalence is at least 170/100,000 population [6].

Pathophysiology

Assumption of the upright posture results in redistributing around 500–1000 ml of blood from the thorax to the lower abdomen and the lower extremities. Due to lower preload, baroreceptors are triggered and there is a compensatory sympathetic activation to counter the initial drop of blood pressure. The pathophysiology of POTS is heterogeneous and includes impaired sympathetically mediated vasoconstriction, excessive sympathetic drive, volume dysregulation, and deconditioning [1].

Clinical features

Symptoms accompanying this syndrome are those due to cerebral hypoperfusion and due to sympathetic hyperactivity and consist of orthostatic, nonorthostatic, and diffuse associated symptoms. Orthostatic symptoms are mainly vertigo, light-headedness, palpitations, presyncope, sense of weakness, and tremulousness. Nonorthostatic symptoms include a wide variety of gastrointestinal or bladder disorders. Diffuse

associated symptoms include sleep disturbance, flushing, fatigue, cognitive and chronic headache, chest discomfort, somatic hypervigilance associated with anxiety, depression and behavioral amplification which contribute to symptom chronicity [5]. Secondary neurally mediated vasovagal syncope can occur only in a minority of patients. Chest pain is almost never associated with coronary artery obstruction.

Classification

For this heterogeneous group of disorders, many different classifications have been proposed. The most practical classification consistent with current medical evidence and in terms of clinical usefulness seems to be the classification based on POTS phenotypes: partial dysautonomic (neuropathic) form of POTS, hyperadrenergic POTS, POTS associated with poor conditioning, and POTS and volume dysregulation [1]. Partial dysautonomic (neuropathic) form is the most frequent form of primary POTS. This condition is characterized by loss of sweating on the feet on thermoregulatory sweat tests and quantitative sudomotor axon reflex testing and impair of noradrenalin release in the lower limb in response to verticalization [7]. Patients suffering from this form of POTS most likely suffer from peripheral autonomic neuropathy and cannot maintain adequate peripheral vasoconstriction to compensate for the sudden gravitational stress [8]. This triggers the compensatory mechanisms including both sympathetic reaction and skeletal muscle pump. This sympathetic reaction also affects the heart and symptoms occur. The extent of venous pooling may continue and patients become more and more dependent on skeletal muscle pump [9]. The venous pooling may eventually overcome this mechanism as well. Due to many patients reporting that their problems occur after acute febrile illness, pregnancy, surgery, trauma or sepsis, and because of the presence of a ganglionic acetylcholine receptor antibody in 14% of patients, an autoimmune cause may be suggested in some cases [10].

Another, less frequent form of POTS is the hyperadrenergic form [11]. In this group of patients, there is often orthostatic hypertension in addition to orthostatic tachycardia. Some of

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