Analysis of Heart Rate Variability During Head-Up Tilt Testing in a Patient With Idiopathic Postural Orthostatic Tachycardia Syndrome (POTS)

Masataka Sumiyoshi, MD; Yasuro Nakata, MD*; Yoriaki Mineda, MD; Masayuki Yasuda, MD; Yuji Nakazato, MD; Hiroshi Yamaguchi, MD

A 16-year-old boy was diagnosed with idiopathic postural orthostatic tachycardia syndrome (POTS) during head-up tilt testing. During a passive tilt, the patient's heart rate (HR) increased by 30 beats/min within 5 min. After 25 min of tilting, his HR further increased to 133 beats/min and he began to complain of lightheadedness and weakness without hypotension. Power spectral analysis of HR variability during the tilt test revealed that the ratio of low and high frequency powers increased with the onset of orthostatic intolerance. Propranolol (10 mg every morning) dramatically alleviated his clinical symptoms, and he has been asymptomatic with gaining weight after discontinuing his crowded train commuting. (Jpn Circ J 1999; 63: 496–498)

Key Words: Beta-blocker; Head-up tilt testing; Heart rate variability; Orthostatic intolerance; Postural orthostatic tachycardia syndrome

Idiopathic postural orthostatic tachycardia syndrome (POTS) has been reported as a mild form of orthostatic intolerance! Recently, POTS was reported to be diagnosable by head-up tilt testing. We report a case of POTS in which we analyzed heart rate (HR) variability during passive tilt testing.

Case Report

A 16-year-old high school boy was referred to hospital for orthostatic intolerance. He had complained of lightheadedness, weakness, and disabling fatigue when in an upright posture since the age of 14 years. He had not experienced any preceding viral illnesses. His symptoms deteriorated after he was admitted to high school and began to commute by train. He experienced presyncope on several occasions in the morning while standing in crowded trains, but syncope never developed. An alpha-agonist (midodrine, 2 mg bid) was not effective for his symptoms. When he came to the outpatient clinic, he was a thin boy with a height of 165 cm and body weight of 50 kg. Physical examination was normal except for hypotension of 86/50 mmHg in the sitting position. The resting 12-lead electrocardiogram showed a normal sinus rhythm of 65 beats/min without any conduction abnormalities or ST-T changes. Neither chest X-ray nor echocardiographic examination revealed any cardiac abnormalities. Hematological examination, blood biochemical analysis, urinary analysis, and thyroid function were all normal. After he and his mother gave informed consent, we performed a head-up tilt test to evaluate his orthostatic intolerance. The tilt test was undertaken in a quiet room in a fasting state. Electrocardiogram and arterial blood pressure were monitored continuously and noninvasively by a tonometry system (BP-508, Colin Electronics, Komaki, Japan). A Holter electrocardiogram was also recorded during the tilt test. After resting for 15 min in the supine position, he was positioned upright at an angle of 80 degrees on a tilt table with a footboard for weight bearing. The passive tilt was performed for a maximum of 30 min. His HR was 84 beats/min and blood pressure was 95/46 mmHg in the supine position. After 5 min of the tilting, his HR increased to 114 beats/min without hypotension. After tilting for 25 min, his HR further increased to 133 beats/min and he began to complain of lightheadedness and weakness, but hypotension did not develop. At the completion of the passive tilt test for 30 min, he had not experienced syncope or marked hypotension (Fig 1). To clarify the autonomic state during the head-up tilt testing, power spectral analysis of HR variability was performed using the Space Labs FT 2000 monitoring system (Redmond, WA). Spectral indices of HR variability were computed by Fast Fourier analysis for each 2-min interval with 1-min overlap during the passive tilt test. The power spectrum was calculated as high frequency (HF, 0.15–0.40 Hz), low frequency (LF, 0.05–0.15 Hz), and as the ratio of LF to HF power (LF/HF). The changes in HF and LF/HF during the tilt test are shown in Fig 2. The HF component value decreased at the beginning of tilting and remained low during the test. The LF/HF tended to fluctuate, but it reached a higher level after 25 min of tilting when the patient began to experience symptoms of orthostatic intolerance. He was diagnosed as POTS because a HR increase of 30 beats/min was obtained within the first 5 min of tilting without profound hypotension or any other evidence of possible causes. After he was prescribed propranolol 10 mg every morning, his clinical symptoms of orthostatic intolerance dramatically improved and he was able to commute to high school by train. We also added midodrine 2 mg bid for hypotension. Later, after graduating from high school and discontinuing his crowded train commute by train.
commuting, he gained 6 kg and his symptoms of orthostatic intolerance resolved spontaneously. He has been asymptomatic for 6 months without any medication.

**Discussion**

POTS is defined as the development of orthostatic symptoms associated with idiopathic postural tachycardia (a heart rate increment $\geq 30$ or an orthostatic HR $\geq 120$ beats/min) without orthostatic hypotension. Recently, Grubb et al reported the usefulness of head-up tilt testing for detecting POTS patients. During passive head-up tilt testing, the development of orthostatic symptoms associated with a heart rate increment of more than 30 beats/min or a HR of more than 120 beats/min within 5 or 10 min of being upright without profound hypotension is diagnosed as POTS. However, dynamic changes in HR variability during tilt testing have been unclear in patients with POTS, especially in association with symptoms of orthostatic intolerance. In the present case, LF/HF increased in parallel with the development of orthostatic intolerance without any changes in the HF value. This change in LF/HF may represent a hyperactivity of beta-receptors. The suggested mechanisms of POTS have included hypovolemia, excessive venous pooling while standing, loss of adequate vascular tone in the lower extremities, and beta-adrenergic hypersensitivity of cardiac receptors. Recently, Furlan et al proposed an increased noradrenergic tone at rest and a blunted postganglionic sympathetic response to standing with compensatory cardiac sympathetic overactivity. Attenuated post-viral panautonomic neuropathy also has been proposed as a cause of dysautonomia. Although no previous viral illness was identified in the present patient, beta-receptor hypersensitivity may have played an important role in his orthostatic intolerance because this symptom was alleviated by a small dose of beta-blocker (propranolol 10 mg daily). In addition, because propranolol is a non-selective beta-blocker, some vasoconstriction may also occur. Interestingly, the clinical symptoms due to POTS were relieved spontaneously when the patient gained weight and no longer had to stand in a crowded train. Weight gain, which is a physiological change that presumably increases the circulating blood volume, may help prevent orthostatic intolerance.

We could not diagnose POTS before tilt testing, because the symptoms of orthostatic intolerance were compatible with neurally mediated syncope. Although there is considerable overlap in the symptoms between POTS and neurally mediated syncope, the blood pressure response in each of these conditions is different. Patients with POTS are symptomatic in the absence of significant blood pressure changes in contrast with patients with neurally mediated syncope in whom symptoms are usually associated with hypotension. It may be due to the different degrees of reflex neural inhibition between POTS and neurally mediated syncope. In POTS, the potentiated tachycardia may induce a partial and selective reflex inhibition of muscle sympathetic nerve activity. Blood pressure is maintained by the faster HR. In neurally mediated syncope, postural change may elicit a more potent neural inhibitory reflex.
that results in hypotension and bradycardia. Therefore, as previously described, head-up tilt testing is useful for differentiating POTS from neurally mediated syncope.

In conclusion, we have reported a case of POTS in which sympathetic tone was strongly activated during a passive tilt with concomitant development of orthostatic intolerance. However, the patient’s orthostatic symptoms have been relieved by a change in his daily life-style and a 6-kg weight gain.

References

2. Grubb BP, Kosinski DJ, Boehm K, Kip K: The postural orthostatic tachycardia syndromes: A neurocardiogenic variant identified during head-up tilt table testing. PACE 1997; 20: 2205–2212