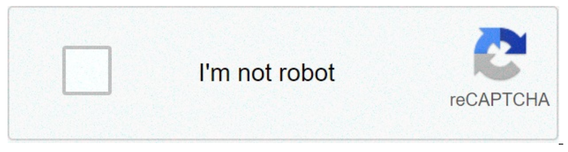

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Orthostatic hypotension in child

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Logistic regression analysis revealed that low supine SBP and headache were independent risk factors for OHT in POTS patients.Conclusions: Headache and low supine SBP were the main clinical characteristics of a novel syndrome of POTS associated with OHT. autonomic nervous system, tachycardia, orthostatic hypertension, children Postural tachycardia syndrome presented orthostatic intolerance symptoms from supine to upright posture and relief of these symptoms with recumbence. Along with raising the level of recognition of the disease, the incidence of postural tachycardia syndrome has been increasing in children and adolescents, with serious consequences for their health [1–3]. The characteristic symptoms of postural tachycardia syndrome are palpitations, light-headedness, chest discomfort, shortness of breath and exercise intolerance, and this disorder can negatively affect an individual's physical and emotional well-being [1–4]. Although postural tachycardia syndrome has unclear pathophysiology increasing fluid and salt intake show beneficial effects on postural tachycardia syndrome in children and adolescents [1–7].Regulation to orthostatic challenge of human body is not only associated with the change of heart rate (HR) but also blood pressure (BP). The most common disorder about changes of BP for orthostatic stress is orthostatic hypotension. This disorder has been widely recognized. In contrast, 'orthostatic hypertension' (OHT), which is an increase in BP upon standing, has been infrequently reported. OHT is defined as BP increase when upright, but precise criteria have not been established [8–10]. OHT is a clinically important problem that is being increasingly recognized because of its association with the progressive target-organ damage and subsequent cardiovascular risk. OHT precedes hypertension and could be considered to be prehypertension [8–13]. Postural tachycardia syndrome and OHT are impairments of circulatory adjustments against gravitational stress. Postural tachycardia syndrome patients were often reported to have great changes in HR, but changes in BP have been largely ignored. Although some reports have focused on postural tachycardia syndrome with concomitant hypotension, few studies have explored postural tachycardia syndrome with concomitant OHT. The only reports on postural tachycardia syndrome and OHT were about patients with pheochromocytoma [14] or mast-cell activation disorder in the context of postural tachycardia syndrome [5]. Moreover, the patients in these reports were adults. To our knowledge, our group is the first to recognize the association between postural tachycardia syndrome and OHT in children and adolescents [15]. The concomitant occurrence of postural tachycardia syndrome with OHT raises an important clinical concern. The link between salt and hypertension is well-established [16–18]. Postural tachycardia syndrome patients with concomitant OHT pose a great challenge to the traditional treatment of postural tachycardia syndrome, since salt supplements must be used with caution in these patients. Here, we described a heterogeneous group of children and adolescents with postural tachycardia syndrome and evident OHT. Methods Subjects This retrospective study involved 96 children and adolescents (58 girls and 38 boys) who were diagnosed with postural tachycardia syndrome in the Department of Pediatrics, Peking University First Hospital, between March 2008 and March 2013. All patients completed our standard questionnaire of symptoms of orthostatic intolerance by face-to-face interview. Patients provided primary information. Parents or guardians assisted with answers as needed. All patients did not have family history of hypertension, stroke or postural tachycardia syndrome.The patients were divided into a postural tachycardia syndrome group (n = 76; 48 girls and 28 boys) and a postural tachycardia syndrome with OHT group (n = 20; 10 girls and 10 boys) by using the head-up tilt test. The mean ages of the patients in the postural tachycardia syndrome and postural tachycardia syndrome with OHT groups were 11 ± 2 years and 11 ± 2 years, respectively. We also included a control group of 20 healthy age-matched children and adolescents (11 girls and 9 boys) during the same period of patient recruitment. None of the control subjects had symptoms of orthostatic intolerance, and they all had normal results on physical examination and routine electrocardiography. They also underwent the same protocol of head-up test in our syncope study unit. The study adhered to the principles of the Declaration of Helsinki. Informed consent was obtained from all individuals and their parents, and the protocol was approved by the ethics committee of Peking University First Hospital, China. Criteria for diagnosis of postural tachycardia syndrome According to the 2011 update of the Consensus of the American Autonomic Society on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome [19], postural tachycardia syndrome with OHT was diagnosed using the quantitative sign of an increase in HR of ≥40 beats/min or a HR of ≥120 beats/min within the first 10 min after standing during the head-up tilt test. All patients had an at least 6 month history of the illness without additional chronic debilitating diseases or prolonged bed rest, and none of them were on medications that could have impaired autonomic tone [1–4, 20]. Symptoms included fatigue, palpitations, light-headedness, shortness of breath, headache, near-syncope and syncope. Criterion for diagnosis of OHT OHT was diagnosed in patients who showed an increase of ≥20 mm Hg in systolic blood pressure (SBP) in 3 min after standing during the head-up tilt test [8, 10, 21]. Pheochromocytoma was ruled out in all the patients through a detailed examination, and other causes of hypertension like coarctation of aorta or other endocrine diseases were also excluded in the study. Protocol for head-up test During head-up test, BP and HR were measured at intervals of 3 min at the baseline and after the subjects had been in a supine position for at least 10 min. The subjects including patients and controls were positioned upright. BP and HR monitoring and electrocardiography were performed simultaneously during the test by using a Dash 2000 Multileads Physiological Monitor (GE Company, NY, New York, USA), which was standardized before the use. The position of the cuff during the measurement of BP was made at heart level. The test was stopped within 10 min. Protocol for head-up tilt test The method used for head-up tilt test was described elsewhere [22]. All data were gathered between 8:00 am and 9:00 am with subjects in a fasting state. Following BP and HR measurement at intervals of 1 min at the baseline for 15 min, they were positioned upright on the tilt table at an angle of 60°, with a footboard being used to bear the subject's weight [22]. BP and HR monitoring and electrocardiography were performed simultaneously during the test, with a Dash 2000 Multileads Physiological Monitor (GE Company, NY, New York, USA). The position of the cuff during the measurement of BP was made at heart level. The test was stopped within the first 10 min for those patients meeting postural tachycardia syndrome criteria, or if syncope or presyncope with hypotension developed during the test, the table was changed to the supine position. Statistical methods Statistical analysis was conducted with a SPSS software, version 13.0 (SPSS, Chicago, Illinois, USA). The data are presented as the mean ± standard deviation. Group comparisons were done with the nonparametric Kruskal–Wallis test. Post hoc analysis between two groups was performed with the nonparametric Mann–Whitney test. Logistic regression analysis was used to identify independent predictors for postural tachycardia syndrome with OHT. A p value less than 0.05 was regarded statistically significant. Results Table 1 summarizes the clinical features of the patients in the postural tachycardia syndrome, postural tachycardia syndrome with OHT and control groups. Ninety-six postural tachycardia syndrome patients were included in this study, and 20.83% (20) of them were found to have OHT. There was no difference in age, sex and body mass index among the postural tachycardia syndrome, postural tachycardia syndrome with OHT and control groups. Table 1Baseline characteristics and hemodynamic parameters in normal controls and patients of POTS and POTS + OHT groups Items . Control (n = 20) . POTS (n = 76) . POTS + OHT (n = 20) . p . Age (years) 11 ± 1 11 ± 2 11 ± 2 0.089 Sex ratio (M/F) 9/11 27/48 10/10 0.512 BMI, kg/m2 18.53 ± 3.54 19.15 ± 4.18 18.00 ± 2.61 0.458 Supine SBP (mm Hg) 105.20 ± 9.43 102.90 ± 7.78 98.20 ± 7.03*, ** 0.018 Supine DBP (mm Hg) 58.05 ± 4.27 55.38 ± 9.43 54.45 ± 6.57 0.344 Supine HR (beats/min) 77.15 ± 7.06 72.96 ± 9.55 76.65 ± 9.31 0.091 Standing SBP (mm Hg) 114.50 ± 8.84 110.67 ± 8.93 122.00 ± 7.11*, ** 0.000 Standing DBP (mm Hg) 66.70 ± 4.35 64.76 ± 8.64 68.40 ± 10.50 0.201 Standing HR (beats/min) 100.40 ± 12.61 118.61 ± 9.46* 126.15 ± 8.79*, ** 0.000 ΔHR (beats/min) 23.25 ± 12.05 45.66 ± 4.55* 49.50 ± 8.95*, ** 0.000 Hemodynamic responses to posture are shown in Table 1 and Figure 1. No significant differences was found in the supine diastolic blood pressure (DBP), standing DBP and supine HR among the postural tachycardia syndrome, postural tachycardia syndrome with OHT and control groups. However, supine SBP was lower in the postural tachycardia syndrome with OHT group than in the postural tachycardia syndrome and control groups ($p < 0.05$). As expected, the upright HR was significantly higher in the postural tachycardia syndrome with OHT and postural tachycardia syndrome groups than in the control group ($p < 0.05$), and the standing SBP was significantly higher in the postural tachycardia syndrome with OHT group than in the postural tachycardia syndrome and control groups ($p < 0.05$). Interestingly, the upright HR and HR changes were significantly greater in the postural tachycardia syndrome with OHT group than in the postural tachycardia syndrome and control groups ($p < 0.05$). Open in new tabDownload slideHemodynamic effect of posture in normal children and adolescents and patients with POTS and POTS + OHT. Postural change in heart rate (HR;•) and systolic/diastolic blood pressure (floating bars). *$p < 0.05$ vs. Control group, **$p < 0.05$ vs. POTS group.No significant difference was found in the frequency of syncopal attacks between the postural tachycardia syndrome group and the postural tachycardia syndrome with OHT group ($p > 0.05$). Dizziness was the most common symptom in both groups. Headache was more common in the postural tachycardia syndrome with OHT group than in the postural tachycardia syndrome group ($p < 0.05$). Other symptoms such as shortness of breath, gastrointestinal symptoms, fatigue, pallor, blurred vision, palpitations, sweating and tremulousness did not significantly differ between the postural tachycardia syndrome and postural tachycardia syndrome with OHT groups. Table 2 summarizes the symptoms encountered in our patient population. Table 2Clinical manifestation of patients with POTS and POTS + OHT Orthostatic symptoms . POTS (n = 76) . POTS + OHT (n = 20) . p . Frequency of syncope (times) 3.7 ± 3.7 3.8 ± 3.3 0.887 Dizziness, n (%) 56 (73.7) 11 (55.0) 0.105 Headache, n (%) 24 (31.6) 13 (65.0)* 0.006 Shortness of breath, n (%) 42 (55.3) 9 (45.0) 0.413 Gastrointestinal symptoms, n (%) 37 (48.7) 6 (30.0) 0.135 Fatigue, n (%) 47 (61.8) 8 (40.0) 0.079 Pallor, n (%) 32 (42.1) 10 (50.0) 0.527 Blurred vision, n (%) 35 (46.1) 7 (35.0) 0.375 Palpitations, n (%) 16 (21.1) 4 (20.0) 0.918 Sweating, n (%) 24 (31.6) 7 (35.0) 0.771 Tremulousness, n (%) 12 (15.8) 3 (15.0) 0.931 To identify the clinical features that influenced the occurrence of OHT in the postural tachycardia syndrome patients in our study, we used stepwise regression analysis. Logistic regression analysis revealed two factors that were independently associated with OHT in postural tachycardia syndrome patients, namely, supine SBP (odds ratio, 0.856; 95% confidence interval: 0.766–0.958; $p < 0.05$) and headache (odds ratio, 4.527; 95% confidence interval: 1.212–16.908; $p < 0.05$; Table 3). Table 3Multiple logistic regression analyses to assess the associations between the factors with POTS + OHT Indices . B . OR (95%CI) . p value . Supine SBP −0.155 0.856 (0.766–0.958) 0.007 Headache 1.510 4.527 (1.212–16.908) 0.025 Discussion Our study involved a heterogeneous group of children and adolescents with postural tachycardia syndrome. Some of the postural tachycardia syndrome patients also showed a significant increase in BP on assuming an upright posture, which indicated OHT. The concomitant occurrence of postural tachycardia syndrome and OHT is of clinical importance. Studies have shown that OHT might be predictive of essential hypertension [13]. We identified the main clinical features of the postural tachycardia syndrome and OHT patients in our study. In addition to a significant increase in BP upon assuming an upright posture, the patients also showed a significant increase in HR after changing from a supine position to an upright posture. They also tended to have low supine SBP. Headache was more frequent in patients with postural tachycardia syndrome with OHT than in patients with postural tachycardia syndrome alone. Multivariate analysis showed that low supine SBP and headache were independent factors that predicted the occurrence of OHT in postural tachycardia syndrome patients. Thus, in postural tachycardia syndrome patients with relatively low supine SBP or headache, we should determine the upright BP during head-up tilt test.The mechanisms for postural tachycardia syndrome, which is a common clinical problem in children and adolescents, have been investigated in recent years [1–7]. Relative hypovolemia is an important cause of postural tachycardia syndrome, and strategies to increase blood volume by increasing fluid and salt intake have proven effective in children and adolescents [1–7, 23]. Many centers recommend salt supplementation as the first-line treatment for postural tachycardia syndrome [1–7].OHT, which is characterized by a rise in BP upon assuming an upright posture and is an important clinical condition, has been gaining increasing research attention in recent years [8–13]. Studies have shown that OHT may be predictive of essential hypertension. The Coronary Artery Risk Development in Young Adults (CARDIA) study assessed the relationship between positional BP change and incidence of hypertension in 2781 young adults who were followed up for 8 years [13]. This study found that OHT patients had a significantly increased risk of developing hypertension. OHT patients were also at risk of cerebral infraction and target-organ damage [8, 12, 24]. OHT was considered as the last hemodynamic frontier [9].Patients with OHT have a high risk of developing hypertension as young adults. The link between salt and hypertension is well-established [16–18], and the restriction of dietary sodium has been demonstrated to decrease BP [25, 26]. Thus, patients with OHT should reduce salt intake. However, as mentioned above, salt supplementation is the first-line treatment in postural tachycardia syndrome patients. This poses a great challenge for the treatment of patients with concomitant postural tachycardia syndrome and OHT. Salt supplementation should be undertaken with caution in these patients.The mechanisms responsible for postural tachycardia syndrome accompanied with OHT were unclear. OHT with an increase in HR when upright is most frequently seen in patients with pheochromocytoma, which is a rare catecholamine-secreting neuroendocrine tumor [14]. In our study, we excluded this condition in all our patients. OHT also occurs in some forms of autonomic dysfunction, which has long been recognized in adult patients. Robertson reported that more than 20% of the postural tachycardia syndrome patients in their center also had OHT [10]. Norepinephrine transporter deficiency, another rare syndrome, has also been associated with an increase in BP and tachycardia with upright posture [27]. Recently, our study group found that the plasma antidiuretic hormone (ADH) level of patients suffering from postural tachycardia syndrome with OHT was much higher than that of children having postural tachycardia syndrome without OHT, which suggested that ADH played a role in the pathogenesis of the condition [15]. Interestingly, Shibao et al. found that 38% of patients who meet the criteria for both postural tachycardia syndrome and disordered mast-cell activation had BP elevation after standing [5]. However, the patients in the above studies were adults. The present study included children and adolescents with postural tachycardia syndrome, and we found that approximately 20% of postural tachycardia syndrome patients had OHT. Thus, this result was similar to those observed in studies of adult postural tachycardia syndrome patients. In our study, compared with patients who had postural tachycardia syndrome alone, those with postural tachycardia syndrome accompanied with OHT showed not only a significant increase in BP after assuming an upright posture but also a significant increase in HR. This phenomenon may indicate that centrally driven, abnormal sympathetic activation may play a direct role in patients with postural tachycardia syndrome accompanied with OHT. Headache was a prominent symptom in patients suffering from postural tachycardia syndrome accompanied with OHT in our study, which also indicated that patients suffering from postural tachycardia syndrome accompanied with OHT had abnormal sympathetic activation, because sympathetic hyperactivation was postulated in the central sensitization which might take part in the augmented pain experience [28]. However, this hypothesis should be investigated further, and whether these patients show mast-cell activation needs to be determined.The present study has limitations, however, including a relatively short follow-up period. The long-term prognosis of patients suffering from postural tachycardia syndrome accompanied with OHT remains unclear. In addition, we did not measure serum catecholamine levels and urine methylhistamine levels in these patients. These tests are the objectives of an ongoing study.We reported a novel syndrome of postural tachycardia syndrome accompanied with OHT in children and adolescents. In terms of clinical manifestations, this syndrome should be considered in postural tachycardia syndrome patients with a history of frequent headaches and relatively low supine systolic BP. Accurate diagnosis is very necessary since OHT needs a different treatment modality. Salt supplementation, the first-line treatment for postural tachycardia syndrome, should be used with caution because of the risk of developing hypertension. β-Blockers may be the best choice of treatment in these patients. Funding This research was supported by Beijing Municipal Science and Technology Commission, China (D121107001012051) and the Major Basic Research Project of China (2012CB517806). Ethical Standards All authors' declaration: All procedures contributing to this work comply with the ethical standards of the national guidelines on human experimentation of China and with the Helsinki Declaration of 1975, as revised in 2008, and have been approved by the ethics committee of Peking University First Hospital, Beijing, China. References 11, , , et al. Orthostatic hypertension detected by self-measured home blood pressure monitoring: a new cardiovascular risk fact or for elderly hypertensives, , , vol. (pg. -)12, , , et al. Disorders of orthostatic blood pressure response are associated with cardiovascular disease and target organ damage in hypertensive patients, , , vol. (pg. -)15, , , et al. Changes of atrial natriuretic peptide and antidiuretic hormone in children with postural tachycardia syndrome and orthostatic hypertension: a case control study, , , vol. (pg. -)17, , , et al. 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