Research Article

Postural Orthostatic Tachycardia Syndrome in Young Diabetics

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Abstract

Postural orthostatic tachycardia syndrome is a condition characterized by imbalance in autonomic reactivity leading to exaggerated heart rate and other symptoms of orthostatic intolerance. In adolescents, it is characterized either by a continuous increase in heart rate of \geq 40 bpm as compared to basal heart rate or sustained basal rate of \geq 130 bpm. The objective of the research was to compare the characteristics of adolescent diabetics with postural orthostatic tachycardia

syndrome with the controls.

Methods. Seventy adolescents diagnosed with type 1 diabetes mellitus who were treated at the department of Endocrinology, Government Medical College and Shri Maharaja Hari Singh hospital, Srinagar, J&K, India were selected for the study. Lying to standing test was performed. Heart rate was recorded at the 2^{nd} , 3^{rd} , 5^{th} and 10^{th} minutes. Based on the results of lying to standing test, there were selected 25 diabetic adolescents with postural orthostatic tachycardia syndrome. Their characteristics were compared with age- and sex-matched adolescents using unpaired T test. P< 0.05 was considered significant.

Results. We observed a significantly lower body mass index (p=0.027), as well as a significantly higher fasting blood glucose level (p<0.0001) in diabetics with postural orthostatic tachycardia syndrome.

Conclusion. It may be concluded that lower body mass index and higher fasting blood glucose level may lead to the development of postural orthostatic tachycardia syndrome in adolescents.

Keywords

POTS; adolescents; diabetes mellitus; neuropathy

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Problem statement and analysis of the recent research

Postural orthostatic tachycardia syndrome (POTS) is a form of autonomic imbalance which leads to the symptoms of orthostatic intolerance and is characterized by an exaggerated heart rate (HR) upon standing from a lying position [1]. Although POTS can affect people at any age, it is usually seen in young and middle-aged people [2]. In adults, POTS is defined as an increase in HR of \geq 30 beats per minute (bpm) within 10 minutes upon standing or sustained basal HR of \geq 120 bpm in a recumbent position [3], while in young people (children and adolescents), POTS is characterized by an increase in HR of >40 bpm within 10 minutes upon standing or sustained basal HR of \geq 130 bpm [4, 5]. The characteristic features of orthostatic intolerance such as headache, palpitations, blurring of vision, nausea, light headedness and presyncope are aggravated upon assuming an upright posture [6]. POTS is usually diagnosed by the head-up tilt test (HUTT); however, bedside testing such as lying to standing test (LST) when blood pressure (BP) and HR is recorded at the 2nd, 3rd, 5th and 10th minutes can be used to diagnose it as well [7]. POTS is classified into primary and secondary forms [8]. Primary form is

the developmental form of POTS, partial dysautonomic (PD) form, which mainly affects adolescents at the age of 14 to 16 years and is considered as physiological [9] and hyperadrenergic POTS [9]. In primary POTS, young females are five times more likely to develop POTS as compared to young males [8]. Secondary POTS is attributed to peripheral neuropathy with intact cardiac innervation [9]. The most common cause of secondary POTS is chronic diabetes mellitus (DM) [9]. POTS impairs the flexibility of autonomic control [10]. Its treatment consists in the identification of the cause and the application of appropriate management plan [10].

1. Materials and methods

Having obtained the clearance from the institutional Ethical Committee, we conducted the study in the department of Physiology and Endocrinology, Government Medical College and Shri Maharaja Hari Singh (SMHS) hospital, Srinagar, J&K, India. We recruited 70 adolescents at the age of $(16.85\pm2.84$ years) diagnosed with type 1 DM who were treated as outpatients at the endocrinology department of SMHS hospital. All the subjects were asked to rest in recumbent position for 10 minutes. Basal BP and HR were measured with the help of a

digital sphygmomanometer (OMRON- HEM 8712, Taiwan). The same digital sphygmomanometer was used throughout the procedure. All the patients with DM underwent LST. Subjects were asked to move into a standing posture from a recumbent posture as quickly as possible. Both audio and visual cues were provided to all the subjects, i.e. the examiner asked the subject to stand up in a clear and loud voice and the examiner made a gesture to the patient to stand up by raising his arm. BP and HR were measured at the 2nd, 3rd, 5th and 10th minutes of standing. Subjects were continuously monitored for any symptoms of orthostatic intolerance such as near-syncope. The test was immediately aborted if the subjects reported symptoms of near-syncope or syncope. Based on the results of LST, we found that 25 diabetic adolescents fulfilled the criteria for POTS. Finally, we recruited 25 diabetic adolescents with POTS and diabetes duration of ≥ 2 years at the age of $(16.15 \pm 2.66 \text{ years})$, and 25 age- and sex-matched adolescents (16.62 \pm 2.24 years) for the control group. LST was performed in the control group as well. Characteristics of diabetics with POTS were compared with controls. Biochemical parameters such as fasting glucose level were recorded in all the subjects. Glycaemic control (HbA1c) was measured in patients with DM. Other parameters such as age, height, weight, body mass index (BMI) were calculated as well. Patients or their caretakers/guardians were invited to the Department of Physiology for further investigations and informed written consent was taken from them. Subjects were strictly advised not to take any coffee/tea, drugs that can affect HR such as β -adrenomimetics or β -blockers on the day of investigation. The data were analyzed using GraphPad 6 (GraphPad software, inc. California, USA). To compare the characteristics of two groups, unpaired t-test was used. Mann Whitney test was used for non-parametric data. P<0.05 was considered significant.

2. Results

LST was used to diagnose POTS in all the subjects. HR was recorded at different time intervals. Subjects were divided into two groups: diabetics with POTS and the control group. Characteristics of both groups were compared. We did not observe any significant difference between basal parameters such as resting HR (p=0.142), resting systolic blood pressure (SBP) (p=0.150) and diastolic blood pressure (DBP) (p=0.166) between diabetics with POTS and the control group. There was no significant difference between age (p=0.196); however, we observed that BMI of diabetics with POTS was significantly lower than that in the control group (p=0.027) and fasting blood glucose level was significantly higher in patients with POTS (p<0.0001) (Table 1).

3. Discussion

Very few studies have been carried out to study POTS in young diabetic patients. We conducted this study among Indian adolescents diagnosed with type 1 DM to compare the basal

hemodynamic parameters and other biochemical parameters of diabetics with POTS with age- and sex-matched controls. We found that there was no significant difference in basal BP and age between two groups; however, BMI was significantly lower and fasting blood glucose level was significantly higher in diabetics with POTS as compared to the control group.

One of the important forms of primary POTS is developmental POTS which is observed during adolescence and is attributed to the period of rapid growth and development resulting in temporary autonomic imbalance [9]. We did not observe POTS in the control group. POTS can often be found in diabetics [8]. Our findings are supported by Graham U et al. [11] who carried out the study on patients diagnosed with type 1 DM. They found that there was an orthostatic increase in HR of >50 bpm without any decrease in BP associated with the symptoms of orthostatic intolerance in some patients. They concluded that these patients had POTS. Deb A et al. [12] studied 39 subjects with a complaint of autonomic symptoms. The HUTT confirmed the diagnosis of POTS showing an increase in HR of >30 bpm upon postural change. Jiminez-Cohl P et al. [13] found 15 patients with autonomic symptoms who fulfilled the criteria for POTS. We did not observe any statistically significant difference between basal SBP, basal DBP, basal HR and age of diabetics with POTS vs. the control group. Similar findings were reported by Lewis I et al. [14] who studied 179 patients diagnosed with POTS. They found that the difference between basal SBP (p=0.7)and DBP (p=0.8) of patients with POTS vs. controls was not statistically significant. In this study, we observed that BMI of diabetics with POTS was significantly lower than that in the control group. Similar findings were observed by Li H et al. [15] who studied 54 children with POTS. They found that BMI in children with POTS was significantly lower than that in the control group (p < 0.01). We observed that fasting blood glucose level was significantly higher in diabetics with POTS as compared to controls (p < 0.0001). Although secondary POTS is attributed to DM which may arise as a result of autonomic neuropathy [8], there is no study which emphasizes the effect of increased fasting glucose level in diabetics more so in adolescents.

4. Conclusions

On the basis of this preliminary study, it may be concluded that in diabetics with POTS, lower BMI and increased fasting blood glucose level play an important role in the development of POTS.

5. Prospects for further research

Further studies with a large sample size need to be carried out to study POTS in diabetic adolescents and the factors influencing POTS.

Parameters	Diabetics with POTS (n=25)	Controls (n=25)	p value
Resting HR (bpm)	87.54±11.29	82.40±21.85	0.142
Resting SBP (mmHg)	112 ± 14.03	$109 {\pm} 8.66$	0.150
Resting DBP (mmHg)	$70.14{\pm}8.16$	66.60 ± 12.42	0.166
Age (years)	16.15 ± 2.66	16.62 ± 2.24	0.196
Duration of diabetes (months)	$41.40{\pm}25.81$		
BMI (kg/m ²)	$16.46{\pm}2.51$	19.67 ± 3.37	0.027*
Fasting glucose	247.8±119.4	80.5±15.9	<0.0001***
HbA1c (%)	10.40 ± 4.20		

Table 1. Characteristics of diabetics with POTS versus controls

Notes: The data were analyzed using unpaired t-test and Mann Whitney test. The data were expressed in terms of mean \pm standard deviation.

References

- McDonald C, Koshi S, Busner L, Kavi L, Newton JL. Postural tachycardia syndrome is associated with significant symptoms and functional impairment predominantly affecting young women: a UK perspective. BMJ Open. 2014;4:1-9. DOI: https://doi.org/10.1136/bmjopen-2013-004127
- [2] Schnodorf R, Low PA. Idiopathic postural tachycardia syndrome. Ann Neurol 1990;28:271.
- Kanjwal Y, Kosinski D, Grubb BP. The postural orthostatic tachycardia syndrome: Definition, diagnosis and management. Pacing Clin Electrophysiol. 2003;26:1747-1757. DOI: https://doi.org/10.1046/j.1460-9592.2003.t01-1-00262.x [PMid:12877710]
- [4] Consensus statement on the definition of orthostatic hypotension, neutrally mediated syncope and the postural orthostatic tachycardia syndrome. Autonomic neuroscience: Basic and Clinical. 2011;16:46-48.
- Singer W, Sletten W, Opfer-Gehrking TL, Brands LK, Fischer PR, Low PA. Postural tachycardia in children and adolescents: What is abnormal? J Pediatr. 2012;160(2):222-226. DOI: https://doi.org/10.1016/j.jpeds.2011.08.054
 [PMid:21996154 PMCid:PMC3258321]
- [6] Benrud-Larsen LM, Dewar MS, Sandroni P. Quality of life in patients with postural tachycardia syndrome. Mayo Clinic Proc. 2002;77:531-537. DOI: https://doi. org/10.4065/77.6.531
- [7] Postural Orthostatic Tachycardia Syndrome. Available from: http://www.dysautonomiainternational.org/
- [8] Grubb BP. The postural tachycardia syndrome: When to consider it in adolescents. Family Practice. 2006;28(3):19-30.
- ^[9] Grubb BP, Rowe P, Calkins H. Postural tachycardia orthostatic intolerance and the chronic fatigue syndrome.

Blackwell Publishing Company. 2005:225-244. DOI: https://doi.org/10.1002/9780470994801.ch13

- [10] Agarwal AK, Garg R, Ritch A, Sarkar P. Postural orthostatic tachycardia syndrome. Postgrad Med J. 2007;83:478-480. DOI: https://doi.org/10. 1136/pgmj.2006.055046
- [11] Graham U, Ritchie KM. Postural orthostatic tachycardia syndrome. BMJ Open. 2009;10:1132. DOI: https:// doi.org/10.1136/bcr.10.2008.1132
- [12] Deb A, Karen M, Collin JC, Liz BW, Anna DH. A survey-based analysis of symptoms in patients with postural orthostatic tachycardia syndrome. Proc (Bayl Univ Med Cent). 2015;28(2):157-159. DOI: https://doi. org/10.1080/08998280.2015.11929217
- [13] Jiménez-Cohl P, Earle NM, González BR, Thieck EJ. Postural orthostatic tachycardia syndrome: a report of 15 cases. Rev Med Chil. 2012;140(2):145-152. DOI: https://doi.org/10.4067/ S0034-98872012000200001
- [14] Lewis I, Pearman J, Spickett G, Newton JL. Clinical characteristics of a novel subgroup of chronic fatigue syndrome patients with postural orthostatic tachycardia syndrome. J Intern Med. 2013;12:501-510. DOI: https: //doi.org/10.1111/joim.12022
- [15] Li H, Wang Y, Liu P. Body Mass Index (BMI) is associated with the therapeutic response to oral rehydration solution in children with postural orthostatic tachycardia syndrome. Pediatr Cardiol. 2016;37(7):1313-1318. DOI: https: //doi.org/10.1007/s00246-016-1436-1 [PMid:27350278]

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