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The Relation Between Non-dipping Blood Pressure Pattern and Postural Orthostatic Tachycardia Syndrome in Hypertensive Patients

Hipertansif Hastalarda Non-dipping Kan Basıncı Biçimi ve Postüral Ortostatik Taşikardi Sendromu Arasındaki İlişki

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ABSTRACT Objective: Postural orthostatic tachycardia syndrome (POTS) is an orthostatic intolerance with a pathophysiology that is likely to be heterogeneous. Hypertensive patients with a non-dipping pattern have an increased cardiovascular risk. There is a possibility that the same pathophysiological mechanisms may have a role in both POTS and the non-dipping pattern. To our knowledge, there are some studies showing the relation between POTS and non-dipping pattern in normotensive patients. However, data about this relation in hypertensive patients is limited. Our aim was to investigate the relation between 24-hour ambulatory blood pressure monitoring (ABPM) parameters and POTS in our study. **Material and Methods:** A total of 271 suitable patients were enrolled in our study. ABPM, Tilt table test, echocardiography, and laboratory tests were performed. **Results:** The study included 39 (14.3%) patients with POTS. There were no differences in the demographic variables, drug therapies, and laboratory as well as echocardiography parameters in all patients. Day-time, night-time, and 24-hour mean systolic and diastolic blood pressures and frequency of non-dipping pattern were significantly higher in patients with POTS. Day-time mean systolic blood pressure and non-dipping pattern were determined as independent predictors for POTS in multivariate logistic regression analysis. **Conclusion:** A relation was observed between POTS and non-dipping pattern in patients with hypertension, which needs further exploration in larger and detailed studies. Clinicians might be aware of POTS if their hypertensive patients complain of palpitations, lightheadedness, and fatigue in an upright position.

Keywords: Hypertension; postural orthostatic tachycardia syndrome; orthostatic intolerance; blood pressure monitoring; ambulatory

ÖZET Amaç: Postüral ortostatik taşikardi sendromu (POTS) bir ortostatik tolerans bozukluğudur. Bu sendromun patofizyolojisi muhtemelen heterojendir. Hipertansif hastalarda non-dipping patern varlığı kardiyovasküler riski artırmaktadır. Aynı patofizyolojik mekanizmaların, hem POTS hem de non-dipping paternde rol alması ihtimali mevcuttur. Bildiğimiz kadarıyla, normotansif hastalarda non-dipping patern ile POTS arasındaki ilişkiyi gösteren bazı yayınlar mevcuttur. Fakat hipertansif hastalarda bu ilişkiye dair veri kısıtlıdır. Amacımız hipertansif hastalarda 24 saatlik ambulatuvar kan basıncı ölçümleri (AKBÖ) ile POTS arasında bir ilişki olup olmadığını incelemekti. **Gereç ve Yöntemler:** 271 hasta çalışmamıza dâhil edildi. Tüm hastalara AKBÖ, Tilt masa testi, ekokardiyografi ve laboratuvar testleri yapıldı. **Bulgular:** 39 (%14,3) hastada POTS saptandı. Demografik veriler, ilaç tedavileri, laboratuvar ve ekokardiyografi parametreleri tüm hastalarda benzerdi. Gündüz, gece ve 24 saatlik ortalama sistolik ve diyastolik kan basınçları ve non-dipping profil sıklığı POTS olan hastalarda belirgin olarak daha yüksekti. Çok değişkenli lojistik regresyon analizinde, gündüz ortalama sistolik kan basıncı ve non-dipping kan basıncı profili POTS gelişimi için bağımsız öngördürücüler olarak saptandı. **Sonuç:** Hipertansif hastalarda non-dipping profil ile POTS arasında bir ilişki gözlenmiştir. Bu ilişkinin varlığını araştırmak için daha büyük ve detaylı çalışmalara ihtiyaç vardır. Klinisyenler hipertansiyon hastalarında ayağa kalkınca meydana gelen çarpıntı, yorgunluk, sersemlik gibi şikâyetler varsa POTS açısından dikkatli olmalıdırlar.

Anahtar Kelimeler: Hipertansiyon; postural ortostatik taşikardi sendromu; ortostatik intolerans; kan basıncı izlemi; ambulatuar

Postural orthostatic tachycardia syndrome (POTS) is a clinical condition with the following features: ≥30 beats per minute (bpm) boost or general heart rate >120 bpm with symptoms of low cerebral perfusion. These conditions must occur within10 minutes of standing or tilt test without any orthostatic hypotension. A ≥40 bpm augmentation in heart rate may be observed in young patients (12-19 years). Lightheadedness, palpitations, exercise intolerance, and fatigue are some of the symptoms in POTS. The pathophysiology and etiology of this syndrome are unknown, and possibly heterogeneous.[1] Some pathophysiologic mechanisms have been assumed, including a hyperadrenergic state, hypovolemia, peripheral adrenergic denervation, venous pooling, deconditioning, and possible central autonomic dysregulation.[2-6]

There are 3 blood pressure (BP) patterns according to ambulatory blood pressure monitoring (ABPM): dipping, non-dipping and extreme-dipping. Cardiovascular risk is increased in hypertensive patients with a non-dipping pattern. Increased sympathetic nervous system activity with or without impaired parasympathetic system activity may be some of the pathologic mechanisms responsible for the non-dipping pattern.[7,8] It was also shown that non-dipping pattern was related to an increased risk of end organ damage even in normotensive people.[9]

There is a possibility that the same pathophysiological mechanisms may have a role in both POTS and non-dipping pattern. To our knowledge, there are some studies showing the relation between POTS and non-dipping pattern in normotensive patients.[10] However, data about this relation in hypertensive (HT) patients is limited. We aimed to investigate the relation between 24-hour ABPM data and POTS in our study.

MATERIAL AND METHODS

STUDY POPULATION

We assessed previously evaluated patients (n=297) with essential HT from 1 January, 2015 to 31 December, 2017.[11] Due to insufficient data, 16 patients were not included to the study, and 10 patients retracted their informed consents. Finally our study consisted of 271 suitable patients. Patients with secondary, malignant or uncontrolled HT, diabetes mellitus (DM) type 1, known autonomic nervous system dysfunction, cardiovascular or cerebrovascular disease, heart failure, severe heart valve disease, cancer, and severe systemic diseases were not included in the study. The reason for excluding patients with type 1 DM was a higher risk of pre-existence or annual development of cardiac autonomic neuropathy according to Dimitropoulos et al.[12]

The protocol of the study was approved by the Local Ethics Committee (Çukurova University Faculty of Medicine, Noninvasive Clinical Researches Committee), and all the subjects provided their written informed consents. The patients were examined physically and their medical history was noted. The baseline characteristics of all patients were recorded including age, sex, HT, DM type 2, active smoking status, body mass index (BMI), weight, height, coronary artery disease family history, and current medications. Electrocardiography (ECG), routine laboratory tests, echocardiography, and noninvasive 24-hour ABPM were performed for all patients.

AMBULATORY BLOOD PRESSURE MONITORING

24-hour ABPM values were recorded by a portable device (DMS 300-ABP Holter Recorder, Beijing, China)-and BP measurements were performed every 30 minutes. Patients noted their sleeping hours. Periods of day-time (awake), night-time (sleeping), and 24-hour mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) values were recorded. Compared to day-time values, BP reduction of less than 10% in the night-time was defined as non-dipping. Blood pressure reduction greater than or equal to 10% in the night-time period was defined as dipping. Night-time BP fall greater than 20% was defined as extreme dipping.[13]

TILT TABLE TESTING AND DEFINITION OF POSTURAL ORTHOSTATIC TACHYCARDIA SYNDROME

Tilt table test (TTT) was used for the diagnosis of POTS. Patients received their routine daily medications before the test. Caffeine consumption, smoking, or exercise was not allowed 30 minutes

before the test. TTT was done using a tilt device (Gemesan Tilt Table G-71; Gemesan, İstanbul, Turkey). Patients rested silently in a supine position for at least 20 minutes and the tilt table was then quickly raised to 60-80°. Patients were monitored for vital signs during the course. The monitored data was recorded at the first, third, and fifth minute and every 5 minutes through the 30 minutes. The test was discontinued if syncope, presyncope, serious bradycardia, or severe symptoms occurred. The test was supervised by an experienced nurse, a technician, and a physician. POTS was determined using all of these findings: 1) frequent occurrence of symptoms like lightheadedness or palpitations after standing; 2) ≥30 bpm boost in heart rate or general heart rate >120 bpm in the upright position (or heart rate increase ≥40 bpm in 12–19 years old patients); 3) no signs of orthostatic hypotension.[14]

ECHOCARDIOGRAPHIC EVALUATION

Doppler and two-dimensional echocardiographic evaluations were performed using an echocardiography device (EPIQ 7; Philips Healthcare, Andover, Massachusetts, USA). Left atrial and left ventricular (LV) diameters and LV wall thicknesses were measured at end-diastole. The standards of the American Society of Echocardiography were used for all measurements.[15] Biplane Simpson's method was used for the calculation of LV ejection fraction.[16]

STATISTICAL ANALYSIS

The variables were separated as categorical and continuous groups. Categorical variables were shown as numbers and percentages and analyzed using the chi-square test. Continuous variables were demonstrated as mean±standard deviation and their distributions were analyzed by the Kolmogorov-Smirnov test. Independent samples t-test was used for normally distributed variables. The variables without normal distributions were analyzed by the Mann-Whitney U-test. Multivariate logistic regression analysis was done using significant variables. Independent predictors were determined for POTS. SPSS 22.0 (SPSS Inc. Chicago, IL, United States) software was used for the statistical analyses. A p-value <0.05 was considered as significant.

RESULTS

Our patients were separated into two groups of with or without POTS. There were 39 (14.3%) patients with POTS.

There were no differences in the demographic variables and drug therapies between the groups (Table 1). Laboratory and echocardiography parameters were also similar (Table 2).

Day-time, night-time, and 24-hour mean SBP and DBP values were significantly higher in patients with POTS ($p<0.05$ for each). Also, the frequency of non-dipping pattern was higher in this group ($p=0.038$) (Table 3).

Day-time mean SBP and non-dipping pattern were found to be independent predictors for POTS in multivariate logistic regression analysis (Table 4). According to this analysis, increased day-time mean SBP (per 1 mmHg) and the presence of a non-dipping pattern were found to increase the risk of POTS by 4.7% and 3.15 times, respectively (Table 4).

DISCUSSION

The most important finding in this study is that a non-dipping pattern is an independent predictor for POTS in HT patients.

POTS decreases the quality of life. Generally, it is observed between 15 and 50 years of age, at a 5 times higher frequency in female patients than male patients.[17] Often, patients with this disorder were misdiagnosed to have anxiety problems, chronic fatigue syndrome, or psychiatric situations by physicians.

The symptoms of POTS appear while standing. Both active standing and passive tilting methods were used in research studies. Plash et al. have conducted a study using both active standing and TTT to define POTS.[18] They reported that the 10-minutes test with ≥30 bpm increase criterion was sufficient to diagnose POTS with an acceptable sensitivity and specificity regardless of the method. Therefore, we used TTT in our patients.

TABLE 1: Comparison of patients' demographic findings and medications.

	POTS (-) n=232	POTS (+) n=39	p
Age (years)	53.4 ± 10.3	52.1 ± 12.6	0.49
Gender (male, %)	152 (65.5)	27 (69.2)	0.650
Office systolic BP (mmHg)	151.6 ± 19.8	156.9 ± 19.4	0.12
Office diastolic BP (mmHg)	90.6 ± 11.0	94.1 ± 11.9	0.069
Heart rate (beats/min)	82.1 ± 8.4	80.7± 8.6	0.313
Weight (kg)	84.1 ± 15.6	85.2 ± 15.5	0.688
Height (cm)	164.5 ± 8.5	165.6 ± 9.1	0.449
BMI (kg/m^2)	24.4 ± 2.3	24.5 ± 2.1	0.859
Smoking, n (%)	48 (20.7)	10 (25.6)	0.485
Diabetes mellitus, n (%)	38 (16.4)	7 (17.9)	0.807
Hypercholesterolemia, n (%)	50 (21.6)	7 (17.9)	0.609
ACE (n, %)	60 (25.9)	11 (28.2)	0.758
ARB (n, %)	39 (16.8)	2 (5.1)	0.06
β blocker (n, %)	91 (39.2)	17 (43.6)	0.606
Ca channel blocker (n, %)	72 (31.0)	16 (41)	0.218
α blocker (n, %)	7 (3.0)	0 (0)	0.598
Diuretic (n, %)	86 (37.1)	10 (25.6)	0.167

Abbreviations: ACE: Angiotensin converting enzyme; **ARB:** Angiotensin receptor blocker; **BP:** Blood pressure; **POTS:** Postural orthostatic tachycardia syndrome; **BMI:** Body mass index.

TABLE 2: Comparison of patients' laboratory and echocardiographic findings.

	POTS (-) n=232	POTS (+) n=39	p
Glucose (mg/dL)	111.1 ± 36.4	116.2 ± 44.1	0.437
BUN (mg/dL)	27.8 ± 8.8	32.6 ± 20.8	0.264
Creatinine (mg/dL)	0.77 ± 0.21	0.99 ± 1.05	0.214
Alanine transferase (u/L)	28.6 ± 11.5	27.2 ± 11.7	0.536
Uric acid (mg/dL)	5.8 ± 0.9	5.7 ± 0.9	0.848
Total cholesterol (mg/dL)	207.9 ± 40.5	205.6 ± 40.8	0.741
LDL cholesterol (mg/dL)	136.3 ± 34.7	135.3 ± 35.7	0.867
HDL cholesterol (mg/dL)	46.9 ± 11.1	47.6 ± 10.1	0.703
Triglyceride (mg/dL)	180.1 ± 96.1	177.0 ± 89.7	0.848
White blood cell count (1000/mm3)	7.8 ± 2.8	7.4± 1.6	0.274
Hemoglobin (g/dL)	13.8 ± 1.6	13.5 ± 1.5	0.265
BNP (ng/mL)	95.4 ± 148.8	75.6 ± 106.7	0.536
Hs-CRP (mg/L)	0.40 ± 0.51	0.35 ± 0.21	0.603
Ejection fraction (%)	61.2 ± 4.7	60.8 ± 4.9	0.587
LV diastolic volume (ml)	109.3 ± 23.1	111.2 ± 21.6	0.635
LV systolic volume (ml)	42.4 ± 10.5	44.7 ± 12.4	0.291
Left atrium diameter (mm)	36.7 ± 4.1	36.7 ± 3.9	0.986
Aortic diastolic diameter (mm)	29.4 ± 0.4	30.3 ± 0.4	0.182
Aortic systolic diameter	32.2 ± 0.3	33.0 ± 0.4	0.184
EFT (mm)	0.5 ± 0.1	0.5 ± 0.1	0.775

Abbreviations: BUN: Blood urea nitrogen; **BNP:** Brain natriuretic peptide; **Hs-CRP:** High sensitive C reactive protein; **LV:** Left ventricle; **POTS:** Postural orthostatic tachycardia syndrome; **EFT:** Epicardial fat thickness.

TABLE 3: Comparison of patients' ambulatory blood pressure findings.

	POTS (-) n=232	POTS (+) n=39	p
Day-time mean SBP (mmHg)	131.2 ± 14.1	138.9 ± 16.0	**0.002**
Day-time mean DBP (mmHg)	75.9 ± 11.6	81.3 ± 12.7	**0.008**
Night-time mean SBP (mmHg)	124.0 ± 17.2	131.4 ± 17.2	**0.015**
Night-time mean DBP (mmHg)	68.7 ± 9.4	74.2 ± 10.6	**0.008**
24-hour mean SBP (mmHg)	129.6 ± 14.1	137.3 ± 15.9	**0.002**
24-hour mean DBP (mmHg)	73.8 ± 11.1	78.3 ± 9.9	**0.023**
Augmentation index	28.9 ± 11.7	28.7 ± 11.5	0.937
Cardiac output	4.7 ± 0.7	4.9 ± 0.8	0.263
Pulse wave velocity	8.1 ± 1.7	8.3 ± 1.6	0.465
Dipping (n,%)	113 (48.7)	13 (33.3)	0.075
Non-dipping (n,%)	107 (46.1)	25 (64.1)	**0.038**
Extreme dipping (n,%)	12 (5.2)	1 (2.6)	0.700

Abbreviations: SBP: Systolic blood pressure; **DBP:** Diastolic blood pressure; **POTS:** Postural orthostatic tachycardia syndrome.

TABLE 4: According to multivariate regression analysis, independent parameters for the prediction of POTS.

	Odds Ratio	95% Confidence Interval	p
Day-time mean SBP (each 1 mmHg)	1.047	1.008-1.086	**0.016**
Non-dipping pattern (presence)	3.152	1.396-7.118	**0.006**

Abbreviations: SBP: Systolic blood pressure; **POTS:** Postural orthostatic tachycardia syndrome.

Increased sympathetic nervous system activity and central autonomic dysregulation are some of the putative mechanisms in POTS.[2,6] Figueroa et al. recently conducted a study which included 51 normotensive patients with POTS.[10] Non-dipping BP profile was seen in 28 (55%) patients. Laboratory evidence of mild autonomic deficits were detected in 29 (57%) patients. The authors extensively tested autonomic nervous system functions. They reported that there were no differences in the neuropathic findings between dippers and non-dippers. Interestingly, non-dipper patients had reduced orthostatic sympathetic reactivity. However, the mean age of patients (29.0 ± 8.0 years) was vastly different from that in our study. We did not perform any autonomic tests or measurements for catecholamine levels in our study. Their results documented that non-dipping was prevalent in POTS. Although our results were parallel to these findings, all of our patients were hypertensive. We found that the mean SBP and DBP of all types, and the frequency of non-dipping HT were significantly higher in our POTS group. High day-time SBP was found to be an independent predictor for POTS development (OR: 1.047, 95% CI: 1.008 – 1.086, p=0.016). We also determined that non-dipping HT increased the risk of POTS development by 3.15 times.

Non-dipping blood profile is harmful for both normotensive and hypertensive patients.[7,9] There are multiple pathological mechanisms in both POTS and non-dipping HT. According to the literature, increased sympathetic activity, decreased parasympathetic activity, or a combination of both clinical situations might have a role in the development of POTS and non-dipping HT.[19] Sympathetic activities are measured by plasma norepinephrine and epinephrine levels or heart rate variability analyses. Norepinephrine is a catecholamine and it has an important role in both central nervous system and cardiovascular system.[20,21] Too often, the upright posture plasma norepinephrine level is increased and above 600 pg/ml in POTS patients.[22] Huang et al. found that the non-dipper hypertensive patients had significantly higher plasma norepinephrine and epinephrine

level than dippers.[23] Hojo et al. reported an impaired sympathovagal balance in non-dipping HT.[24]

Obesity is another risk factor for non-dipping HT. Sympathetic system activation, hormone level elevations, endothelial dysfunction, and vascular structural changes are potential factors for impaired nocturnal BP drop in obese patients.[25] There were no obese patients in our groups. Diabetic patients are susceptible to developing non-dipping HT.[26] Spallone et al. stated that patients with neuropathic type 2 DM had decreased nocturnal BP fall.[27] Same correlation was found in type 1 diabetic patients with autonomic neuropathy.[28] We only enrolled type 2 DM patients without known autonomic nervous system dysfunction into our study. There were no differences in the frequency of type 2 DM between our groups. Hyperuricaemia and chronic kidney disease were reported as risk factors for non-dipping HT.[29,30] Uric acid levels were similar and there were no patients with chronic kidney disease in our groups.

There are several limitations to be mentioned in our study. Our sample size was small and we did not perform any kind of autonomic nervous system tests, and plasma or urine catecholamine level measurements. The mean age of patients in our study was markedly higher than in the aforementioned study groups.[10] Clearly, there is a need for larger and detailed studies in order to explain the relation between non-dipping HT and POTS.

CONCLUSION

We found a relation between POTS and non-dipping pattern in hypertensive patients in our study. POTS and non-dipping HT might have similar underlying pathophysiological mechanisms. It can be difficult to diagnose POTS, but clinicians might be aware of it if their HT patients complain of palpitations, lightheadedness, and fatigue especially in the upright position.

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Yurdaer Dönmez, Mevlüt Koç;* ***Design:*** *Yurdaer Dönmez, Hakan Caf, Yahya Kemal Içen;* ***Control/Supervision:*** *Yurdaer Dönmez, Hakan Caf, Atilla Bulut;* ***Data Collection and/or Processing:*** *Yurdaer Dönmez, Hakan Caf;* ***Analysis and/or Interpretation:*** *Yurdaer Dönmez, Yahya Kemal Içen;* ***Literature Review:*** *Yurdaer Dönmez;* ***Writing the Article:*** *Yurdaer Dönmez, Atilla Bulut;* ***Critical Review:*** *Mevlüt Koç, Yahya Kemal Içen.*

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