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RESEARCH

Relationship between panic disorder and non-dipper pattern

Panik bozukluk ile non-dipper tansiyon arasındaki ilişki

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Abstract

Purpose: In healthy people, blood pressure falls at night due to hormonal mechanisms. The non-dipper pattern, characterised by no blood pressure drop at night, is strongly associated with cardiovascular disease. Although many studies have been conducted on blood pressure elevation in patients with panic attacks, the non-dipper pattern has not been studied. In this study, we aimed to investigate the relationship between the non-dipper pattern and panic attacks.

Materials and Methods: Our study included 168 patients with panic disorders who had not been treated for at least one month and 210 control patients without the disease. Twenty-four-hour blood pressure monitoring was performed in all patients, and patients with a nocturnal blood pressure fall of less than %10 were considered non-dippers. All groups were compared according to baseline biochemical parameters and Holter results. In addition, non-dipper pattern was compared according to disease duration and severity.

Results: There were no significant differences between the groups in biochemical parameters such as urea, creatinine, albümin). Sistole blood pressure, diastole blood pressure, dipper pattern were statistically significantly higher in the panic disorder group. In addition, the non-dipper pattern, which was higher in the panic disorder group, was found to be associated with the duration and severity of the illness.

Conclusion: In our study, non-dipper pattern was found to be higher in patients with panic disorders, and this was found to be related to the severity and duration of the disease. As a result of our study, it can be assumed that the increased mortality in panic disorder patients is related to the non-dipper pattern, which is a cardiovascular risk factor.

Keywords:. Dipper pattern, non-dipper pattern, panic disorder, cardiovascular mortality

Öz

Amaç: Sağlıklı kişilerde nörohormonal mekanizmalar nedeniyle gece tansiyon düşmektedir. Gece tansiyonunun düşmemesi ile karakterize olan non-dipper durumu kardiyovasküler hastalıklarla yakından ilişkilidir. Daha önce panik atak hastalarında kan basıncı yükselmesi ile ilgili birçok çalışma yapılmasına rağmen non-dipper tansiyon üzerine çalışılmamıştır. Biz de bu çalışmada non-dipper tansiyon ile panik atak arasındaki ilişkiyi araştırmak istedik.

Gereç ve Yöntem: Çalışmamıza en az bir aydır tedavi almayan panik atak hastası olan 168 hasta ile hiçbir hastalığı olmayan 210 kontrol hastası dahil edildi. Tüm hastalara yirmi dört saatlik tansiyon holter takıldı ve gece tansiyonları %10'dan az düşen hatalar non-dipper olarak kabul edildi. Tüm gruplar bazal biyokimyasal parametreler ve tansiyon holter sonuçlarına göre karşılaştırıldı. Ayrıca non-dipper durumu hastalık süresi ve şiddetine göre kıyaslandı.

Bulgular: Üre, albümin, kreatinin gibi biyokimyasal parametreler açısıdan iki grup arasında anlamlı farklılık saptanmadı. Ortalama, gece ve gündüz tansiyonları panik atak grubunda daha yüksek ve istatistiksel olarak anlamlı yüksek bulunmuştur. Ayrıca panik atak grubunda daha yüksek saptanan non-dipper durumu hastalığın süresi ve şiddeti ilişkili olduğu saptandı.

Sonuç: Çalışmamızda non-dipper tansiyon panik atak hastalarında daha yüksek saptanmış olup, bu durum hastalık şiddeti ve süresiyle ilişkili bulunmuştur. Çalışmamız sonucunda panik atak hastalarında artan mortalitenin kardiyovasküler bir risk faktörü olan non-dipper tansiyon ile ilişkili olduğu düşünülebilir.

Anahtar kelimeler: Dipper tansiyon, non-dipper tansiyon, panik bozukluk, kardiyovasküler mortalite

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INTRODUCTION

Twenty-four-hour blood pressure monitoring (ABPM) reveals a distinct circadian rhythm known as 'dipping', characterised by a decline of approximately 10-30% during sleep. Individuals exhibiting a nocturnal blood pressure drop of less than 10% are designated as 'non-dippers' and constitute approximately 25% of patients with hypertension[1,2]. It has been observed that these patients demonstrate an elevated risk of cardiovascular incidents, encompassing myocardial infarction, cerebrovascular events, and sudden death[3,4].

It has been demonstrated that exposure to stress instigates the activation of adrenocorticotropic hormone (ACTH) by the anterior pituitary gland. This process is initiated through the production of corticotropin-releasing factor by the hypothalamus. This, in turn, results in the release of serotonin from the amygdala, which modulates cortisol production in the adrenal gland[5]. In patients diagnosed with anxiety or panic disorder, research has shown that there are persistent stress-related changes in the sympathetic nervous system, and an excessive systemic response to stress due to increased catecholamine production[6]. Consequently, an increase in blood pressure (systolic and diastolic), heart rate, and vascular resistance is evident[6].

Sympathetic and general monoaminergic mechanisms have been demonstrated to play an important role in regulating endogenous biological clocks and rhythms[7]. The impact of physical and emotional stimuli on these rhythms is facilitated by activating the autonomic nervous system[7]. Peak concentrations of both norepinephrine and epinephrine are typically observed in the early morning hours. Conversely, these concentrations undergo a decline, reaching their nadir in the late evening and during the early hours of night sleep, which results in substantial circadian fluctuations[7]. ACTH and cortisol exhibit a diurnal rhythm. ACTH and cortisol peak concentrations are observed in the morning, while trough concentrations are observed in the first hours of night sleep. In cases where this diurnal rhythm is lost, such as in primary aldosteronism, Cushing's syndrome, and pheochromocytoma, nocturnal blood pressure decrease is not observed in the expected amount.

In some cases, even elevations in blood pressure can be observed[8]. Circadian rhythm is influenced by adrenergic and catecholaminergic systems and serotonergic activity. Therefore, the exact mechanism cannot be understood.

Despite the plethora of studies that have identified a correlation between blood pressure and psychological symptoms, such as anxiety and depression[9-12], there is a paucity of research focusing on circadian blood pressure fluctuations. A review of existing studies reveals that patients diagnosed with major depressive disorder and anxiety disorder exhibited a reduced decline in nocturnal blood pressure[13,14]. In a small-sample study conducted with panic disorder patients, less decreases in nocturnal blood pressure were observed[15]. The present study was conducted to elucidate the relationship between dipper and non-dipper blood pressure patterns and the severity and duration of panic disorder in patients who were not taking medication.

MATERIALS AND METHODS

Sample

The study was approved by the Harran University Clinical Research Ethics Committee on 23.01.2023 (decision no: HRU/23.02.19). The study was designed and conducted following the Declaration of Helsinki. The study population comprised 96 female and 72 male patients diagnosed with panic disorder who presented to the Psychiatry Outpatient Clinic between 1 December 2023 and 1 December 2024. Exclusion criteria included patients with hypertension (office Blood pressure (BP) >140/90 mm Hg and mean 24-hour ABPM >130/80 mm Hg). Harran University Faculty of Medicine Hospital is a tertiary hospital located in Şanlıurfa in southeastern Turkey, serving a population of 3 million people annually, including the surrounding provinces. Two psychiatrists with at least five years of experience in the field assessed patients.

Patients who met the DSM-5 criteria for panic disorder and had not received treatment for a period of four weeks were included in the study. A total of 168 consecutive patients fulfilling these criteria were included in the study. A control group of 210 healthy volunteers was selected. Patients under the age of 18 and over the age of 65 were excluded from the study. Furthermore, patients with a history of psychiatric diseases other than panic disorder, as well as those with conditions such as hypertension, heart failure, chronic kidney and liver disease, active malignancy, or active infection, were excluded from the study. The participants were asked to complete a sociodemographic form. At the same time, the patient group was also asked to complete a panic disorder severity scale in order to evaluate the severity of their condition. The post-hoc power analysis of the study was conducted with G Power 3.1.2 program. It was concluded that the sample consisting of two groups of 168 and 210 participants had 99% power.

Assessment of 24-hour ABPM

24-hour blood pressure monitoring was performed with a portable device (name of device). Systolic, diastolic, and mean blood pressure were measured and recorded for all patients. Daytime was defined as 07:00-23:00 h and night-time as 23:00-07:00 h. Measurements were taken at intervals of 20 minutes during the day and 30 minutes at night. Systolic blood pressure values below 70 and above 250 were excluded. Diastolic blood pressure readings below 40 and above 150 were excluded. The nocturnal fall rates of SBP, DBP, and MBP were calculated according to the following formula: the rate of fall in night (%) = (daytime BP - night-time BP) × 100/ daytime BP (16). Patients with a 10-20% rate of fall in night of MBP were classified with dipper hypertension, while patients with <10% were classified with non-dipper hypertension[17].

Panic Disorder Severity Scale (PDSS)

The PDSS is a seven-item, semi-structured, physician-scored scale with good psychometric properties. It provides grading of panic frequency, anticipatory anxiety, avoidance of physical sensations, and impairment in work and social functioning[18]. The interviewer rates each of these symptoms between 0 and 4. The total score range is 0 to 28. Validity, reliability, and standardisation studies have been conducted in Turkish[19].

Statistical analysis

The collected data was analyzed using the SPSS 23.0 (Statistical Product and Service Solutions for Windows (Version 23.0, IBM Corp., Armonk, NY, U.S., 2015) software package. The Kolmogorov-Smirnov test was used to analyze the normal distribution characteristics of the numerical variables. Continuous variables are expressed as mean ± standard deviation or median (interquartile range, 25-75) values, whereas categorical variables are expressed as numbers and percentages. The Chi-square test was used to analyze categorical variables. The Mann-Whitney U (Triglyceride, troid stimulating hormone, duration of illness) and Student's t-test (Age, body mass index, urea) were used to compare continuous variables between the groups. Statistical significance was set at a probability (p) of ≤ .05.

Table 1. Clinical, demographic and laboratory characteristics of the study sample.

Variables	Panic disorder(n:168)	Control(n:210)	p
Age	37.47 ± 11.46	39.11 ± 9.59	0.137*
Gender Female Male	 96 (%57.1) 72 (%42.9)	 124 (%59) 86 (%41)	0.709 β
Duration of illness(month)	12.00 (3.75-36.00)		
Panic Disorder Severity Scale	17.00 (14.00-22.00)		
Smoking	61 (%36.3)	67(%31.9)	0.369 β
CAD	6 (%3.6)	9 (%4.3)	0.724 β
Diabetes mellitus	12 (%7.1)	21 (%10.0)	0.328β
Body Mass Index	25.44 ± 3.99	26.27 ± 4.20	0.053*
Glucose	97.50 ± 19.68	101.03 ± 23.61	0.157*
Urea	27.03 ± 7.99	27.93 ± 10.48	0.352*
Creatinine	0.81 ± 0.20	0.80 ± 0.21	0.704*
Albumin	4.56 ± 0.47	4.46 ± 0.40	0.072*
TSH	1.22 (0.89-1.87)	1.31(1.00-1.82)	0.306α
Triglyceride	132.00 (90.00-188.00)	152.00 (106.50-190.75)	0.110α
Cholesterol	176.46 ± 36.57	178.94 ± 28.92	0.522*
HDL	43.91 ± 11.35	45.27 ± 12.03	0.334*
LDL	100.31 ± 28.79	104.80 ± 25.55	0.164*
White blood cell	8.35 ± 2.19	8.33 ± 2.71	0.943*
Hemoglobin	13.98 ± 1.72	13.76 ± 1.66	0.232*
Hemotocrit	43.48 ± 4.99	42.71 ± 4 ,63	0.129*
Platelet	296.73 ± 84.88	290.88 ± 60.42	0.461*

CAD: Coronary artery disease, TSH: Thyroid stimulating hormone, HDL: High density lipoprotein, LDL: Low density lipoprotein
*: Student t test, α :Mann Whitney U Test β :Chi-Square Test

RESULTS

The present study comprised 168 patients diagnosed with panic disorder (96 female, 72 male; mean age 37.47 ± 11.46 years) and 210 healthy individuals (124 female, 86 male; mean age 39.11 ± 9.59 years). The demographic variables of the groups were similar in age, gender, smoking, diabetes mellitus, and coronary artery disease. The demographic variables and laboratory findings of the groups are presented in Table 1 ($p < 0.05$).

A significant elevation in both the 24-hour mean systolic and diastolic blood pressure levels was observed in patients diagnosed with panic disorder when compared to the control group (116.30 ± 10.118 mm Hg vs. 112.56 ± 12.162 mm Hg, $p = 0.001$; 80.36 ± 8.847 mm Hg vs. 73.91 ± 10.811 mm Hg, $p < 0.001$, respectively). The findings of this study demonstrate that daytime systolic and diastolic blood pressure levels were significantly elevated in patients diagnosed with panic disorder ($p < 0.001$ and $p < 0.001$, respectively). Furthermore, nocturnal systolic and diastolic blood pressure levels were found to be significantly elevated in panic disorder patients when compared to the control group ($p < 0.001$; $p < 0.001$, respectively). When the data were analysed in terms of dipper pattern, non-dipper was observed in 51.2% of panic disorder patients and 32.4% of the control group ($p < 0.001$). The full range of blood pressure variables can be found in Table 2.

The patients diagnosed with panic disorder were divided into two groups according to the duration of their illness. Upon dividing the duration of illness according to the median, it was observed that the non-dipper rate was significantly higher in those with a disease duration exceeding 12 months ($p = 0.032$). The second grouping was based on PDSS. The analysis of PDSS according to median revealed a significantly higher non-dipper rate in patients with PDSS scores above 17 points ($p = 0.014$) (Table 3).

Table 2. Blood pressure characteristics of the study sample.

Variables	Panic disorder(n:168)	Control(n:210)	p
24-hour Mean Systole BP (mmHg)	116.30 ± 10.12	112.56 ± 12.16	0.001 *
24-hour Mean Diastole BP (mmHg)	80.36 ± 8.85	73.91 ± 10.81	< 0.001*
Daytime Sistole BP (mmHg)	118.30 ± 11.06	113.79 ± 12.21	< 0.001*
Daytime Diastole BP (mmHg)	82.39 ± 9.58	75.38 ± 10.68	< 0.001*
Night Systole BP (mmHg)	111.27 ± 11.40	103.58 ± 14.79	< 0.001*
Night Diastole BP (mmHg)	73.44 ± 10.38	63.80 ± 13.47	< 0.001*
BP dipper pattern			< 0.001β
Dipper (%)	82 (%48.8)	142 (%67.6)	
Non-dipper (%)	86 (%51.2)	68 (%32.4)	

BP: Blood pressure; *: Student t test, α :Mann Whitney U Test β :Chi-Square Test

Table 3. Comparison of the study sample according to clinical characteristics.

Variables	Dipper	Non-dipper	
Duration of illness(month)			0.032 β
<12	38 (%59.4)	26 (%40.6)	
≥12	44 (%42.3)	60 (%57.7)	
Panic Disorder Severity Scale			0.014 β
<17	39 (%60.9)	25 (%39.1)	
≥17	43 (%41.3)	61 (%58.7)	

β :Chi-Square Test

DISCUSSION

In the present study, panic disorder patients were included, and ABPM was performed. Within the scope of our study, ambulatory blood pressure was analysed in panic attack patients without hypertension and chronic disease, who had not taken any medication for the last four weeks. The findings revealed that the night, daytime, and mean BP were elevated in this patient group compared to the control group. Furthermore, the non-dipper pattern was more prevalent in the patient group than in the control group. When the non-dipper pattern was

identified as elevated, the patient cohort was stratified into two distinct groups based on a median duration of illness spanning 12 months. Subsequent analysis of these two groups revealed a statistically significant predominance of the non-dipper pattern in the group exhibiting panic attack symptoms for a duration exceeding 12 months. In conjunction with these findings, when the patient group was divided into two groups according to the PDSS median value of 17, a statistically significantly higher prevalence of non-dipper patterns was observed in the group with higher PDSS.

Firstly, as demonstrated in earlier studies, the non-dipper pattern observed in individuals with hypertension can manifest as end-organ damage in normotensive individuals who do not have hypertension[20]. Despite the absence of a comprehensive understanding of the underlying causes, this condition has been linked to elevated left ventricular mass index and urinary albumin excretion[21]. Although the precise mechanism remains fully elucidated, researchers initially hypothesised that this condition was attributable to increased sympathetic activity[22-24]. Despite the absence of a definitive mechanism, studies have demonstrated that cardiovascular mortality is elevated in patients exhibiting the non-dipper pattern[3,4].

Despite the sporadic progression of the disease in patients diagnosed with panic disorder, the observed alterations in the patients' physiology are deemed to be irreversible. In this patient group, the condition cannot be explained by excessive sympathetic system activity, as observed in non-dipper pattern patients. Studies conducted on patients with panic disorder have revealed that their norepinephrine levels remain consistent when not experiencing panic attacks, and that their serotonergic activity increases significantly compared to control groups[25-30]. Whilst the extant literature does not yet furnish a comprehensive explanation of the complex situation in patients with panic disorder, studies suggest that a series of hormonal changes are active in these patients[25-30]. Furthermore, it has been established that emotional processes in patients with panic disorder are not only active during the waking state but also during nocturnal periods[31]. This observation supports the hypothesis that disruption of the circadian rhythm may underlie the non-dipper pattern observed in these patients.

Panic disorder and cardiovascular diseases have been demonstrated to be closely related. A substantial body of previous research has identified elevated cardiovascular mortality and morbidity rates in this specific patient demographic[20-24]. Despite the extensive research conducted in this field, the multifaceted nature of the cardiovascular interaction with panic disorder has not been fully demonstrated. In smaller studies, a non-dipper pattern was found to be more common in patients with panic disorder; however, these two conditions were not analysed in depth in these studies[15].

A review of studies evaluating the diurnal rhythm of BP reveals that the decrease in night-time systolic BP in elderly individuals diagnosed with depression was less pronounced than in the control group[13]. In a separate study, which focused on the evaluation of generalised anxiety disorder and panic disorder, it was revealed that the average night-time BP decrease was observed in both groups; however, this decrease was more pronounced in the panic disorder group[14]. The present study lends further support to the extant literature on this subject.

The present study was conducted with a relatively larger patient group compared to previous studies, and was evaluated in detail with the duration of illness and PDSS score. The study revealed that, while daytime and night-time BP values were elevated in patients with panic disorder, the non-dipper pattern was more prevalent in this patient group. The findings indicate a correlation between the non-dipper pattern, disease duration, and PDSS score. When the patient group was divided into two groups according to the median values of duration of illness and PDSS score, the non-dipper pattern was statistically significantly higher in the group with higher duration of illness and PDSS score. This finding suggests a potential association between panic disorder and the non-dipper pattern, which may contribute to cardiovascular mortality. The correlation between the disease duration and PDSS score reinforces the established relationship between these two diseases.

Despite the study's strengths, it is important to acknowledge its limitations. Firstly, the relatively small number of patients served to limit the generalisability of the study to the general population. Secondly, the relatively brief observation period precludes the capacity to draw robust conclusions regarding mortality. Thirdly, the investigation did not encompass the impact of medications administered concomitantly with panic disorder treatment on the patient's condition. Finally, the selection of patients

and controls from normotensive individuals without comorbidities limits the generalisability of the study to the general population.

In conclusion, the present study found that the non-dipper pattern was more prevalent in patients diagnosed with panic disorder. This finding was associated with the duration and severity of the condition. It is hypothesised that the increased cardiovascular mortality and morbidity observed in patients with panic disorder may be associated with the non-dipper pattern. Further research is warranted, particularly through large-scale, long-term observational studies.

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