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Original Article

The evaluation of ventricular arrhythmia risk by using electrocardiographic parameters in patients with dipper and non-dipper hypertension

Dipper ve nondipper hipertansiyonda ventriküler aritmi riskinin elektrokardiyografik parametreler üzerinden değerlendirilmesi

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Abstract

Aim: Non-dipper hypertension (NDHT) is associated with increased cardiovascular mortality. Lots of different electrocardiographic parameters can be used for this purpose. Some electrocardiographic repolarization parameters and some particular parameters obtained from 24 hours holter recordings frequently were used. The aim of this study was to evaluate ventricular arrhythmia risk by using most of this electrocardiographic parameters in patients with dipper hypertension (DHT) and NDHT.

Material and methods: 220 patients with hypertension were included this study. Patients were divided into two groups as DHT and NDHT according to the 24 hours ambulatory blood pressure monitoring. Two groups were compared with electrocardiography and echocardiography parameters and also were compared with heart rate variability (HRV) and heart rate turbulence (HRT) parameters.

Results: There were no significant differences between DHT and NDHT groups with regard to demographic and laboratory datas. Also echocardiography parameters normally distributed and have no significant differences between two groups. There were no significant differences between DHT and NDHT groups with regard to left ventricular mass index (p=0.280). Although QT, QT dispersion, HRV and HRT parameters differences were not statistically significant , results were in favour of DHT in terms of ventricular arrhythmia risk.

Conclusion: When hypertensive patients having no statistically significant differences in terms of left ventricular diamaters and left ventricular mass between them were divided as DHT and NDHT; there were no statistically significant differences between two groups with regard to electrocardiographic ventricular arrhythmia parameters although results were in favour of DHT.

Keywords: hypertension; dipper; non-dipper; ventricular arrythmia parameters

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Öz

Amaç: Non-dipper hipertansiyon (NDHT) artmış kardiyovasküler mortalite ve morbidite ile ilişkilidir. Çeşitli çalışmalar çok sayıda farklı elektrokardiyografik parametrenin bu amaçla kullanılabileceğini göstermektedir. Özellikle bazı elektrokardiyografik repolarizasyon parametreleri ve 24 saatlik holter kayıtlarından elde edilen bazı özel parametreler sıklıkla kullanılmıştır. Bu çalışmada amaç dipper hipertansiyonlu (DHT) ve NDHT hastalarda ventriküler aritmi riskinin bu elektrokadiyografik parametrelerin çoğunun birlikte kullanılarak değerlendirilmesidir.

Gereç ve Yöntemler: Çalışmaya esansiyel hipertansiyon tanısıyla izlenen 220 hasta alındı. Hastalar 24 saatlik ambulatuvar kan basıncı izlemesi sonuçlarına göre iki grupta değerlendirildi. Sistolik ve/veya diyastolik kan basıncı gece değerleri ortalaması gündüz değerleri ortalamasından %10 veya daha fazla düşük olanlar DHT grubuna, %10'dan az düşük olanlar NDHT grubuna alındı. İki grubun kan basıncı, ekokardiyografi, laboratuar verileri, EKG ve 24 saatlik ritim holter kaydından elde edilen kalp hızı değişkenliği (KHD) ve kalp hızı türbülansı (KHT) verileri karşılaştırıldı.

Bulgular: Demografik bulgular, laboratuar verileri ve ekokardiyografik veriler normal dağılıma uymakta idi ve gruplar arasında anlamlı fark yoktu. Her iki grup arasında sol ventrikül kitle indeksi açısından fark saptanmadı (p=0,280). QT, QT dispersiyonu, KHD, KHT parametrelerinde de istatistiksel anlamlı fark tespit edilememesine rağmen ventriküler aritmi riski açısından verilerin DHT lehine olduğu görüldü. Ayrıca NDHT' da otonom fonksiyonlarda sempatik sistem lehine baskınlığı teyit edecek şekilde ortalama kalp hızı istatistiksel olarak anlamlı olacak şekilde daha yüksek bulundu.

Sonuç: Sol ventrikül çapları ve kitleleri bakımından aralarında anlamlı fark olmayan hipertansif hastalar, dipper ve non-dipper olarak ayrıldığında elektrokardiyografik ventriküler aritmi öngördürücüleri bakımından veriler iki grup arasında dipper lehine olmasına karşın bu fark istatistiksel olarak anlamlı düzeye ulaşmamıştır.

Anahtar kelimeler: hipertansiyon; dipper; non-dipper; ventriküler aritmi parametreleri

Introduction

Cardiovascular diseases (CVD) are seen as the primary cause of death in the world. Hypertension (HT) is one of the main causes of CVDs and an important risk factor for sudden cardiac death. The incidence of sudden cardiac death increases with elevated blood pressure (BP) in parallel with BP values [1]. There is a strong relationship between the systolic BP and diastolic BP and the cardiovascular (CV) risk. HT is responsible for 45% of heart disease-related deaths and 51% of stroke-related deaths [2]. There are studies showing that the frequency of ventricular arrhythmia and consequently the risk of sudden death increases in patients with HT. However, there are limited data on which HT patients have a higher tendency to ventricular arrhythmia.

It has been shown that BP levels obtained by ambulatory measurement are more valuable in predicting HT complications and CV morbidity when compared with BP levels measured in the office [3]. BP changes with circadian rhythm during the day. Studies have shown that BP shows a nocturnal decline in healthy individuals. The rate of this decline varies from person to person. According to ambulatory blood pressure monitoring (ABPM) data obtained from healthy individuals, BP is highest in the morning, shows a slow decrease during the day, and remains at its lowest levels during the night [4]. The circadian rhythm of BP has led to the development of a new classification which is made by ABPM. A decrease in BP of ≥ 10% when compared to daytime was defined as dipper hypertension (DHT) and a < 10% decrease as non-dipper hypertension (NDHT).

In our study, we separated HT patients as DHT and NDHT. Our aim was to determine which group had a higher risk of ventricular arrhythmia and sudden cardiac death and whether this classification is associated with the risk of arrhythmia by using ventricular arrhythmia parameters.

Material and Methods

Our study included 220 HT patients. They rested for at least five minutes before measuring tension. They were seated with their feet on the ground. Their arm was supported by bringing it to the heart level. The cuff of the sphygmomanometer was placed 2.5-3 cm above the bend of their elbows wrapping at least 80% of their arms and the measurement was performed. It was repeated at least ten minutes later. A blood pressure of ≥ 140/90 mmHg at each measurement was diagnosed as HT. Cases with and suspected to have secondary HT were excluded from the study. The exclusion criteria for all groups were non-reliable T-waves on the electrocardiograhpy (ECG), atrial fibrillation, bundle branch block, moderate or severe valvular heart diseases, thyroid disorders, cardiomyopathies,

congenital heart diseases, malignancy, pulmonary HT, electrolyte disturbances, acute coronary syndromes, heart failure, history of myocardial infarction, history of coronary artery bypass grafting, implanted permanent pacemaker, and left ventricular segmental wall-motion defects in the echocardiographic exam. The local ethics committee approval and informed consent from all patients were obtained (Tokat Gaziosmanpasa University ethics committee with project number 14-KAEK-208). All people included in the study signed the informed consent form.

Electrocardiographic examination

A 12-lead superficial ECG was recorded from all patients. 12-lead electrocardiography was performed in a supine position at a rate of 25 mm/sec and an amplitude of 10 mm/mV after 20 minutes of rest (Cardiofax V; Nihon Kohden Corp., Tokyo, Japan). The ECG images of the patients were scanned and examined at a magnification of 400% using the Adobe Photoshop software. The RR distance from the DII lead was calculated.

QT times for each derivation were measured separately. QTcs were calculated by using the Bazet formula. QTc dispersion was calculated. Tp-e times were also calculated from leads V2 and V5. QTc/Tp-e times were calculated. Each measurement was repeated at least twice by two separate researchers and the means of the data were used.

Ambulatory blood pressure monitoring

ABPM was performed using a noninvasive recording system. The device (SunTechAccuwin ProV3) was programmed to perform the measurement for 24 hours, every 30 minutes during the day (07.00-22.00) and every 60 minutes at night (22.00-07.00). The mean levels of ≥ 10% of the daytime levels were in the DHT group and those of <10% were in the NDHT group.

24-hour ECG Holter recording

24-hour holter, a non-invasive recording of the electrocardiogram, was performed using a device with a three-channel analog recording system. The device's software was used to calculate the parameters. Ambulatory ECG values were measured by digital recording on a flashcard using a DL 700 Digital Holter recorder (Ela medical SyneScope V3.10). The minimum and maximum distances between the heart rate variability (HRV), heart rate turbulence (HRT), lowest and highest heart rate values, and the consecutive R waves were calculated by the 24-hour ECG Holter recording.

The same recording device (Ela medical SyneScope V3.10) was used to calculate the HRV. We made the time-domain analysis of the HRV as follows: We obtained the average heart rate for 24 hours and for the day- and night-time separately. We used the ratio of the number of intervals to the total number of R-Rs (pNN50) where the difference between the consecutive R-R intervals was over 50 milliseconds. We used the ratio of the number of intervals to the total number of R-Rs (pNN30) where the difference between the consecutive R-R intervals was over 30 milliseconds. We used the arithmetic mean (RMSSD) of the square root of the difference between consecutive R-R intervals. We obtained the standard deviation (SDNN) of the time (R-R interval) between consecutive normal QRS complexes. For 24 hours, we used the standard deviation (SDANN) and the variability index (VarIndex) of the average R-R intervals of five-minute recordings.

The total power (TP) (<0.4 Hz) obtained from the 24-hour recordings by frequency-domain method, the lowest frequency (VLF) (0.003-0.04 Hz), the low frequency (LF) (0.04-0.15 Hz), high frequency (HF) (0.15-0.40 Hz) and, normalized (nu) equivalents of these values were evaluated. These variables were digitized using power spectral curves and expressed as Ln (ms2/Hz). LF/HF ratio was determined. All measurements were made according to the recommendations of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology [5].

Using ventricular premature beats that met the eligibility criteria for HRT measurement, turbulence onset (TO) and turbulence slope (TS) values were automatically calculated from the Holter recording by the software program (HRT View Version 0.60-0.1). TO indicating an early acceleration phase was measured as follows: the two sinus rhythm lengths measured immediately prior to the ventricular premature beats were subtracted from the sum of the two sinus rhythm lengths measured after the ventricular premature beats. The result was divided into two sinus rhythms measured before the ventricular premature beat and expressed as a percentage (%). TS showing late deceleration was calculated by determining the length of the five most sloping sinus cycles in 20 sinus cycles measured after ventricular premature beat and expressed in milliseconds. Levels <0% were considered normal for TO, and > 2.5 ms/RR for TS. Turbulence loss was accepted as an increase in TO and a decrease in TS.

Echocardiographic examination

All echocardiography examinations (General Electric Vivid S5, Milwaukee, WI, USA) were performed by an experienced cardiologist in all subjects using a 2.5–3.5 MHz transducer in the left decubitus position. Two-dimensional and pulsed Doppler measurements were obtained using the criteria of

the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Twelve left ventricular ejection fraction (LVEF) was assessed using Simpson's method [6]. Left ventricular mass was calculated using the Devereux formula. The body surface area of the patients was calculated by the Dubois formula and left ventricular mass index (LVMI) was calculated by dividing the left ventricular mass into the body surface area. LVMI values above 125 g/m2 in men and 110 g/m2 in women were accepted as left ventricular hypertrophy (LVH) findings [7].

Statistical analysis

SPSS 18.0 software package (SPSS Inc., Chicago, IL, USA) was used for statistical analyses. All values are given as mean ± standard deviation. Mean values of continuous variables were compared between the groups using the Student's t-test or the Mann-Whitney U test, according to whether normally distributed or not, as tested by the Kolmogorov-Smirnov test.

We used Pearson's correlation test to evaluate the relationship between normally distributed parameters, and Spearman's Rho correlation test to examine the relationship between non-normally distributed parameters.

Results

Evaluation of basic clinical and demographic characteristics revealed no statistically significant difference between the two groups in terms of age, gender distribution, body mass index, and smoking status (Table 1).

Table 1: Basic clinical, demographic and laboratory data of patients

	DHT	NDHT	P value
Age	55 ± 8.367	52.94 ± 8.247	0.243
Sex (male %)	34 (%37.7)	30 (%27.7)	0.902
BMI (kg/m^2)	26.76 [26-30.4]	27.58 [26,6-30,4]	0.282
Smoking	5 (%15.2)	8(%17)	0.823
Glucose (mg/dL)	97.85 [93.7-104]	97 [89.8-113.7]	0.494
Sodium (mmol/L)	141 [139-142]	140 [139-142]	0.319
Potassium (mmol/L)	4.4 ± 0.28	4.5 ± 0.4	0.125
Calcium (mg/dL)	9.6 ± 0.4	9.4 ± 0.3	0.079
Magnesium (mg/dL)	2.205 [2.11-2.315]	2.25 [2.06-2.345]	0.965
TSH (µIU/mL)	1.41 [1.12-1.86]	1.39 [1.0-2.26]	0.953
Kreatinin (mg/dL)	0.8 [0.7-0.965]	0,76 [0,62-9]	0.049

TSH: Thyroid Stimulating Hormone, BMI:Body mass index

DHT was detected in 90 patients (40.9%) and NDHT in 130 patients (59.1%) according to their ABPM results. There was no statistically significant difference between patients with DHT and NDHT in terms of echocardiographic left ventricular parameters (Table 2).

Table 2: Echocardiographic data of patient groups

	DHT	NDHT	P value
LVDd (mm)	45 [43.5-48]	46 [44-48.5]	0.659
LVSd (mm)	30 [26-53.3]	30 [28-32]	0.616
IVS(mm)	11 [10-12]	10 [9-11]	0.176
Posterior wall (mm)	10 [9-11]	10 [8-10]	0.280
Left atrium (mm)	36.88 ± 3.7	35.77 ± 4.4	0.243
LV EF (%)	60 [60-65]	65 [60-65]	0.219
LV Mass Index	86.4 ± 99.19	80.15 ± 91.18	0.280

EF: Ejection Fraction, IVS: Interventricular septum, LVH: Left Ventricle, LVDd: Left Ventricular End Diastolic Diameter, LVSd: Left Ventricular End Systolic Diameter

When the DHT group and the NDHT group were compared, we observed that almost all of the electrocardiographic data showed elongation in milliseconds in the NDHT group. However, this difference did not reach statistical significance.

QTc dispersion was also higher in NDHT group, however, there was no statistically significant difference (Table 3).

Table 3: Comparison of ECG data between the patient groups

	DHT	NDHT	P value
QTV2 (millisecond)	372 [352-383]	376 [347-386]	0.528
QT V5	377 ± 25	367 ± 31	0.153
QTc V2	414 ± 28	414 ± 25	0.952
QTc V5	423 ± 28	411 ± 26	0.060
QTc dispersion	35 [29-58]	44 [27.5-59.5]	0.494
TPe V2	103.44 ± 17.16	105.66 ± 16.01	0.555
TPe V5	91.52 [81.36-96.61]	91.52 [86.44-101.69]	0.654
TPe /QT V2	0.280 ± 0.04	0.285 ± 0.04	0.623
TPe/QT V5	0.244 ± 0.04	0.248 ± 0.03	0.653
TPe/QTc V2	0.250 ± 0.04	0.255 ± 0.03	0.564
TPE /QTc V5	0.217 [0.196-0.228]	0.222 [0.199-0.239]	0.356

The average heart rate was significantly higher in the NDHT group with 78 ± 8.1 and in the DHT group it was 73 ± 8.3 group (p = 0.032). Furthermore, the minimum heart rate values were found to be 61 ± 7.2 in NDHT and 58 ± 7.8 in DHT, and these values were found to be statistically significant (Table 4).

Table 4: Comparison of General ECG Holter Data Between the Patient Groups

	DHT	NDHT	P value
Minimum Heart Rate	58 ± 7.8	61 ± 7.2	0.036
Maximum Heart Rate	101 ± 11.8	97 ± 13.6	0.223
Average Daytime Heart Rate	71 ± 11.4	75 ± 8.5	0.052
Average Night-time Heart Rate	77 ± 10.7	81 ± 9.8	0.079
Average Heart Rate	73 ± 8.3	78 ± 8.1	0.032

There was no statistically significant difference between the patient groups in parameters related to HRV and HRT data (Table 5) and both were obtained by time-domain and frequency-domain methods (Table 5).

Table 5: Comparison of Time-Domain and Frequency-Domain Method and HRV Data, Comparison of HRT data

	DHT	NDHT	P value
PNN50 (%)	4.61 [1.67-11.34]	2.22 [1.14-6.19]	0.141
PNN30 (%)	16.27 [7.41-24.89]	12.43 [5.17-20.52]	0.206
RMSSD (ms)	28.57 [19.54-35.68]	23.43 [18.09-30.49]	0.324
Variable Index	2.07 [1.64-2.69]	1.96 [1.6-2.4]	0.594
SDNN (ms)	48.14 ± 15.25	46.29 ± 11.7	0.544
SDANN (ms)	105.9[89.46-139.84]	97.85 [86.76-108.37]	0.052
Total Power	2465 [1234-3379]	1947 [1463-2621]	0.629
VLF Power (ms^2)	1726 [825-2251]	1342 [1021-1799]	0.625
LF Power (ms^2)	248 [246-691]	341 [238-474]	0.261
HF Power (ms^2)	151 [65-270]	106 [59-168]	0.123
LF nu	61 [51-66]	63 [54-68]	0.356
HF nu	23 ± 9	20 ± 10	0.124
Turbulence Onset	-0.002 [-0.03-0.006]	-0.053 [-0.01-0.001]	0.968
Turbulence Slope	5.8 [3.8-12.3]	5 [2.4-7.8]	0.111

PNN50: the ratio of the number of intervals where the difference between consecutive R-R intervals is greater than 50 milliseconds to the total number of R-Rs, PNN30: the ratio of the number of intervals where the difference between consecutive R-R intervals is over 30 milliseconds to the total number of R-Rs, RMSSD: the arithmetic mean of square root of the difference between consecutive R-R intervals, SDNN: the standard deviation of time (R-R interval) between consecutive normal QRS complexes, SDANN: the standard deviation of average R-R intervals of five minute recordings over 24 hours, HF:High Frequency, LF:Low Frequency, VLF:Very Low Frequency

The correlation analysis between the LVMI and QTc data measured separately from each lead in the ECG data were examined. There was a statistically significant difference in the results regarding QTc V3 ($p = 0.04$), QTc V2 ($p = 0.01$), QTc V1 ($p = 0.04$), and QTc D1 ($p = 0.04$).

Discussion

To the best of our knowledge, if there is no cardiac end-organ damage, the separation between DHT and NDHT has no effect on the risk of arrhythmia. HT is one of the important risk factors of CVDs. The prevalence of HT in society is increasing, both in relation to the ease of access to diagnosis and healthcare centers and to the increase in other CVD risk factors. HT-related research answers questions about etiology, classification, and treatment. However, regardless of other factors, it has not yet been elucidated what role personal differences play in the risk levels of different people with close BP values [8].

There are many studies showing that ABPM predicts CV mortality and morbidity better and that they are higher in NDHT [9]. There is a physiological decline in BP at night. This reflex reduces with age. The reasons for this condition are thought to be as follows: Vascular elasticity decreases due to aging and atherosclerosis. The regulation of the autonomic nervous system is impaired and the vasoconstriction associated with the sympathetic nervous system dominates the vasodilatation relevant to the parasympathetic nervous system [10]. There are several causes of night-time BP decline in the normal circadian rhythm, such as decreased blood levels of cortisol, adrenaline, and noradrenaline. Patients with NDHT have lower levels of decline than those with DHT. In addition, an increased α1 adrenergic receptor response and a decreased parasympathetic activity were found in NDHT patients [11]. It is known that lack of expected decrease in night-time BP is associated with increased CV morbidity. CV risk factors such as a decrease in HRV, an increase in plasma creatinine level, and a decrease in high-density lipoprotein level are more common in patients with low BP at night [12]. It has been shown that target organ damage is higher in NDHT due to greater deterioration in endothelial functions than in DHT [13].

The risk of ventricular arrhythmia and sudden death is increased in hypertensive patients. Data on the incidence of arrhythmia in HT and the prognostic value of these indicators are limited. Some data even contain contradictions. There are limited studies on the relationship between ambulatory measured BP data and non-invasive ventricular arrhythmia parameters.

HT is a pathologic condition known as hypertensive heart disease, which develops as a result of structural and functional adaptation with hemodynamic effects. It manifests itself as blood flow disorders due to increased LV mass, diastolic dysfunction, congestive heart failure, arrhythmia, and microvascular diseases [14]. One of the most common cardiac complications is LVH. In our study, no significant difference was found between the two groups in terms of LVH.

Studies investigating the relationship between the diurnal course of BP and the LVH found that night-time BP values were more correlated with LVH [15,16]. However, in the study performed by Grandi et al., no correlation was found between LV morphology and night-time BP elevation [17]. In LVH, the coronary reserve is reduced resulting in ischemia and fibrosis which may impair homogeneity in myocardial repolarization. Therefore, the variability in QT interval is an indicator of arrhythmogenicity. It was reported that there was a linear relationship between the

LVMI and QTd in HT [18]. QTd was increased in non-proportional LVH, such as HT, while it was found normal in proportional LVH, such as an athlete's heart [19].

Cavallini showed that QTd increased in HT and LVH patients, but this increase was not associated with complex ventricular arrhythmias [20]. Galinier followed up 214 hypertensive patients (33.7% of whom were hypertrophic) after an average of 42 months. He found an increased QTd (>80 ms) associated with cardiac mortality in univariate analyses. He reported that this relationship was absent in multivariate analyses. In our study, we found that there was an increase in QTd duration in the NDHT group compared to the DHT group, but this increase was not statistically significant.

Recently, new electrocardiographic parameters Tpe, Tpe/QT, and Tpe/QTc parameters have emerged in relation to increased repolarization dispersion [21]. These markers can be used as an electrocardiographic predictor for ventricular arrhythmogenicity and sudden cardiac death [22]. Demir et al showed that Tpe and Tpe/QT ratio increased in patients with NDHT [23]. In our study, there were differences in Tpe interval, Tpe/QT and Tpe/QTc ratios between the

groups. The NDHT group had higher results than the DHT group. However, these differences were not statistically significant. It is clear that more studies are needed to demonstrate the relationship between the patients with NDHT and ventricular arrhythmias and the Tpe interval and Tpe/QT ratio.

Our study showed that HRV parameters were generally smaller in the NDHT group than in the DHT group, but this difference was not statistically significant. Similarly, Poanta et al. compared normotensive patients with type 2 diabetes and NDHT. In their study, HRV parameters were smaller in the NDHT group. However, very few of these parameters showed a statistically significant change [24]. This result was attributed to the fact that autonomic functions may be impaired in relation to the pathophysiology of diabetes. In our study, the small number of patients may be the reason why the results did not reach statistical significance. Another recent study by Dauphinot et al. examined the relationship between DHT and NDHT risk changes and a decreased autonomic nervous system activity assessed by HRV parameters in the elderly population [25]. In their study, increased risk of the non-dipper pattern was detected in patients with low autonomic nervous system activity. Regardless of HT, a decreased autonomic nervous system activity was associated with non-dipper blood pressure pattern. This study also demonstrated that autonomic dysfunction may be a predictive and etiological factor in non-dipper blood pressure pattern.

In our study, we also evaluated TO and TS, which are HRT parameters. There was no statistically significant difference between the groups. This may be associated with the small size of the selected patient population.

Autonomic nervous system dysfunction is usually associated with non-dipper BP phenomenon [26]. HRV and HRT reflect a reduced cardiac autonomic nervous system activity, a particularly increased sympathetic activity, and a decreased parasympathetic activity [27]. These two methods can evaluate cardiac autonomic dysfunction as noninvasive and have been accepted as new risk parameters for sudden cardiac death [28].

According to the European Society of Cardiology, HRT is an independent predictor of total mortality after myocardial infarction and a marker of vagal activity [29]. In one study, the average heart rate was found to be higher in non-dipper patients than in dipper patients. This may be due to the predominance of sympathetic activity due to cardiac autonomic dysfunction in subjects with nondipper blood pressure pattern [30]. In our study, the average heart rate supporting this data was found to be statistically higher in the NDHT group.

Study limitations

The main limitation of our study was the small number of patients. In addition, only individuals with HT were included in the study and therefore no comparison was made with healthy controls. In addition, since there was no follow-up study, the effects of duration of exposure to DHT and NDHT on the risk of arrhythmias were not taken.

Conclusion

When HT patients were grouped as DHT and NDHT, there was no difference between the two groups in terms of the risk of ventricular arrhythmia, the HRV and HRT parameters evaluated by ECG parameters, and the HRV and HRT parameters evaluated with a 24-hour Holter. As a result, if there is no cardiac end-organ damage, the separation between DHT and NDHT has no effect on the risk of arrhythmia. It was concluded that this may be due to the fact that there was no difference between the groups in terms of left ventricular functions, left ventricular masses, and LVMI between our DHT and NDHT groups.

Declaration of conflict of interest

The authors received no financial support for the research and/or authorship of this article. There is no conflict of interest

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