RESEARCH ARTICLE / *Araştırma Makalesi*

The Status of Frontal QRS-T Angle in Hypertensive Patients with Different Left Ventricular Geometry

Farklı Sol Ventrikül Geometrik Paterne Sahip Hipertansiyon Hastalarında Frontal QRS-T Açısının Değerlendirilmesi

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Abstract

Objective	Assessing left ventricular (LV) structure and function gives information on cardiovascular morbidity and mortality, making it essential for evaluating hypertensive heart disease. Frontal QRS-T angle (fQRSTa) is a novel approach to quantify the heterogeneity between myocardial depolarization and repolarization. The main purpose of the present study was to define the correlation between different LV geometric patterns and fQRSTa in patients with hypertension (HT).
Materials and Methods	273 patients with hypertension admitted to the cardiology out-patient clinic were enrolled consecutively. All patients were evaluated by transthoracic echocardiography and classified into three groups based on LV hypertensive geometry as normal geometry (group 0), concentric remodeling (group 1), and concentric or eccentric hypertrophy (group 2). The fQRSTa was defined as the absolute angle difference between the frontal plane QRS axis and T wave axis.
Results	Compared with group 0, fQRSTa was higher in group 1 (12 [6 – 19] vs. 17 [12 – 24], p=0.023) and group 2 (12 [6 – 19] vs. 39 [28 – 54], p<0.001). Also, fQRSTa was higher in group 2 than group 1 (p<0.001). Correlation analysis revealed a significant correlation between fQRSTa and LV geometry (r=0.525, p<0.001). Multiple linear regression analysis revealed that fQRSTa was independently correlated with Em to Am ratio (β=0.104, p=0.045), left ventricle mass index (β=0.342, p<0.001), QTc (β=0.194, p<0.001), and LV geometry (β=0.257, p<0.001).
Conclusion	Patients with LVH were found to have wider fQRSTa and longer QT duration than those with normal ventricles or concentric remodeling.
Keywords	Left ventricular geometry; frontal QRS-T angle; hypertension

Öz

Amaç	*Hipertansif kalp hastalığının değerlendirilmesinde sol ventrikül (SV) yapısının ve fonksiyonunun incelenmesi, kardiyovasküler morbidite ve mortalite hakkında önemli bilgiler sağlar. Frontal QRS-T açısı (fQRSTa), miyokardiyal depolarizasyon ve repolarizasyon arasındaki heterojenliği ölçmek için kullanılan yeni bir yöntemdir. Bu çalışmanın temel amacı, hipertansiyonlu (HT) hastalarda farklı SV geometrik paternleri ile fQRSTa arasındaki ilişkiyi incelemektir.*
Gereç ve Yöntemle	*Kardiyoloji polikliniğine başvuran ardışık 273 hipertansiyon hastası çalışmaya dahil edildi. Tüm hastalar transtorasik ekokardiyografi ile değerlendirildi ve SV geometrik yapısına göre normal geometri (grup 0), konsantrik yeniden şekillenme (grup 1) ve konsantrik veya eksantrik hipertrofi (grup 2) olarak üç gruba ayrıldı. fQRSTa, frontol düzlem QRS aksı ile T dalga aksı arasındaki mutlak açı farkı olarak tanımlandı.*
Bulgular	*fQRSTa' sı grup 0 ile karşılaştırıldığında, grup 1' de (12 [6 - 19] - 17 [12 - 24], p = 0,023) ve grup 2' de (12 [6 - 19] - 39 [28 – 54], p <0,001) anlamlı olarak daha yüksekti. Ayrıca, fQRSTa grup 2' de grup 1' den daha yüksekti (p <0,001). Korelasyon analizi, fQRSTa ve LV geometrisi arasında anlamlı bir korelasyon ortaya çıkardı (r = 0,525, p <0,001). Çoklu lineer regresyon analizi, fQRSTa' nın E/A oranı (β=0,104, p=0,045), sol ventrikül kitle indeksi (β=0,342, p<0,001), QTc (β=0,194, p<0,001), ve SV geometrisi (β=0,257, p<0,001) ile bağımsız olarak ilişkili olduğunu ortaya çıkarmıştır.*
Sonuç	*SV hipertrofisi olan hastaların, normal ventrikül veya konsantrik yeniden şekillenme olanlara kıyasla daha geniş fQRSTa ve daha uzun QT süresine sahip olduğu bulundu*
Anahtar Kelimeler	*sol ventrikül geometrisi; frontal QRS-T açısı; hipertansiyon*

INTRODUCTION

Chronic arterial hypertension (HT) causes pressure and volume changes in the myocardium, often results in a rise in left ventricular (LV) mass.[1] LV hypertrophy (LVH), which can traditionally be detected by echocardiography and electrocardiography, can significantly predict mortality and morbidity in cardiovascular diseases.[2,3] Left ventricular geometric patterns include normal LV structure, LVH, and concentric remodeling, affecting prognosis and LV function differently.[4-6] Previously, it has been reported that LV geometric patterns, particularly LVH, affect ventricular repolarization parameters in hypertensive patients.[7,8] Moreover, Keung et al. reported prolonged duration and higher homogeneity of ventricular repolarization in LVH.[9]

Frontal QRS-T angle (fQRSTa) is defined as the absolute difference between QRS and T wave axes on 12-lead ECG and is considered a parameter for ventricular repolarization.[10-12] Besides, fQRSTa helps for estimating clinical events such as the development of fatal ventricular arrhythmias or sudden death in cardiovascular diseases.[10,12,13] The main purpose here was to define the correlation between different LV patterns and fQRSTa in patients with HT.

MATERIALS and METHODS

This cross-sectional descriptive study was conducted according to the principles of the Declaration of Helsinki and approval for the study was obtained from the local Institutional Review Board (decision no: 2018/60). Written informed consents were obtained from all included patients. A total of 273 consecutive hypertensive patients without exclusion criteria admitted to our outpatient clinic at Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital between January 2019 and January 2020 were enrolled. Patients with secondary causes of HT, valvular heart disease (moderate to severe), symptoms of congestive heart failure, LV ejection fraction (EF) below 55%, arrhythmia, complete or incomplete bundle branch block, chronic renal failure, congenital heart disease, acute or chronic infectious or inflammatory disease, pregnancy, or chronic liver disease were excluded. By the European Society of Cardiology recommendations, HT was diagnosed in patient with systolic blood pressure above 140 mmHg or diastolic blood pressure above 90 mmHg measured in the supine position, or under the treatment of antihypertensive drug.[14] Diabetes mellitus (DM) was diagnosed with fasting blood glucose above 126 mg/dl or HbA1c above 6.5% or in the presence of hypoglycemic drug use.15 Body mass index (BMI) was calculated as weight/height^2 (kg/m^2).

Electrocardiography

Electrocardiography was performed using a 12-lead surface ECG (Nihon Kohden Corporation, Tokyo, Japan). Measurements were performed in supine position and at a paper speed of 25 mm/s and 10 mm/s voltage. All measurements were scanned and subjected to 400× magnification using Adobe Photoshop for minimizing errors. QRS duration was measured from the beginning of the QRS complex until the J point. QT interval was measured from the beginning of the QRS complex to the end of the T wave. QTc for heart rate was calculated using Bazett's formula: QTc=QT/√RR. QRS duration and corrected QT interval measurements were done on the precordium and the averages were obtained. For manual analyses, the end of the T wave was found using the threshold method16. The fQRSTa was defined as the absolute angle difference between frontal plane QRS and T wave axes (Figure 1). If FQRST angle was above 180°, it was subtracted from 360° and set to the minimum angle17. All differences between observers for QTc interval and frontal QRS-T angle were <5%.

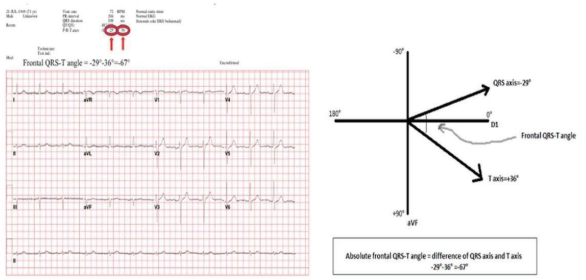

Figure 1: An illustration of the measurement of frontal QRS-T angle

Echocardiography

All participants underwent echocardiography by a single experienced operator who was blinded to the clinical status of the patients. The examination was performed using a Philips Epiq 7C machine (Philips Healthcare Andover, MA, USA). The LV dimensions, intraventricular septal wall thickness (IVSth), and posterior wall thickness (PWth) were measured in M-mode according to the guidelines of the ASE (American Society of Echocardiography). Ejection fraction (EF) was measured from apical four-chamber and two-chamber views using Simpson's method (modified). LV mass (LVM) was calculated by the Devereux equation as follows: LVM=0.8 [1.04(LVEDD + IVSth + PWth)3 – (LVEDD3)] + 0.6, where LVEDD stands for LV end-diastolic diameter. LVM index (LVMI) was obtained by dividing LVM by body surface area (LVMI/BSA). Relative wall thickness (RWth) was calculated as 2(PWth)/LVEDD at the end- diastole. RWth was considered increased when above 0.45. LV hypertrophy was defined as an LVMI value above 115 g/m2 for males and above 95 g/m2 for females.[12] Patients were divided into three groups based on LV hypertensive geometry as normal structure (non-LVH, normal RWth), concentric remodeling (non-LVH, increased RWth), and LVH.

Statistical analysis

We used the SPSS software (IBM, 21.0, 2012, Armonk, USA) for statistical analysis. Conformity to normal distribution was tested by the Kolmogorov-Smirnov test. Data are given as mean ± standard deviation, median (25 to 75 percentile), and number and percentage. Normally distributed quantitative variables were compared using the One-way ANOVA test. Post hoc subgroup tests were done using the LSD test. Non-normally distributed quantitative variables were compared using the Kruskal-Wallis test. Categorical variables were tested using the Chi-squared test, and subgroup analysis was done using the Bonferroni method. Correlations between fQRSTa and other variables were tested by Spearman correlation analysis. We conducted a multivariable linear regression model including the variables that were significantly correlated with fQRSTa in bivariate analyses. The level of statistical significance was taken as $p<0.05$.

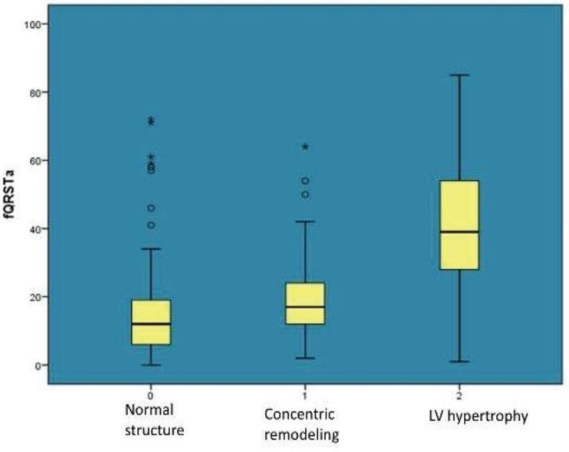

Figure 2: Box-plot graphs comparing frontal QRS-T angle with different left ventricular geometric patterns

RESULTS

A total of 273 outpatients with HT were included in this study. A comparison of baseline demographic, clinical, laboratory, echocardiographic, and electrocardiographic characteristics are given in Table 1. All the groups were balanced in terms of sex, DM, smoking, biochemical parameters, diastolic blood pressure, left ventricle end-diastolic, and end-systolic diameter. Group 2 had significantly higher age, BMI, systolic blood pressure, EF, IVSd, PWd, LVMI, RWT, QRS duration, and QTC duration than group 0. Group 2 was associated with a significantly lower E/A ra-

tio than group 0. Compared to group 0, fQRSTa was higher in group 1 (12 [6 – 19] vs. 17 [12 – 24], p=0.023) and group 2 (12 [6 – 19] vs. 39 [28 – 54], p<0.001). Also, fQRSTa was higher in group 2 compared to group 1 (p<0.001).

Correlation analysis revealed a significant correlation between fQRSTa and IVSd (r=0.395, p<0.001), PWd (r=0.389, p<0.001), Em to Am ratio (r=- 0.175, p=0.004), LVMI (r=0.491, p<0.001), RWT (r=0.295, p <0.001), QRS duration (r=0.163, p <0.007), QTc (r=0.419, p <0.001), and LV geometry (r=0.525, p<0.001). Multiple linear regression analysis revealed that fQRSTa was independently correlated with Em to Am ratio (β=0.104, p=0.045), LVMI (β=0.342, p<0.001), QTc (β=0.194, p<0.001), and LV geometry (β=0.257, p<0.001).

Table 1: Comparison of baseline demographic, clinical, laboratory, echocardiographic, and electrocardiographic characteristics of hypertensive patients

	Normal structure (n=128)	Concentric Remodeling (n=86)	LV Hypertrophy (n=59)	p
Age, years	47 ± 10	52 ± 10 a	54 ± 10a	<0.001
BMI, kg/m²	28.67 [25.41-32.23]	30.40 [27.68-32.89]	30.90 [27.77-33.23]a	0.011
Gender, male n (%)	67 (52.3)	39 (45.3)	33 (55.9)	0.414
Smoking, n (%)	34 (26.6)	18 (20.9)	13 (22.0)	0.597
Diabetes, n (%)	27 (21.1)	13 (22.0)	25 (29.1)a	0.380
SBP; mmHg	136.5 [123.8-150]	140 [132-153.9]	150 [139-159]a	0.001
DKB; mmHg	88.2 [80-95]	87.5 [80-95]	90 [83-100]	0.153
Total cholesterol, mg/dl	200±34	201±40	210±41	0.318
LDL-c, mg/dl	124 [104-140]	117 [101-143]	130 [108-152]	0.308
HDL-c, mg/dl	45 [38-52]	47 [40-57]	46 [36-54]	0.357
Triglyceride, mg/dl	132 [99-206]	145 [106-213]	145 [90-212]	0.711
Serum creatinine, mg/dl	0.80 [0.60-0.88]	0.76 [0.70-0.90]	0.80 [0.70-0.90]	0.236
Ejection fraction, %	65 [63-71]	65 [60-65]b	65 [63-70]	0.004
LVEDd, mm	48 [46-50]	47 [43-55]	50 [46-53]	0.506
LVESd, mm	29 [27-31]	28 [26-34]	31 [28-33]	0.124
IVSd, mm	10 [9-11]	11 [10-12]a	13 [12-14]a,c	<0.001
PWd, mm	9 [8-9]	11 [10-11]a	12 [11-12]a	<0.001
Em to Am ratio	1.16 [0.84-1.40]	0.88 [0.76-1.17]b	0.80 [0.68-0.97]b	<0.001
LVMI, g/m2	81 [70-86]	85 [74-98]a	122 [108-131]a,c	<0.001
RWT, mm	0.36 [0.33-0.39]	0.47 [0.44-0.52]a	0.47 [0.42-0.51]a	<0.001
QRS duration, ms	82 [78-88]	86 [78-90]	88 [80-96]a	0.001
QTc interval, ms	413±22	414±19	441±25a,c	<0.001
Frontal QRS-T angle, deg	12 [6-19]	17 [12-24]a	39 [28-54]a,c	<0.001

Abbreviations: BMI, body mass index; HDL-c, high-density lipoprotein cholesterol; IVSd, interventricular septum diameter; LDL-c, low-density lipoprotein cholesterol; LVEDd, left ventricle end-diastolic diameter; LVEF, left ventricle ejection fraction; LVESd, left ventricle end-systolic diameter; LVMI, left ventricular mass index; PWd, posterior wall diameter; RRI, renal resistive index.
a Significantly higher than group 0, b Significantly lower than group 0, c Significantly higher than group 1, d Significantly lower than group 0
Note: Quantitative variables with normal distribution are given as mean ± standard deviation, and without normal distribution are given as median [25 to 75 percentile].

Table 2: Bivariate and multivariate relationships between frontal QRS-T angle and clinical, demographic, echocardiographic, and electrocardiographic variables.

	Frontal QRS-T angle			
	Correlation coefficient	**p**	**Standardized β regressioncoefficient a**	**p**
Age	0.056	0.357		
Body mass index	0.002	0.970		
Systolic blood pressure	0.089	0.143		
Ejection fraction	0.035	0.570		
Septal wall diameter	0.395	< 0.001		
Posterior wall diameter	0.389	< 0.001		
Em to Am ratio	-0.175	0.004	0.104	0.045
Left ventricle mass index	0.491	< 0.001	0.342	< 0.001
Relative wall thickness	0.295	< 0.001		
QRS duration	0.163	0.007	-0.027	0.591
Corrected QT	0.419	< 0.001	0.194	< 0.001
Left ventricle geometry	0.525	< 0.001	0.257	< 0.001
a From multiple linear regression.				

DISCUSSION

The most significant finding obtained here was the higher fQRSTa width and longer QT duration in LVH patients in compared to those with normal ventricles or concentric remodeling. This association was tried to be explained through several structural and electrophysiological myocardial changes.[4] Electrical ventricular remodeling includes nonuniform prolonged action potential and a heterogeneous relation between refractory periods and conduction velocities of nearby myocardial regions. These are known as increased dispersion of ventricular repolarization, underlying distinct electrophysiological properties in epicardial, endocardial, and midmyocardial cells (M cells).[5] Based on the reflection of all these, it can be concluded that some simple tools can predict arrhythmic events caused by ventricular damage due to HT.

Studies showed that the myocardial depolarization and repolarization parameters determined in the ECG are related to cardiac outcomes. Mozos et al. found an association between HT and prolonged QT intervals with higher prevalence. QT intervals and T wave variables are reported to be closely linked in this patient group.[7] Another ECG parameter showing myocardial repolarization in the ECG is the QRS-T angle. Previously, several researchers cited difficulties in applying spatial QRSTa in clinical practice as the main tool for studying fQRSTa.[18,19] The challenges include high complexity, absence of standardization, and the potential need for expensive hardware and software for calculating spatial QRSTa.[18,19] Hence, we aimed to investigate the correlation between LV geometric patterns and fQRSTa in HT patients for a more straightforward and more practical clinical application. fQRSTa is a marker that indicates heterogeneous myocardial repolarization and electrically unstable myocardium.[10,11,17-19] These axes are expected to be in a similar direction under normal circumstances.

Yet, in myocardial ischemia and fibrosis, damaged or inhomogeneous regions in the myocardium leading to wider fQRSTa. Borleffs et al. associated wide fQRSTa with adverse clinical outcomes in ischemic heart diseas.[20] In 2008, DEFINITE investigators reported that for nonischemic cardiomyopathy patients with no pacemaker and mild to moderate symptoms, fQRSTa above 90 degrees could indicate a composite endpoint of mortality, cardioverter-defibrillator shock, or cardiac arrest.[21] A meta-analysis by Zhang et al. showed that both spatial QRS-T angle and fQRSTa carry promising prognostic information on all-cause mortality.[22] It seems to be evidence that strengthens

the correlation of these two ECG parameters. Previous research has demonstrated fQRSTa to predict cardiovascular mortality, sudden cardiac death, and heart failure (reduced or preserved EF).[23-26] Underlying abnormal fQRSTa are changes in myocardial ion channels that result in abnormal ventricular repolarization.[8] This impaired fQRSTa has been reported to increase a person's risk of malignant arrhythmia 16-fold. Accordingly, abnormalities of this measure are associated with many adverse cardiovascular outcomes, including fatal ventricular arrhythmia, sudden cardiac death.[22,24]

Ventricular structural disorders occurring in hypertensive patients are expressed as LV geometry.[27] Cardiovascular outcomes are found with a higher frequency in patients with LV hypertrophy (eccentric and concentric). Previously, several researchers reported worse clinical outcomes in patients with concentric remodeling than those with normal ventricular structure.[27-29] LV geometric changes are considered a preclinical form of cardiac failure and may be related to survival. In HT patients, deterioration of the LV structure occurs because of high blood pressure.[28,29]

Moreover, HT, but even preclinical blood pressure elevations may result in changes in LV geometry. This damage in the structure of the LV creates adverse effects on myocardial depolarization and repolarization, increasing cardiovascular outcomes and the risk of sudden cardiac death.[27] Simple parameters, which can predict these changes in patients with HT and correlated with remodeling structure, can increase treatment aggression by revealing risky patients. Saba et al. (2005) investigated the relationship between electrocardiographic parameters showing transmural repolarization dispersion (TRD) and LV geometry. In the study, the concept of TDR was determined by measuring the Tp-e distance. As a result of the study, it was determined that compared to normal LV geometry, the Tp-e interval was prolonged in LVH and shortened in concentric remodeling.[30] However, in this study, the Tp-e measurement is not standardized. In our study, the fQRSTa measurement was made by the same ECG device from the same center and it offers a standard and straightforward simple approach. In another study by Malmqvist et al., it was shown that many electrocardiographic repolarization such as QT dispersion, QT/RR ratio, JT dispersion, was more frequently prolonged in patients with impaired LV geometry.[31] In our study, following previous research, there was a strong association between fQRSTa and LVH in essential HT. Also, we determined that the fQRSTa was higher and the QT duration, which is the traditional parameter indicating repolarization, was longer compared to the patients with normal ventricular structure.

Limitations

This research had certain limitations. First, the patient population was relatively small. Second, manual measurements of electrophysiologic parameters off ECG tracing led to variability, consistent with the nature of similar research. The third limitation was the lack of quantification for myocardial ischemia.

CONCLUSION

In conclusion, we found wider fQRSTa and longer QT duration in the LVH group than the normal ventricles and concentric remodeling groups. Further research on a larger scale should aim to confirm these findings.

There are no conflicts of interest.

Written informed consent was obtained from patients who participated in this study.

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The study was approved by the Clinical Studies Ethical Committe of Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital by the decision no 2018/60 date: 09/06/2020

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