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Relationship Between Frontal QRS-T Angle and Ascending Aortic Dilatation: A Cross Sectional Study

Frontal QRS-T Açısı ve Asendan Aort Dilatasyonu Arasındaki İlişki: Kesitsel Bir Çalışma

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ABSTRACT Objective: Few studies have shown that certain myocardial repolarization markers from surface electrocardiogram (ECG) are associated with ascending aortic (AA) dilatation (AAD). We aimed to investigate the association between 12-lead surface ECG markers and AAD. **Material and Methods:** Consecutive patients without active complaints, who were admitted to the outpatient clinic for routine control, were included in the study. Transthoracic echocardiography (TTE) was performed to measure AA diameter. ECG markers, including QRS duration, TP-e interval, QTc interval, and frontal QRS-T angle were calculated. Patients were divided into two groups based on their AA diameter: those with an AA diameter ≥40 mm [AAD (+)] and those with an AA diameter <40 mm [AAD (-)]. Statistical analysis was performed to compare the two groups using a p value <0.05 as statistically significant. **Results:** Among the 251 patients, 31 (12.3%) had AAD. Patients with AAD had a significantly higher rate of coronary artery disease (CAD) history. Fragmented QRS, pathological Q-waves, longer P-maximum, P-minimum, P-dispersion, QRS duration, Tp-e duration, R peak time, and increased frontal QRS-T angle were more common in the AAD(+) group (all p<0.05). Correlation analysis revealed a significant correlation between the frontal QRS-T angle and AAD (R=0.379, p<0.001). In multivariate logistic regression analysis, AAD showed an independent association with the frontal QRS-T angle (OR: 3.886, 95% CI: 1.270-11.893, p=0.017) and history of CAD (OR: 10.689, 95% CI: 2.151-53.121, p=0.004). **Conclusion:** AAD was independently associated with a CAD history and frontal QRS-T angle.

Keywords: Electrocardiography; frontal QRS-T angle; ascending aorta; ascending aortic dilatation

ÖZET Amaç: Az sayıda çalışmada, yüzey elektrokardiyografisinden (EKG) elde edilen bazı miyokardiyal repolarizasyon belirteçlerinin asendan aort (AA) dilatasyonu (AAD) ile ilişkili olduğu gösterilmiştir. Bu çalışmada, 12 derivasyonlu yüzey EKG belirteçleri ile AAD arasındaki ilişkiyi araştırmayı amaçladık. **Gereç ve Yöntemler:** Aktif şikâyeti olmayan, rutin kontrol için polikliniğe başvuran ardışık hastalar çalışmaya dâhil edildi. AA çapını ölçmek için transtorasik ekokardiyografi (TTE) yapıldı. QRS süresi, TP-e aralığı, QTc aralığı ve frontal QRS-T açısı gibi EKG belirteçleri hesaplandı. Hastalar AA çaplarına göre iki gruba ayrıldı: AA çapı ≥ 40 mm olanlar [AAD (+)] ve AA çapı <40 mm olanlar [AAD (-)]. İki grubu karşılaştırmak için istatistiksel analiz yapıldı ve p değeri <0,05 istatistiksel olarak anlamlı kabul edildi. **Bulgular:** Çalışmaya dâhil edilen 251 hastanın 31'inde (%12,3) AAD vardı. AAD'li hastalarda koroner arter hastalığı (KAH) öyküsünün anlamlı derecede yüksek olduğu görülmüştür. AAD (+) grupta; EKG parametreleri arasından fragmante QRS, patolojik Q dalgaları, daha uzun P-maksimum, P-minimum, P-dispersiyonu, QRS süresi, Tp-e süresi, R pik zamanı ve artmış frontal QRS-T açısı daha yaygındı (tümü p<0,05). Korelasyon analizi frontal QRS-T açısının AAD ile ilişkili olduğunu göstermiştir (R=0,379, p<0,001). Çok değişkenli lojistik regresyon analizinde, AAD ile frontal QRS-T açısı [göreceli olasılıklar oranı (odds ratio "OR"): 3,886, %95 güven aralığı (confidence interval "CI") 1,270-11,893, p=0,017] ve KAH öyküsü (OR: 10,689, %95 CI 2,151-53,121, p=0,004) arasında bağımsız bir ilişki olduğu bulunmuştur. **Sonuç:** Çalışmamızda, AAD ile frontal QRS-T açısı ve KAH öyküsü arasında anlamlı bir ilişki bulunmuştur.

Anahtar Kelimeler: Elektrokardiyografi; frontal QRS-T açısı; asendan aort; asendan aort dilatasyonu

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Electrocardiography (ECG) is a widely used non-invasive diagnostic tool for evaluating cardiac function and identifying arrhythmias. Recent studies have demonstrated that ECG markers may provide valuable information about heart structure, including left ventricular (LV) ejection fraction (LVEF), heart chambers, and calcification of heart valves, offering a potential opportunity for early risk stratification and monitoring.[1]

Projection of the three-dimensional spatial QRS and T vectors onto the frontal plane produces frontal QRS and T vectors, respectively. The angle between the frontal QRS and T vectors is called the frontal QRS-T angle.[2] The frontal QRS-T angle can be easily calculated from a 12-lead surface ECG as the absolute value of the difference between the frontal plane QRS axis and T axis and has been shown to correlate well with the three-dimensional spatial QRS-T angle for risk prediction.[3] The QRS-T angle reflects deviations between ventricular depolarization and repolarization, and its prognostic implications have been extensively demonstrated in both the general population and specific subpopulations.[4] The frontal QRS-T angle has been shown to be significantly associated with myocardial ischemia, myocardial hypertrophy, ventricular arrhythmias, hypertension (HT), diabetes mellitus (DM), new coronary heart events, cardiovascular (CV), and all-cause mortality.[5,6]

There is a well-known relationship between ECG and LV remodelling.[7] Because of myocardial structural changes, it is likely that some ECG parameters will change. These ECG changes may provide insights into ascending aortic (AA) remodeling based on the relationship between LV and AA remodeling. Previous studies have shown that patients with AA aneurysms have increased aortic stiffness and myocardial structural changes such as an increased LV mass index.[8,9] Moreover, previous studies have shown that the LV mass index decreases, especially after aortic aneurysm or stenosis repair.[10,11] Two recent studies have shown an association between myocardial repolarization parameters on ECG, including QTc and Tp-e, and AA aneurysm.[8,9] We designed a study focusing on ECG parameters to investigate the potential associations between different ECG markers and AA dilatation (AAD) in a selected cohort of patients. The findings of this study may have important clinical implications, as the identification of ECG markers associated with AAD could potentially enhance risk stratification and monitoring strategies for patients at risk of adverse CV events related to AAD.

MATERIAL AND METHODS

STUDY DESIGN AND PARTICIPANTS

We conducted a cross-sectional observational cohort study to investigate the potential associations between ECG markers and AAD. Patients attending the cardiology clinic of Recep Tayyip Erdoğan University Faculty of Medicine Hospital for routine control without complaints were included in the study. All patients included in the study underwent transthoracic echocardiography (TTE) and 12-lead surface ECG. We used a cut-off value of 40 mm for AAD, as recommended by the current guidelines.[12] Patients were divided into two groups: those with an AA diameter of 40 mm or more [AAD (+)] and those with an AA diameter less than 40 mm [AAD (-)]. Statistical analysis was conducted by comparing the two groups to determine any significant associations between ECG markers and AAD.

Patients on antiarrhythmic drugs, patients with connective tissue disorders (Marfan, Ehlers-Danlos, or Loey-Dietz syndrome), bicuspid aortic valve, acute coronary syndrome, LVEF<50%, left and right bundle branch block, pacemaker rhythm, history of cerebrovascular disease, acute or chronic renal failure, active inflammatory disease, myocarditis, cardiomyopathy, cardiac surgery with any indication, anemia, secondary HT, significant heart valve disorder, endocrinological conditions, electrolyte imbalance, pulmonary thromboembolism, malignancy, or end-stage liver disease were excluded from the study.

This study was conducted in accordance with the "Declaration of Helsinki" and approved by the "Institutional Review Committee". The Recep Tayyip Erdoğan University Non-Invasive Clinical Research Ethics Committee approved the study (The ethics committee decision date and decision number: November 10, 2023, E-64960800-799-229073237). All patients were informed of the study, and written consent was obtained for the study.

DATA COLLECTION AND ECG ANALYSIS

Demographic and clinical data, including age, sex, comorbidities, and medication use, were collected from electronic medical records.

Standard 12-lead surface ECGs, recorded at a paper speed of 25 mm/s and a voltage of 10 mm/mV (Nihon Kohden, ECG 1250-12 ECG Device), were analysed by two independent cardiologists blinded to the patients' clinical information. ECGs were scanned at 600 DPI and magnified 10-fold. ECG images were analysed using the online tool "GeoGebra Software, version 4.2 (International GeoGebra Institute, IGI, Austria)" (accessible at https://www.geogebra.org). Upon calibrating one small square on the ECG to represent 40 ms, the software allowed users to manually draw lines on the ECG and measure the time interval in milliseconds between two points.

The frontal plane QRS axis and T axis were acquired through automated calculations performed by the ECG device. The frontal QRS-T angle was determined by calculating the absolute difference between the frontal plane QRS axis and T axis. In cases where the angle exceeded 180°, it was adjusted to an acute angle by subtracting it from 360°.[2]

The QRS duration was defined as the time interval between the onset of the q or R-wave and the end of the S-wave (J point). The longest QRS duration among leads V1 to V6 has been recorded.[13]

A fragmented QRS is characterized by the existence of additional deflections or notches within the QRS complex, which indicates abnormal depolarization patterns. The presence and characteristics of fragmented QRS patterns in corresponding leads have been documented.[14]

The presence of pathological Q-waves on ECG was identified. The duration and depth of the Q-wave were measured in the leads where they appeared most prominently. Pathologic Q-waves were defined as those with a duration greater than 40 ms and a depth exceeding 25% of the subsequent R- or S-waves.[14]

The interatrial block was assessed by examining the morphology of the P-wave in various leads. This investigation focused on identifying prolonged or altered P-wave duration, morphology, or axis deviation. Interatrial block was diagnosed using specific criteria, including a P-wave duration exceeding 120 ms, with or without a biphasic negative P-wave in leads II, III, and aVF.[13]

R peak time was assessed by measuring the duration from the onset of the QRS complex to the peak of the R-wave and quantified in milliseconds.[13]

P-maximum and P-minimum were determined by identifying the maximum and minimum durations of the P-waves. The onset and offset points of the P-wave were located in the lead where it was most prominent. P-dispersion was calculated by subtracting the minimum duration of the P-wave from the maximum duration. This calculation provided a measure of P-wave dispersion across different leads, and the results were reported in milliseconds.[13]

The duration of the QT interval was measured in milliseconds, from the beginning of the Q-wave to the end of the T-wave. The QTc interval, which accounts for the heart rate, was calculated using Bazett's formula ($QTc=QT/\sqrt{RR}$) based on the RR interval.[13]

The TP interval was measured from the end of the T-wave to the onset of the subsequent P-wave. This interval represents the period from the end of ventricular repolarization to the onset of atrial depolarization.[8] Tp-e duration was measured as the duration from the peak of the T-wave to the end of the T-wave in leads V2 or V5, where the T-wave exhibited the most prominent features.[8,13]

The patients' baseline characteristics were recorded during the interviews. The diagnosis of HT was based on the current use of antihypertensive medication or a mean blood pressure of 135/85 mmHg or higher at home twice a day for two weeks in a quiet environment, with at least one hour's rest and without smoking or coffee consumption. Individuals with a fasting blood glucose level of 126 mg/dL or above, glycosylated hemoglobin level of 6.5% or above, or use of antidiabetic drugs were diagnosed with DM. Individuals who currently smoked at least one cigarette per day or who had quit smoking within the last 12 months were considered smokers. Body mass index (BMI) was calculated by dividing the weight in kilograms (kg) by the square of the height in meters (m^2). ($BMI=weight\ [kg]/height\ [m]^2$).

AORTIC DIAMETER MEASUREMENT

Diameter of the AA was measured by two experienced echocardiographers using TTE with a high-resolution ultrasound system (Vivid E95, Ultra Edition, GE Healthcare, Horten, Norway). The AA diameter was measured at the widest part of the AA that could be seen on TTE, as recommended by the American Society of Echocardiography guidelines.[15] The average of two measurements was used for the analysis.

STATISTICAL ANALYSIS

Statistical analysis of the cases was performed using SPSS version 26, developed by SPSS Inc. (Chicago, Armonk, NY, USA). The data distribution of the variables was assessed using visual methods, including histograms and probability plots, as well as analytical methods, such as the Kolmogorov-Smirnov and Shapiro-Wilk tests, to determine whether they followed a normal distribution. Based on the diameter of the AA (≥40 mm and <40 mm), patients were divided into two groups to compare the obtained parameters. Continuous variables with a normal distribution are presented as mean±standard deviation, while categorical variables are presented as percentages. The median values of the 25th and 75th percentiles are reported for continuous variables that did not follow a normal distribution. To compare the means between the groups, Student's t-test was used for normally distributed variables, while the Mann-Whitney U test was used for non-normally distributed variables. Categorical variables were compared using either the chi-squared test or Fisher's exact chi-squared test depending on the sample size.

Parameters with statistically significant differences in the univariate analysis were first evaluated using univariate logistic regression analysis to determine their relationship with AAD. As a result of this analysis, multivariate logistic regression (backward method) analysis was performed by using the parameters marked with "*" among the parameters that remained significant. The results are presented as 95% confidence intervals (CI) and odds ratios (OR). Before this analysis, a logarithmic transformation was performed because the frontal QRS-T angle did not show a normal distribution. Correlation analyses were performed using Pearson or Spearman correlation tests. The ROC curve was analysed to calculate the cut-off value of the statistically significant parameters. Statistical significance was set at $p<0.05$.

RESULTS

In total, 251 consecutive patients (141 men and 110 women) were included in the analysis. The age distribution of the samples participating in the study was 54 (45-62). There were 220 individuals in the group with an AA diameter of <40 mm and 31 in the group with an AA diameter of ≥40 mm. The demographic, laboratory, medication, and ECG parameters were compared between the two groups (Table 1). This analysis showed changes in many parameters on the 12-lead surface ECG in patients with AAD. Age (61.8±10.5 vs. 52.1±12.8, p<0.001), DM (38.7% vs. 16.8, p=0.004), HT (71.0% vs. 39.5, p<0.001), coronary artery disease (CAD) history (87.1% vs. 36.4, p<0.001), the use of beta blockers (35.9% vs. 17.7, p=0.020), angiotensin receptor blockers (29.0% vs. 14.5, p=0.041), diuretics (19.4% vs. 4.5, p=0.012), and oral anti-diabetics drugs/insulin (38.7% vs. 15.9, p=0.002) were higher in the AAD (+) group, reaching statistical significance. The proportion of the male gender was higher in the AAD (+) group (83.9% vs. 52.3, p<0.001). As expected, serum creatinine was higher in the AAD (+) group (0.98±0.27 vs. 0.83±0.22, p<0.001). Fragmented QRS (38.7% vs. 19.1%, p=0.013) and pathological Q-wave (47.1% vs. 17.9%, p=0.009) were more common in the AAD (+) group. However, there was also a prolongation in parameters related to repolarization and depolarization of the ventricles. Of these parameters, QRS duration (97.8±12.6 vs. 92.2±10.8, p=0.001) and Tp-e duration (103.8±11.7 vs. 95.1±14.4, p<0.001) increased in the group with larger AA. Notably, the frontal QRS-T angle [46.5 (22.0-116.0) vs. 23.0 (12.0-38.2)], (p<0.001) and the R peak time (37.2±11.4 vs. 32.1±8.2°, p=0.001) were found to be increased. Moreover, P-maximum, P-minimum and P-dispersion (106.1±12.9 vs. 103.9±11.8, p=0.012, 60.8±8.6 vs. 59.7±9.3, p<0.001, 45.7±9.3 vs. 44.6±6.8, p=0.001; respectively) were higher in the AAD (+) group.

After the parameters that showed statistically significant differences between the two groups were

TABLE 1: Comparison of baseline characteristics and electrocardiographic data in the study population.

Variable	Ascending aortic diamater <40 mm (n=220) (AAD-)	Ascending aortic diamater ≥40 mm (n=31) (AAD+)	p value
Demographic data			
Age (years)	52.1±12.8	61.8±10.5	<0.001
Sex (male) n (%)	115 (52.3)	26 (83.9)	<0.001
BMI (kg/m^2)	28.9±4.9	30.6±4.3	0.709
HL n (%)	51 (23.2)	11 (35.5)	0.137
DM n (%)	37 (16.8)	12 (38.7)	0.004
HT n (%)	87 (39.5)	22 (71.0)	<0.001
History of CAD n (%)	80 (36.4)	27 (87.1)	<0.001
Current smoking n (%)	90 (41.3)	14 (45.2)	0.682
Medications in use			
Beta blocker n (%)	39 (17.7)	11 (35.9)	0.020
ACE-i n (%)	42 (19.1)	10 (32.3)	0.090
ARB n (%)	32 (14.5)	9 (29.0)	0.041
Statin n (%)	29 (13.2)	3 (9.7)	0.584
CCB n (%)	28 (12.7)	8 (25.8)	0.052
Diuretic n (%)	10 (4.5)	6 (19.4)	0.012
OAD/insulin n (%)	35 (15.9)	12 (38.7)	0.002
Laboratory data			
Fasting glucose (mg/dL)	109.1±37.3	135.8±52.9	0.545
WBC (103/μL)	8.07±2.3	9.30±2.5	0.170
˙CRP (mg/dL)	4.2 (2.5-6.2)	4.2 (3.0-5.9)	0.810
Hemoglobin (g/dL)	13.8±1.7	14.2±1.6	0.391
Serum creatinine (mg/dL)	0.83±0.22	0.98±0.27	<0.001
Electrocardiographic data			
Fragmanted QRS n (%)	42 (19.1)	12 (38.7)	0.013
Pathological Q wave n (%)	24 (17.9)	10 (47.1)	0.009
Interatrial block n (%)	31 (14.3)	5 (16.1)	0.785
˙QRS axis (°)	21 (12.7-31.2)	19 (8.2-36.5)	0.870
˙T axis (°)	37 (29.5-60.5)	70 (41.2-103.0)	<0.001
˙Frontal QRS-T angel (°)	23.0 (12.0-38.2)	46.5 (22.0-116.0)	<0.001
QRS duration (ms)	92.2±10.8	97.8±12.6	0.001
P maximum (ms)	103.9±11.8	106.1±12.9	0.012
P minimum (ms)	59.7±9.3	60.8±8.6	<0.001
P dispersion (ms)	44.6±6.8	45.7±9.3	0.001
QTc interval (ms)	412.5±26.1	420.1±26.1	0.239
QT interval (ms)	385.8±34.4	395.8±36.4	0.503
TP interval (ms)	293±116	273±93	0.492
Tp-e duration (ms)	95.1±14.4	103.8±11.7	<0.001
R peak time (ms)	32.1±8.2	37.2±11.4	0.001

˙Median, interquartile range [range, (25% percentile-75% percentile)]; Continuous variables are given as mean±standard deviation; AAD: Ascending aortic dilatation; BMI: Body mass index; HL: Hyperlipidemia; DM: Diabetes mellitus; HT: Hypertension; CAD: Coronary artery disease; ACE-i: Angiotensin converting enzyme inhibitor; ARB: Angiotensin receptor blocker; CCB: Calcium channel blocker; OAD: Oral antidiabetic drugs; WBC: White blood cell; CRP: C-reactive protein; Tp-e: T peak to end.

evaluated using univariate logistic regression analysis, we created a model for multivariate logistic regression analysis using the parameters marked with "*" among the parameters that remained significant (Table 2). As a result of this analysis, a history of CAD (OR: 10.689, 95% CI 2.151-53.121, p=0.004) and frontal QRS-T angle (OR: 3.886, 95% CI 1.270-11.893, p=0.017) were independently associated with AAD. A strong statistical relationship between the frontal QRS-T angle and AAD is shown in Figure 1

TABLE 2: Univariate and multivariate logistic regression analyses of the variables.

	Univariate			Multivariate		
Variable	**OR**	**95% CI lower-upper**	**p value**	**OR**	**95% CI lower-upper**	**p value**
Age (years)*	1.069	1.033-1.107	<0.001			
Sex (male)*	4.748	1.759-12.81	0.002			
DM*	3.124	1.397-6.983	0.006			
HT*	3.737	1.644-8.495	0.002			
History of CAD*	11.81	3.990-34.97	<0.001	10.689	2.151-53.121	0.004
Beta blocker*	2.553	1.132-5.755	0.024			
ARB*	2.403	1.016-5.687	0.046			
Diuretic*	5.040	1.688-15.047	0.004			
OAD/insulin	3.338	1.487-7.489	0.003			
Serum creatinine*	10.25	2.417-43.53	0.002			
Fragmented QRS*	2.677	1.206-5.940	0.015			
Pathological Q Wave*	3.247	1.300-8.247	0.012			
R-peak time*	1.058	1.019-1.099	0.003			
Frontal QRS-T angel (log)*	7.339	2.629-20.48	<0.001	3.886	1.270-11.893	0.017
QRS duration*	1.041	1.009-1.075	0.012			
TP-E interval*	1.049	1.017-1.081	0.002			
P maximum	1.017	0.983-1.052	0.334			
P minimum	1.012	0.972-1.053	0.560			
P dispersion	1.021	0.966-1.079	0.468			
T axis (log)*	12.11	3.300-44.453	<0.001			

*Parameters included in the multivariate logistic regression analysis are indicated by; OR: Odds ratio; CI: Confidence interval; DM: Diabetes mellitus; HT: Hypertension; CAD: Coronary artery disease; ARB: Angiotensin receptor blocker; OAD: Oral antidiabetic drug.

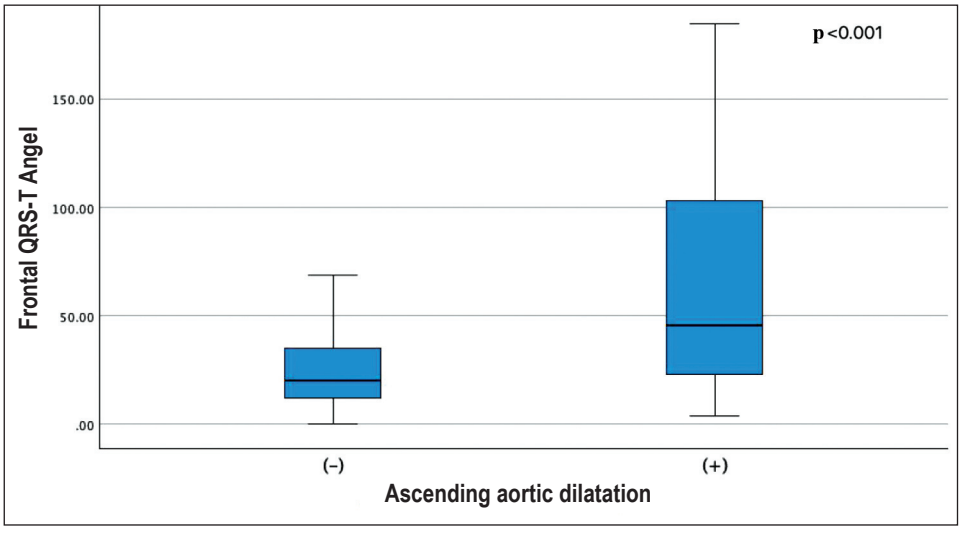

FIGURE 1: The graph illustrate that significant association between frontal QRS-T angel and ascending aortic dilatation (AAD).

(p<0.001). Correlation analysis showed that the frontal QRS-T angle correlated with AAD (R=0.379, p<0.001) (Figure 2). In ROC analysis, the frontal QRS-T angle had an area under the curve value of 0.742 (p<0.001) (Figure 3).

DISCUSSION

The association between ECG markers and AAD is a topic of growing interest in CV research. The present study adds to the existing literature by providing

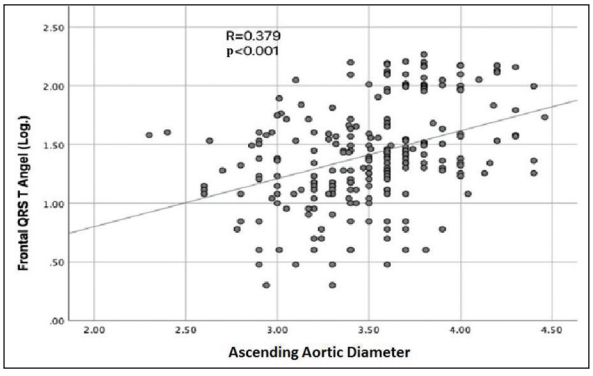

FIGURE 2: Correlation graph between ascending aortic diameter and frontal QRS-T angel.

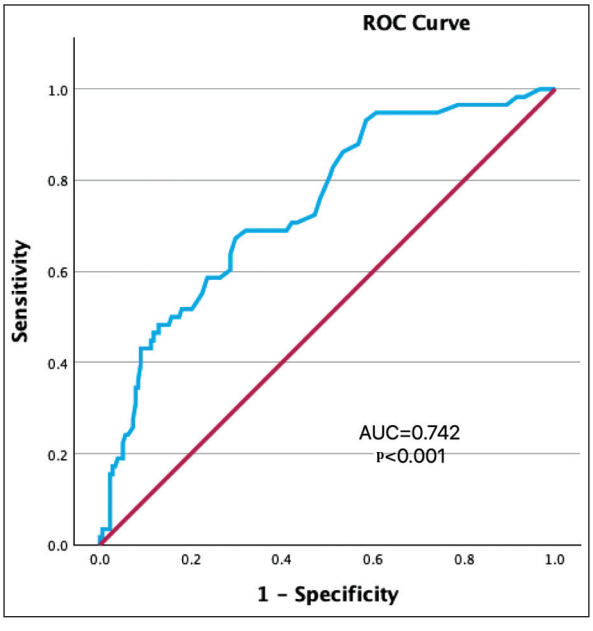

FIGURE 3: Frontal QRS-T angel had an AUC value of 0.742 with p<0.001. AUC: Area under the curve; ROC: Receiver operating characteristic.

evidence of a significant association between frontal QRS-T angle on 12-lead surface ECG and a history of CAD with AAD. Although different studies have focused on the relationship between myocardial depolarization parameters and AAD, to our knowledge, this study is the first to demonstrate an independent relationship between frontal QRS-T angle and AAD.

Our hypothesis in this study was that AA remodeling is not independent of LV remodeling; therefore, ECG parameters may change with LV remodeling in patients with AAD. A study by Topuz et al. showed that echocardiographic diastolic parameters were impaired in patients with AA aneurysms and that ECG-derived repolarization parameters such as QT dispersion, QTc, and Tp-e were prolonged.[8] In a study by Boduroglu and Son the relationship between the Tp-e interval and its ratio in QT and AA aneurysms was evaluated. Correlation analyses showed a relationship between AA aneurysm and several transmural dispersions of repolarization parameters, including QTc and Tp-e.[9] These studies have emphasized that diastolic function of the LV is particularly impaired in patients with AA aneurysms, predisposing them to arrhythmogenic conditions. Additionally, these studies show that LV structure changes with AAD, which in turn alters ECG parameters, especially depolarization and repolarization parameters. The results of the present study support these findings.

The frontal QRS-T angle, which represents the frontal orientation of the QRS complex and the T-wave on ECG, has been proposed as a marker of ventricular depolarization and repolarization heterogeneity.[16] This parameter has been shown to have a prognostic value in patients with and without a history of cardiovascular disease.[17,18] In a review by Oehler et al., a statistically significant correlation was observed between QRS-T angle and sudden cardiac death, ventricular arrhythmias, CV mortality, and all-cause mortality.[2] In a study by Kurisu et al., in patients with advanced chronic kidney disease, a wide QRS-T angle was correlated with LV remodeling, which can lead to conditions such as an enlarged LV end-diastolic volume or diminished LVEF.[19] In a study conducted by Li et al., optimized heart failure therapy for idiopathic dilated heart failure patients led to a significant reduction in the QRS-T angle.[20] Furthermore, our study revealed a significant association between the frontal QRS-T angle and AAD, indicating that changes in the frontal orientation of the QRS complex and T-wave may reflect structural changes in the aorta. This may be related to altered mechanical properties of the AA wall or changes in the electrical conduction system, leading to changes in AA remodeling. Moreover, it can be hypothesized that structural changes and altered electrical conduction patterns resulting in the development of depolarization and repolarization heterogeneity in the LV and atrium also affect AA remodeling. Supporting the results of our study, a recent study by Ergül

et al. showed a strong correlation between AAD with left atrial function and LV mass index in patients with normal LV systolic function, which can affect the QRS-T angle.[21]

Our study also showed an independent association between a history of CAD and AAD. Furthermore, pathological Q-waves and fragmented QRS, which may suggest a history of CAD, were associated with AAD in our study. This strong association may be due to the common risk factors shared by AAD and CAD, such as HT and hyperlipidemia. In a study by Yildiz et al., aortic strain and distensibility, which indicate aortic elasticity, were strongly associated with the severity of CAD.[22] In a similar study by Lu and Liu the elasticity indices of the AA correlated well with the severity of coronary stenosis.[23] Furthermore, rather than coexisting CAD and AAD, patients with CAD are more likely to be at a higher risk of AAD in the long term.[24] Moreover, LV remodeling is also expected in patients with CAD; similarly, remodeling of the AA may be observed in the long term.

In patients with HT, increased pressure in the aorta can lead to changes in its structure and function. In a study on prehypertensive patients, the QRS-T angle was shown to be increased compared to the normotensive group.[25] A recent study by Tanriverdi et al. showed that the frontal QRS-T angle is a strong predictor of LV hypertrophy in patients with HT.[26] In the present study, as expected, the rate of patients with HT was higher in the AAD group. However, this did not reach statistical significance in the logistic regression analysis. Due to the design of our study, we did not evaluate the relationship between HT and frontal QRS-T angle. Nevertheless, HT can be considered as a common factor that contributes to aortic dilatation and increases the frontal QRS-T angle. In a study by Gür et al., a strong correlation was observed between QT dispersion and aortic elastic indicators in newly diagnosed hypertensive patients.[27] However, in our study, although there was no significant relationship between the QT and QTc interval with AAD, there was a significant relationship between Tp-e and AAD. Nonetheless, this relationship was not statistically significant in the logistic regression analysis.

There are studies in the literature on detecting aortic valve stenosis and aortic and mitral valve regurgitation using artificial intelligence (AI)-assisted ECG analysis. The basic rationale of these studies is similar to our hypothesis that valvular diseases lead to cardiac remodeling with concomitant electrocardiographic changes.[28,29] In our study, the correlation between the frontal QRS-T angle and AA may offer insights into aortic changes through AI-based systems using ECG in the future. Further research is required to confirm this relationship.

The clinical implications of this study are as follows. ECG is a noninvasive, cost-effective, and widely available diagnostic tool routinely used in clinical practice. Our study suggests that simple ECG markers, such as the frontal QRS-T angle, may serve as valuable indicators for identifying individuals at risk for AAD. Early detection of AAD is crucial for timely intervention and prevention of life-threatening complications, such as aortic aneurysm rupture or aortic dissection. Incorporating these ECG markers into routine clinical practice may aid in the risk stratification and identification or close monitoring of high-risk individuals who may require further evaluation and intervention.

LIMITATIONS

This study has several limitations that need to be acknowledged. First, its cross-sectional and observational design introduces the possibility of a selection bias and limits its ability to establish causal relationships. Prospective, longitudinal studies would be beneficial to confirm these findings and investigate the temporal relationship between the frontal QRS-T angle and aortic health. Second, intra- and inter-observer variability results for aortic diameters and ECG parameters were not provided in this study. Third, this study was conducted at a single centre, which may restrict the generalizability of the results to other populations. Conducting large-scale, multicentre studies would be valuable for validating these findings and improving their external validity. The sample size was relatively small and further studies with larger cohorts are necessary to validate our results. Finally, although efforts were made to control for potential confounders, residual confounding fac-

tors may still not have been accounted for in the analysis.

CONCLUSION

Our study provides evidence of an independent association between the frontal QRS-T angle and a history of CAD with AAD. The underlying mechanisms linking frontal QRS-T angle and AAD remain unclear and require further investigation through experimental and mechanistic studies.

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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: *Hüseyin Durak, Mustafa Çetin, Nadir Emlek;* ***Design:*** *Hüseyin Durak, Mustafa Çetin;* ***Control/Supervision:*** *Elif Ergül, Hakan Duman, Muhammet Öztürk;* ***Data Collection and/or Processing:*** *Muhammet Öztürk, Elif Ergül, Ali Gökhan Özyıldız;* ***Analysis and/or Interpretation:*** *Mustafa Çetin, Hüseyin Durak;* ***Literature Review:*** *Mustafa Çetin, Hüseyin Durak, Elif Ergül;* ***Writing the Article:*** *Hüseyin Durak, Ali Gökhan Özyıldız, Elif Ergül;* ***Critical Review:*** *Mustafa Çetin, Hakan Duman, Madir Emlek;* ***References and Fundings:*** *Mustafa Çetin, Hakan Duman;* ***Materials:*** *Mustafa Çetin, Elif Ergül, Muhammet Öztürk.*

REFERENCES

1. Harris PR. The Normal Electrocardiogram: Resting 12-Lead and Electrocardiogram Monitoring in the Hospital. Crit Care Nurs Clin North Am. 2016;28(3):281-96. [Crossref] [PubMed]
2. Oehler A, Feldman T, Henrikson CA, Tereshchenko LG. QRS-T angle: a review. Ann Noninvasive Electrocardiol. 2014;19(6):534-42. [Crossref] [PubMed] [PMC]
3. Zhang ZM, Prineas RJ, Case D, Soliman EZ, Rautaharju PM; ARIC Research Group. Comparison of the prognostic significance of the electrocardiographic QRS/T angles in predicting incident coronary heart disease and total mortality (from the atherosclerosis risk in communities study). Am J Cardiol. 2007;100(5):844-9. [Crossref] [PubMed] [PMC]
4. Macfarlane PW. The frontal plane QRS-T angle. Europace. 2012;14(6):773-5. [Crossref] [PubMed]
5. Zhang X, Zhu Q, Zhu L, Jiang H, Xie J, Huang W, et al. Spatial/frontal QRS-T angle predicts all-cause mortality and cardiac mortality: a meta-analysis. PLoS One. 2015;10(8):e0136174. [Crossref] [PubMed] [PMC]
6. Wang D, Jiang L, Cai Z, Yang Y, Li J. View on the Clinical Value of QRS-T Angle. Journal of Clinical and Nursing Research. 2021;5(2):50-4. [Crossref]
7. Bacharova L, Estes EH. Left Ventricular Hypertrophy by the Surface ECG. J Electrocardiol. 2017;50(6):906-8. [Crossref] [PubMed]
8. Topuz M, Genç Ö, Acele A, Koc M. Myocardial repolarization is affected in patients with ascending aortic aneurysm. J Electrocardiol. 2018;51(4):738-41. [Crossref] [PubMed]
9. Boduroglu Y, Son O. Assessment of Tp-Te interval and Tp-Te/Qt ratio in patients with aortic aneurysm. Open Access Maced J Med Sci. 2019;7(6):943-8. [Crossref] [PubMed] [PMC]
10. Ikeno Y, Truong VTT, Tanaka A, Prakash SK. The effect of ascending aortic repair on left ventricular remodeling. Am J Cardiol. 2022;182:89-94. [Crossref] [PubMed]
11. Sarubbi B, Calvanese R, Cappelli Bigazzi M, Santoro G, Giovanna Russo M, Calabrò R. Electrophysiological changes following balloon valvuloplasty and angioplasty for aortic stenosis and coartaction of aorta: clinical evidence for mechano-electrical feedback in humans. Int J Cardiol. 2004;93(1):7-11. [Crossref] [PubMed]
12. Writing Committee Members; Isselbacher EM, Preventza O, Hamilton Black Iii J, Augoustides JG, Beck AW, Bolen MA, et al. 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2022;80(24):e223-e393. [PubMed] [PMC]
13. Surawicz B, Childers R, Deal BJ, Gettes LS, Bailey JJ, Gorgels A, et al; American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009;53(11):976-81. [Crossref] [PubMed]
14. Das MK, Khan B, Jacob S, Kumar A, Mahenthiran J. Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation. 2006;113(21):2495-501. [Crossref] [PubMed]
15. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, et al. Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults: Recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr. 2019;32(1):1-64. [Crossref] [PubMed]
16. Cardoso CR, Leite NC, Salles GF. Factors associated with abnormal T-wave axis and increased QRS-T angle in type 2 diabetes. Acta Diabetol. 2013;50(6):919-25. [Crossref] [PubMed]

17. Walsh JA 3rd, Soliman EZ, Ilkhanoff L, Ning H, Liu K, Nazarian S, et al. Prognostic value of frontal QRS-T angle in patients without clinical evidence of cardiovascular disease (from the Multi-Ethnic Study of Atherosclerosis). Am J Cardiol. 2013;112(12):1880-4. [Crossref] [PubMed] [PMC]

18. Karadeniz FÖ, Altuntaş E. Correlation between frontal QRS-T angle, Tp-e interval, and Tp-e/QT ratio to coronary artery severity assessed with SYNTAX score in stable coronary artery disease patients. J Arrhythm. 2022;38(5):783-9. [Crossref] [PubMed] [PMC]

19. Kurisu S, Nitta K, Watanabe N, Ikenaga H, Ishibashi K, Fukuda Y, et al. Associations of frontal QRS-T angle with left ventricular volume and function derived from ECG-gated SPECT in patients with advanced chronic kidney disease. Ann Nucl Med. 2021;35(6):662-8. [Crossref] [PubMed]

20. Li SN, Zhang XL, Cai GL, Lin RW, Jiang H, Chen JZ, et al. Prognostic significance of frontal QRS-T angle in patients with idiopathic dilated cardiomyopathy. Chin Med J (Engl). 2016;129(16):1904-11. [Crossref] [PubMed] [PMC]

21. Ergül E, Özyıldız AG, Emlek N, Özyıldız A, Durak H, Duman H. The relationship between ascending aortic diameter with left atrial functions and left ventricular mass index in a population with normal left ventricular systolic function. Echocardiography (Mount Kisco, NY). 2023;40(7):687-94. [Crossref] [PubMed]

22. Yildiz A, Gur M, Yilmaz R, Demirbag R. The association of elasticity indexes of ascending aorta and the presence and the severity of coronary artery disease. Coron Artery Dis. 2008;19(5):311-7. [Crossref] [PubMed]

23. Lu Q, Liu H. Correlation of ascending aorta elasticity and the severity of coronary artery stenosis in hypertensive patients with coronary heart disease assessed by M-mode and tissue Doppler echocardiography. Cell Biochem Biophys. 2015;71(2):785-8. [Crossref] [PubMed]

24. Jackson V, Eriksson MJ, Caidahl K, Eriksson P, Franco-Cereceda A. Ascending aortic dilatation is rarely associated with coronary artery disease regardless of aortic valve morphology. J Thorac Cardiovasc Surg. 2014;148(6):2973-80.e1. [Crossref] [PubMed]

25. Bağcı A, Aksoy F. The frontal plane QRS-T angle may affect our perspective on prehypertension: A prospective study. Clin Exp Hypertens. 2021;43(5):402-7. [PubMed]

26. Tanriverdi Z, Besli F, Gungoren F, Altiparmak I, Yesilay A, Erkus M, et al. Esansiyel hipertansiyonlu hastalarda sol ventrikül hipertrofisinin bir göstergesi olarak frontal QRS-T açısı [Frontal QRS-T angle as a marker of left ventricular hypertrophy in patients with essential hypertension]. DEÜ Tıp Fakultesi Dergisi. 2018;32(2):77-87. [Link]

27. Gür M, Yilmaz R, Demirbağ R, Yildiz A, Akyol S, Polat M, et al. Relationship between the elastic properties of aorta and QT dispersion in newly diagnosed arterial adult hypertensives. Anadolu Kardiyol Derg. 2007;7(3):275-80. [PubMed]

28. Elias P, Poterucha TJ, Rajaram V, Moller LM, Rodriguez V, Bhave S, et al. Deep learning electrocardiographic analysis for detection of left-sided valvular heart disease. J Am Coll Cardiol. 2022;80(6):613-26. [Crossref] [PubMed]

29. Cohen-Shelly M, Attia ZI, Friedman PA, Ito S, Essayagh BA, Ko WY, et al. Electrocardiogram screening for aortic valve stenosis using artificial intelligence. Eur Heart J. 2021;42(30):2885-96. [Crossref] [PubMed]