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RESEARCH

Evaluation of frontal QRS-T angle and cardiac electrophysiological balance index in patients with coronary artery ectasia

Koroner arter ektazisi olan hastalarda frontal QRS-T açısı ve kardiyak elektrofizyolojik denge indeksinin değerlendirilmesi

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Abstract

Purpose: The aim of this study was to evaluate arrhythmogenic risk in patients with Coronary artery ectasia (CAE) using the frontal QRS-T (fQRS-T) angle and the index of cardiac electrophysiological balance (iCEB), together with conventional ventricular repolarization parameters.

Materials and Methods: This retrospective study included 103 patients who underwent coronary angiography. Individuals with coronary segment dilatation ≥1.5 times the adjacent normal vessel without significant stenosis were classified as the CAE group, while those with angiographically normal coronary arteries comprised the control group. Electrocardiographic (ECG) parameters were analyzed: QT and corrected QT (QTc) intervals, T wave peak-to-end (Tp-e) interval, Tp-e/QT and Tp-e/QTc ratios, fQRS-T angle, and iCEB calculated as QT/QRS; its corrected form (iCEBc) was computed as QTc/QRS. Subgroup analysis compared single-vessel and multivessel ectasia.

Results: Patients with CAE demonstrated significantly wider fQRS-T angles (68.0° vs. 22.0°) and higher iCEB (4.39 vs. 4.04) and iCEBc values (4.82 vs. 4.43) compared with controls. QT, QTc, Tp-e, Tp-e/QT, and Tp-e/QTc values were also significantly increased. Multivessel ectasia was associated with a significantly greater fQRS-T angle (84.0° vs. 48.5°) and QTc prolongation (446.6 vs. 414.6 ms).

Conclusion: CAE is associated with substantial electrophysiological alterations indicative of heightened arrhythmogenic susceptibility. Increasing anatomical extent further amplifies this burden, particularly as reflected by a wider fQRS-T angle and the global repolarization burden demonstrated by prolonged QTc.

Keywords: Coronary ectasia; electrocardiography; ischemia; repolarization.

Öz

Amaç: Bu çalışma, koroner arter ektazisili (CAE)hastalarda aritmojenik riski; frontal QRS-T (fQRS-T) açısı ve kardiyak elektrofizyolojik denge indeksi (iCEB) ile birlikte konvansiyonel ventriküler repolarizasyon parametrelerini değerlendirerek belirlemeyi amaçlamıştır.

Gereç ve Yöntem: Bu retrospektif çalışma, koroner anjiyografi uygulanan 103 hastayı içermektedir. Komşu normal segmentin çapının ≥1.5 katı düzeyinde, anlamlı stenoz olmaksızın koroner arter dilatasyonu bulunan bireyler CAE grubu olarak sınıflandırılırken; anjiyografik olarak normal koroner arterlere sahip hastalar kontrol grubunu oluşturmuştur. Elektrokardiyografik (EKG) parametreler değerlendirilmiştir: QT ve düzeltilmiş QT (QTc) aralıkları, T dalgası pikten sonuna kadar olan süre (Tp-e), Tp-e/QT ve Tp-e/QTc oranları, fQRS-T açısı ve QT/QRS olarak hesaplanan iCEB; düzeltilmiş formu (iCEBc) QTc/QRS olarak hesaplanmıştır. Alt grup analizinde tek damar ve çok damar ektazisi karşılaştırılmıştır.

Bulgular: CAE'li hastalarda fQRS-T açısı kontrol grubuna kıyasla anlamlı derecede daha geniş bulunmuştur (68.0° karşı 22.0°) ve iCEB (4.39 karşı 4.04) ile iCEBc (4.82 karşı 4.43) değerleri de daha yüksektir. Ayrıca QT, QTc, Tp-e, Tp-e/QT ve Tp-e/QTc değerleri anlamlı şekilde artmıştır. Çok damar ektazisi olan hastalarda fQRS-T açısı anlamlı olarak daha geniştir (84.0° karşı 48.5°) ve QTc daha uzundur (446.6 karşı 414.6 ms).

Sonuç: CAE, artmış aritmojenik yatkınlığı yansıtan belirgin elektrofizyolojik değişikliklerle ilişkilidir. Ektazinin anatomik yaygınlığındaki artış bu yükü daha da artırmakta olup, özellikle fQRS-T açısının belirgin genişlemesi ve QTc uzamasında görülmektedir.

Anahtar kelimeler: Koroner ektazi; elektrokardiyografi; iskemi; repolarizasyon.

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INTRODUCTION

Coronary artery ectasia (CAE) is defined as an abnormal enlargement of an epicardial coronary artery segment, in which the lumen diameter reaches at least 1.5 times that of nearby reference vessel that was morphologically normal[1]. When the arterial diameter exceeds twice its normal size, the condition is referred to as an aneurysm[2]. If CAE is not accompanied by any significant obstructive lesion within the vessel, it is termed isolated CAE[3]. The incidence of CAE ranges from 1.2% to 4.9% in coronary angiography series, whereas isolated CAE is comparatively rarer, with prevalence reported between 0.1% and nearly 0.8% in cases evaluated via coronary angiography[1,4].

CAE may result from both congenital and acquired causes. Among the acquired forms, atherosclerosis is the most common etiology, while other causes include various types of vasculitis and connective tissue disorders[5]. Due to stagnant and turbulent blood flow within ectatic coronary segments, coronary flow velocity decreases, leading to the coronary slow-flow phenomenon, which may contribute to exercise-induced myocardial ischemia and impaired microvascular perfusion[6].

CAE has been linked to malignant ventricular arrhythmias, likely due to ischemia-induced ventricular repolarization abnormalities[7]. In attempts to delineate arrhythmogenic risk in patients with CAE, multiple electrocardiographic (ECG) indices reflecting ventricular repolarization have been explored. Prior studies have shown that metrics such as corrected QT interval (QTc), T-wave peak-to-end interval (Tp-e), Tp-e/QT, and Tp-e/QTc are markedly elevated in patients with CAE relative to healthy subjects[3,8].

The frontal QRS-T (fQRS-T) angle and the index of cardiac electrophysiological balance (iCEB), as novel indicators of cardiac electrical stability, have recently emerged as reliable and easily applicable ECG markers for the assessment of arrhythmogenic risk[9,10]. However, to date, no study has concurrently evaluated the relationship between these two parameters and CAE.

In this study, we investigated how CAE influences myocardial electrical behavior by analyzing the fQRS-T angle and the iCEB as markers of arrhythmic vulnerability. To our knowledge, no previous study has concurrently examined the fQRS-T angle and the iCEB in patients with CAE. We hypothesized that patients with CAE would exhibit increased fQRS-T angles and higher iCEB values compared with individuals with normal coronary anatomy, and that these abnormalities would be more pronounced in multivessel ectasia.

MATERIALS AND METHODS

Sample

This study employed a retrospective, observational case–control design using archived data from the Cardiology Clinic of Adana City Training and Research Hospital. Adult individuals who underwent coronary angiography from November 1, 2024 to October 31, 2025, were evaluated. Grouping was based on coronary angiography findings, yielding two study cohorts. Patients were considered to have CAE when at least one epicardial coronary segment exhibited disproportionate enlargement, measuring ≥1.5 times the caliber of a contiguous reference segment, without angiographically significant stenosis elsewhere in the coronary circulation. Individuals with angiographically intact coronary anatomy were assigned to the control group.

The exclusion criteria included the presence of coronary artery stenosis, atrial fibrillation, atrial flutter, bundle branch block, or pre-excitation on ECG, the presence of a pacemaker, a reduced left ventricular ejection fraction (<50%) and treatment with antiarrhythmic drugs known to influence QT interval duration.

Procedure

This retrospective study was reviewed and approved by the Ethics Committee for Scientific Research of Adana City Training and Research Hospital (Meeting no: 19, Date: 20.11.2025, Approval no: 863). Owing to its retrospective design, informed consent was waived.

Clinical and demographic characteristics such as age, sex, body mass index, hypertension, diabetes mellitus, hyperlipidemia, smoking status, and family history of coronary artery disease were obtained from patient records. Laboratory data including hemoglobin, leukocyte count, platelet count, glucose, creatinine, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides,

potassium, and calcium levels, as well as echocardiographic left ventricular ejection fraction, were collected from the Hospital Information Management System (HIMS) and patient files.

Angiographic analysis

Coronary angiography images were reviewed using the digital angiography archive system. Angiographic parameters included the presence of ectasia and the number of ectatic vessels. Angiographic views were evaluated directly by a cardiology specialist. Ectasia involving any one of the left anterior descending artery, left circumflex artery, or right coronary artery was classified as single-vessel ectasia. Ectasia involving any two of these arteries was defined as two-vessel ectasia, and the presence of ectasia in all three arteries was defined as three-vessel ectasia. Patients with ectasia in two or more of these arteries were categorized as having multivessel ectasia.

Electrocardiographic analysis

Twelve-lead ECGs were obtained from all participants in the supine resting position at a paper speed of 25 mm/s and a standard calibration of 10 mm/mV. The recordings were subsequently digitized and transferred to an electronic platform to reduce measurement variability. Each ECG was reviewed to exclude atrial fibrillation, atrial flutter, bundle branch block, or pre-excitation. All measurements were manually performed by an experienced cardiologist who was blinded to the clinical data. The QT, Tp-e, and QRS intervals were evaluated in leads V5 and V6, and the longest measurements were selected for statistical analysis. The QT interval was measured from the beginning of the QRS complex to the end of the T wave. The QRS duration was measured from the initial deflection to the terminal point of the QRS complex, whereas the Tp-e interval was defined as the time from the peak of the T wave to its return to baseline. The QTc was calculated using Bazett's formula, defined as the QT interval divided by the square root of the RR interval. The Tp-e/QT and Tp-e/QTc ratios were calculated for each recording. The fQRS-T angle was obtained by calculating the absolute difference between the frontal plane QRS and T-wave axes. When this difference exceeded 180°, the angle was reassigned as 360° minus the absolute value of that difference[9]. The axis values used in this calculation were derived from the automated ECG report. Determination of iCEB was by dividing the QT interval by the QRS duration (QT/QRS), and the corrected iCEB (iCEBc) was calculated using QTc divided by QRS duration (QTc/QRS)[10]. For each patient, heart rate, QT interval, QTc interval, QRS duration, and Tp-e interval were recorded for analysis.

Statistical analysis

All statistical analyses were conducted using IBM SPSS Statistics software, version 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was examined for normality within each group using the Shapiro–Wilk test. Continuous variables demonstrating a normal distribution were expressed as mean ± standard deviation, whereas those that were not normally distributed were presented as median (interquartile range, IQR). Categorical variables were summarized as frequency and percentage (%). Comparisons between two independent groups were performed using the independent samples t-test for normally distributed variables and the Mann–Whitney U test for variables not following a normal distribution. The chi-square test was applied for comparisons of categorical data between groups. A p-value <0.05 was considered statistically significant. In the present study, patients with CAE were compared with the control group in terms of demographic characteristics, clinical findings, laboratory parameters, and ECG indices. According to the extent of ectasia observed on coronary angiography, patients were initially categorized as single-vessel (n = 24), two-vessel (n = 15), and three-vessel (n = 8) ectasia. However, since the number of patients in the three-vessel group was relatively small, the analysis was performed by combining the two-vessel and three-vessel subgroups into a single multivessel ectasia group. Consequently, single-vessel and multivessel CAE groups were compared with respect to ECG parameters.

RESULTS

A total of 103 participants were included, consisting of 56 individuals in the control group and 47 patients with CAE. There were no significant differences between the two groups with respect to age (53.6 ± 9.6 vs. 55.4 ± 9.3 years, p = 0.338) and sex distribution (48.2% vs. 51.1% male, p = 0.844). No statistically significant differences were detected regarding body mass index, smoking status, hypertension, diabetes mellitus, hyperlipidemia, or family history of coronary artery disease (all $p > 0.05$). The median left ventricular ejection fraction showed no statistically significant variation between patients

with ectasia and controls, measured at 55.0% and 58.0%, respectively (p = 0.159). Overall, demographic risk profiles were similar between groups (Table 1).

There were no significant differences in routine hematologic or metabolic parameters, with the exception of hemoglobin levels, which were lower in the ectasia group (14.2 vs. 14.9 g/dL, p = 0.010). Fasting glucose levels were slightly higher among ectasia patients (114.0 vs. 98.0 mg/dL, p = 0.050). Lipid parameters, renal function, and electrolyte levels were comparable between the two groups (all p > 0.05) (Table 2). Patients with CAE demonstrated marked alterations in ventricular repolarization indices. The fQRS-T angle was significantly broader in the ectasia group (median 68° vs. 22°, p < 0.001), and both iCEB and iCEBc showed increased values (4.39 ± 0.61 vs. 4.04 ± 0.54, p = 0.003 and 4.82 ± 0.68 vs. 4.43 ± 0.56, p = 0.001, respectively), as illustrated in Figure 1. In addition, the QTc interval was prolonged (422.0 vs. 404.0 ms, p = 0.007), and Tp-e duration was significantly longer in ectasia patients (86.9 vs. 73.1 ms, p < 0.001). Although QT interval was also increased (391.8 ± 42.1 vs. 372.9 ± 28.9 ms, p = 0.011), QRS duration remained similar between groups (p = 0.305). Tp-e/QT (0.22 ± 0.03 vs. 0.20 ± 0.05, p = 0.002) and Tp-e/QTc ratios (0.20 vs. 0.19, p = 0.013) were higher in the ectasia group (Table 3).

Table 1. Demographic and clinical characteristics of the study sample

Variable	Control (n=56)	Ectasia (n=47)	P value
Age (years)	53.6 ± 9.6	55.4 ± 9.3	0.338
Male sex, n (%)	27 (48.2)	24 (51.1)	0.844
BMI (kg/m²)	28.1 ± 4.2	27.9 ± 3.4	0.749
Smoking, n (%)	19 (33.9)	18 (38.3)	0.684
Hypertension, n (%)	18 (32.1)	14 (29.8)	0.834
Diabetes mellitus, n (%)	16 (28.6)	15 (31.9)	0.830
Hyperlipidemia, n (%)	24 (42.9)	18 (38.3)	0.690
Family history of CAD, n (%)	20 (35.7)	15 (31.9)	0.835
EF (%)	58.0 (55.0 - 60.0)	55.0 (50.0 - 60.0)	0.159

Independent samples t-test, Mann–Whitney U test, and chi-square test were used; Values are presented as mean ± standard deviation, median (interquartile range) or number (percentage); A p value < 0.05 was considered statistically significant; BMI: body mass index; CAD: coronary artery disease; EF: ejection fraction.

Table 2. Laboratory parameters of the study sample

Variable	Control (n=56)	Ectasia (n=47)	P value
Hemoglobin (g/dL)	14.9 (13.7 - 15.9)	14.2 (12.8 - 14.8)	0.010
Leukocyte count (/μL)	8.2 (7.2 - 9.9)	8.3 (7.0 - 11.2)	0.822
Platelet count (/μL)	262.5 (214.5 - 301.0)	233.0 (200.0 - 293.0)	0.191
Glucose (mg/dL)	98.0 (86.5 - 135.5)	114.0 (95.0 - 146.0)	0.050
Creatinine (mg/dL)	0.85 (0.77 - 0.97)	0.80 (0.72 - 0.94)	0.534
Total cholesterol (mg/dL)	176.5 ± 41.3	188.5 ± 47.7	0.174
HDL cholesterol (mg/dL)	43.0 (38.0 - 48.5)	40.0 (35.0 – 45.0)	0.069
LDL cholesterol (mg/dL)	120.4 ± 29.7	128.3 ± 36.7	0.232
Triglycerides (mg/dL)	134.0 (105.0 - 171.5)	143.0 (91.0 - 205.0)	0.521
Calcium (mg/dL)	9.2 (9.0 - 9.5)	9.1 (8.8 - 9.4)	0.270
Potassium (mEq/L)	4.3 ± 0.4	4.2 ± 0.4	0.217

Independent samples t-test and Mann–Whitney U test were used; Values are presented as mean ± standard deviation or median (interquartile range); A p value < 0.05 was considered statistically significant; HDL: high-density lipoprotein; LDL: low-density lipoprotein.

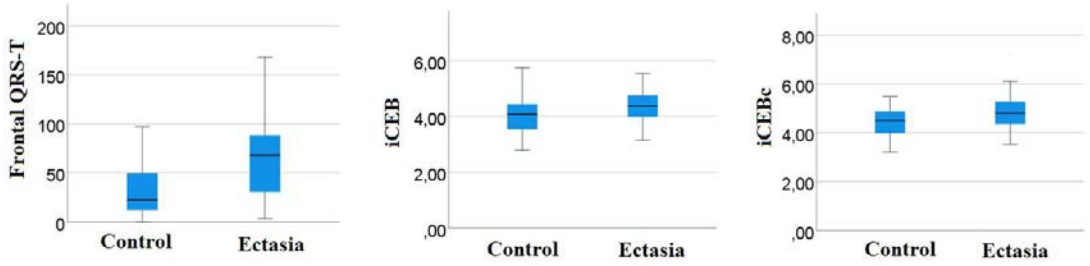


Figure 1. Box-and-whisker plots comparing fQRS-T angle, iCEB, and iCEBc between patients with coronary artery ectasia and controls.

Table 3. Comparison of ECG repolarization parameters between CAE patients and controls

Variable	Control (n=56)	Ectasia (n=47)	P value
Heart rate (beats/min)	70.5 (65.0 - 81.0)	72.0 (64.0 - 82.0)	0.868
fQRS-T angle (°)	22.0 (12.0 - 49.5)	68.0 (30.0 - 90.0)	<0.001
QTc interval (ms)	404.0 (395.0 - 418.0)	422.0 (401.0 - 457.0)	0.007
QT interval (ms)	372.9 ± 28.9	391.8 ± 42.1	0.011
QRS duration (ms)	90.0 (84.0 - 104.0)	90.0 (84.0 - 96.0)	0.305
Tp-e interval (ms)	73.1 ± 20.1	86.9 ± 16.3	<0.001
Tp-e/QT ratio	0.20 ± 0.05	0.22 ± 0.03	0.002
Tp-e/QTc ratio	0.19 (0.14 - 0.21)	0.20 (0.18 - 0.22)	0.013
iCEB	4.04 ± 0.54	4.39 ± 0.61	0.003
iCEBc	4.43 ± 0.56	4.82 ± 0.68	0.001

Independent samples t-test and Mann–Whitney U test were used; Values are presented as mean ± standard deviation or median (interquartile range); A p value < 0.05 was considered statistically significant; CAE: coronary artery ectasia; ECG: electrocardiographic; fQRS-T: frontal QRS-T; QTc: corrected QT; Tp-e: T-peak to T-end; iCEB: index of cardio-electrophysiological balance; iCEBc: corrected index of cardio-electrophysiological balance; ms: milliseconds.

Table 4. Comparison of ECG repolarization parameters between single-vessel and multivessel coronary ectasia

Variable	Single-vessel (n = 24)	Multivessel (n = 23)	p value
Heart rate (beats/min)	69.0 (62.0 -75.5)	75.0 (68.0 -84.0)	0.030
fQRS-T angle (°)	48.5 (20.0 -69.0)	84.0 (65.0 -117.0)	0.005
QTc interval (ms)	414.6 ± 28.3	446.6 ± 43.1	0.005
QT interval (ms)	387.0 ± 32.0	396.9 ± 50.9	0.484
QRS duration (ms)	90.0 (84.0 –96.0)	90.0 (82.0 -98.0)	0.856
Tp-e interval (ms)	84.8 ± 17.9	89.0 ± 14.6	0.388
Tp-e/QT ratio	0.22 ± 0.04	0.23 ± 0.03	0.503
Tp-e/QTc ratio	0.20 ± 0.04	0.20 ± 0.03	0.554
iCEB	4.35 ± 0.53	4.43 ± 0.70	0.670
iCEBc	4.66 ± 0.55	4.99 ± 0.76	0.097

Independent samples t-test and Mann–Whitney U test were used; Values are presented as mean ± standard deviation or median (interquartile range); A p value < 0.05 was considered statistically significant; ECG: electrocardiographic; fQRS-T: frontal QRS-T; QTc: corrected QT; Tp-e: T-peak to T-end; iCEB: index of cardio-electrophysiological balance; iCEBc: corrected index of cardio-electrophysiological balance; ms: milliseconds.

A subgroup analysis was performed to compare patients with single-vessel and multivessel coronary ectasia. Individuals with multivessel involvement exhibited a significantly higher heart rate (75.0 vs. 69.0 bpm, p = 0.030) and a markedly greater fQRS-T angle (84.0° vs. 48.5°, p = 0.005). QTc duration was also prolonged in the multivessel group (446.6 ± 43.1 vs. 414.6 ± 28.3 ms, p = 0.005), whereas the QT interval itself did not differ significantly (p = 0.484) (Table 4). Other repolarization markers, including Tp-e interval, Tp-e/QT, and Tp-e/QTc ratios, showed numerically higher values in patients with

multivessel disease, although these differences did not reach statistical significance. Similarly, iCEB and iCEBc values were slightly elevated in multivessel ectasia, but without statistical significance ($p = 0.670$ and $p = 0.097$, respectively) (Table 4).

DISCUSSION

To our knowledge, no prior study has concurrently examined the fQRS-T angle and iCEB in patients with CAE. Although these markers have been investigated separately across various cardiovascular conditions, their combined assessment in the context of CAE has not been documented. In the current study, we examined these two indices together with conventional repolarization parameters and compared them between patients with angiographically confirmed CAE and individuals with normal coronary anatomy. Among the measured parameters, the fQRS-T angle showed the largest numerical difference between CAE patients and controls, whereas both the QRS-T angle and the QTc interval demonstrated statistically significant differences between single-vessel and multivessel ectasia groups.

Our findings partly align with previous work. Alsancak et al. reported that iCEB exhibited only a numerical increase in CAE, without significant differences between single-vessel and multivessel disease, whereas Karahan et al. demonstrated that the fQRS-T angle was significantly wider in CAE but did not stratify patients by disease extent[11,12]. By assessing both markers in the same cohort, our study extends these observations. Similar to earlier reports, we found significantly higher fQRS-T angles in CAE, and, unlike Alsancak et al., we also observed significantly elevated iCEB and iCEBc values when comparing CAE patients with controls. Although both studies demonstrated numerically higher iCEB/iCEBc values in multivessel ectasia, the larger variability in the previous cohort and the overlapping distributions across subgroups likely prevented statistical significance. Likewise, in our study, despite numerically higher iCEB/iCEBc values in multivessel disease, subgroup overlap also limited statistical separation.

Our results regarding repolarization parameters are in agreement with prior investigations. In line with earlier investigations by Karaagac et al. and Alsancak et al., we observed prolonged Tp-e intervals and higher Tp-e/QT or Tp-e/QTc ratios in CAE, consistent with enhanced transmural repolarization heterogeneity[8,11]. As previously described by Salari et al. and Karahan et al., we also identified longer QT and QTc intervals in CAE patients[3,12]. However, unlike Alsancak et al., who reported markedly higher Tp-e and Tp-e/QT values in multivessel ectasia, we did not observe significant subgroup differences for these indices. Instead, QTc was the only conventional repolarization parameter that distinguished single-vessel from multivessel disease in our cohort.

Each ECG parameter examined reflects a distinct electrophysiological domain. QT and QTc intervals quantify global ventricular repolarization and typically lengthen under generalized electrical instability[13]. Tp-e and its derived ratios estimate transmural repolarization heterogeneity, governed by differences in repolarization between epicardial, endocardial, and mid-myocardial cells[14]. In contrast, iCEB and iCEBc quantify the balance between ventricular activation and recovery, offering insight into susceptibility to reentry-mediated arrhythmias[10]. The fQRS-T angle captures the divergence between depolarization and repolarization vectors and thus acts as an integrated marker of global electrical discordance[15]. These physiological distinctions explain why their discriminatory performance across anatomical subgroups varies. In our cohort, QTc, which reflects global repolarization burden, was the only conventional repolarization parameter that differentiated single-vessel from multivessel ectasia. In contrast, Tp-e indices failed to show similar differences, indicating that transmural dispersion does not necessarily increase in proportion to angiographic extent. The fQRS-T angle, integrating both depolarization and repolarization vectors, demonstrated clear subgroup discrimination, highlighting its sensitivity to widespread electrical heterogeneity. Although iCEB values tended to be higher in the multivessel group, the wide dispersion of measurements within subgroups reduced the ability to detect a statistically significant difference. Collectively, these findings suggest that CAE affects multiple aspects of ventricular electrophysiology; however, the extent to which each ECG parameter reflects disease severity varies because each marker captures a distinct electrical process, which likely explains their differing discriminatory performance across anatomical subgroups.

Hemodynamic disturbances inherent to CAE may provide a mechanistic basis for the observed electrophysiological abnormalities. Turbulent and

decelerated coronary flow disrupts endothelial function, alters shear stress, and promotes local thrombotic microembolization, leading to impaired microvascular perfusion and uneven repolarization gradients across the ventricular myocardium[16,17]. As this ischemic burden increases, particularly in multivessel ectasia, global repolarization prolongation becomes more pronounced. This pathophysiological cascade is consistent with our findings: patients with CAE demonstrated marked augmentation of ventricular repolarization indices relative to controls, and individuals with multivessel ectasia exhibited a substantially wider fQRS-T angle and a longer QTc interval than those with single-vessel involvement.

Several important limitations should be considered. The present study was conducted in a single tertiary center with a relatively modest sample size, which may limit external validity and reduce the applicability of the findings to broader populations. Because of its retrospective design, residual confounding cannot be completely excluded despite careful patient selection. ECG-derived electrophysiological parameters were obtained from single recordings under resting conditions; thus, transient or exercise-induced changes in repolarization and conduction could not be assessed. Additionally, the indices investigated represent indirect markers of arrhythmogenic risk. The absence of rhythm-related clinical outcomes, device-recorded arrhythmia burden, or long-term follow-up data limits the ability to infer prognostic relevance. Coronary angiography served as the diagnostic reference for CAE, but this modality cannot characterize microvascular dysfunction, coronary flow reserve, or endothelial performance, all of which may critically mediate repolarization heterogeneity. Future prospective studies incorporating physiological imaging, serial ECG assessments, and clinical endpoints are warranted to validate these findings.

In conclusion, the presence of CAE was associated with heightened arrhythmogenic propensity, as demonstrated by consistent alterations in ECG-derived indices of electrical instability. These observations align with the concept that ectasia promotes ventricular repolarization heterogeneity and vectorial discordance, creating a more vulnerable electrophysiological substrate. Importantly, the severity of this burden appeared to parallel anatomical extent: patients with multivessel disease exhibited a markedly wider fQRS-T angle and prolonged QTc interval compared with those with single-vessel ectasia. These readily obtainable parameters may therefore hold value for non-invasive risk stratification and early identification of patients at increased electrical risk. Nonetheless, prospective longitudinal studies incorporating clinical arrhythmic endpoints are needed to determine whether these indices translate into meaningful prognostic utility.

Author Contributions: Concept/Design : PÖY; Data acquisition: PÖY; Data analysis and interpretation: PÖY; Drafting manuscript: PÖY; Critical revision of manuscript: PÖY; Final approval and accountability: PÖY; Technical or material support: PÖY; Supervision: PÖY; Securing funding (if available): n/a.
Ethical Approval: The present study protocol was reviewed and approved by the institutional review board of Health Sciences University, Adana City Training and Research Hospital (Reg. No. 19/863, Date: 20.11.2025).
Peer-review: Externally peer-reviewed.
Conflict of Interest: The authors have nothing to disclose.
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