Original Article

HEALTH SCIENCES
MEDICINE

DOI: 10.32322/jhsm.1627315
J Health Sci Med. 2025;8(2):356-360

The relationship between QT dispersion, fragmented QRS, and collateral circulation in patients with chronic total coronary artery occlusion

Serdar Gökhan Nurkoç, Orhan Karayiğit, İmam Günay
Department of Cardiology, Yozgat City Hospital, Yozgat, Turkiye

Cite this article as: Nurkoç SG, Karayiğit O, Günay İ. The relationship between QT dispersion, fragmented QRS, and collateral circulation in patients with chronic total coronary artery occlusion. *J Health Sci Med.* 2025;8(2):356-360.

Received: 26.01.2025 ◆ **Accepted**: 16.03.2025 ◆ **Published**: 21.03.2025

ABSTRACT

Aims: Corrected QT dispersion (QTcD), prolonged QT dispersion (QTD), and fragmented QRS (fQRS) are known as indicators of high risk for cardiac arrhythmias. Within this context, our research was conducted to assess the role of coronary collateral circulation (CCC) on QTD and fQRS in participants with chronic total coronary artery occlusion.

Methods: This study examined 131 participants with CCC in total. The participants were divided into two groups based on the Rentrop classification: group 1 (Rentrop 0 and 1) and group 2 (rentrop 2 and 3). Demographic data, laboratory results, and electrocardiogram findings were analyzed retrospectively.

Results: Significantly, variability was observed between the poor and good collateral groups in terms of QTcD (91.3±21.6 vs 57.2±26.2, p<0.001), QTD (87.6±21.3 vs. 55.2±26.2, p<0.001), and the presence of fQRS (139.5±8.0 vs. 128.1±13.1, p<0.001). Correlation analysis indicated a significant connection between the Rentrop classification and diabetes mellitus, creatinine levels, QTD, QTcD, and the existence of fQRS.

Conclusion: Poorly developed CCC was associated with increased QTcD, QTD, and the presence of fQRS. These parameters QTD, QTcD, and fQRS may serve as important, easily accessible, and effective tools in predicting the quality of CCC in individuals with coronary artery disorder. Nevertheless, these outcomes should be investigated through further research.

Keywords: Fragmented QRS, coronary collateral circulation, QT dispersion

INTRODUCTION

The prevalence of coronary artery disease (CAD) is widely considered a leading cause of death globally, with death mainly caused by arrhythmias, especially sudden cardiac arrest.[1] The presence of scar tissue in the ventricle, along with viable myocardial tissue that survives, is believed to significantly contribute to the heterogeneity of ventricular repolarization and the subsequent arrhythmias observed in CAD.[2] It is proposed that the variation in impulse formation and conduction between normal, ischemic, and necrotic tissues is a basic reason in the emergence of ventricular arrhythmias in CAD.[3] The severity of CAD and left ventricular dysfunction are associated with the prevalence of arrhythmias.[3]

Chronic total coronary occlusion (CTO) refers to the complete blockage of a coronary artery, which occurs as a result of thrombosis following a myocardial infarction (MI) and is marked by TIMI 0 flow that persists for at least three months.[4] CTO is observed in approximately 16% of individuals undergoing coronary imaging.[5] Coronary collateral circulation (CCC) is made up of potential vessels that are usually undetectable with conventional coronary angiography but become visible when coronary arteries are occluded, as collateral vessels expand to supply blood due to pressure differences.[6]

Various ECG markers, including QT dispersion (QTD) and fragmented QRS (fQRS), are used to clarify participants at increased risk for ventricular arrhythmias, and their relationship with collateral circulation has been studied in CTO participants.[7-9] QTD, which is described as the distinction between the minimum and maximum QT intervals, is a non-invasive method to assess irregularities in myocardial repolarization. Differences in the QT interval between leads indicate regional disparities in cardiac repolarization, and increased dispersion of ventricular recovery time is considered a substrate for serious arrhythmias and sudden cardiac death.[10] fQRS complexes are characterized by abnormal patterns or Q waves in consecutive leads that correspond to a primary coronary artery region, without a classic bundle branch block.[11] They are classified into two types based on their duration: narrow fQRS complexes (QRS <120 ms) and wide fQRS complexes (QRS ≥120 ms), which can appear in

Corresponding Author: Serdar Gökhan Nurkoç, serdar_nurkoc@hotmail.com

various QRS morphologies. Occasionally, fQRS is the single ECG indicator of myocardial destruction in individuals with non-Q myocardial infarctions or those with a resolved Q wave.[11]

Our research purposed to find out whether the presence of collateral CCC in individuals with coronary CTO influences QTD, corrected QT dispersion (QTcD), and fQRS, which are markers used to predict ventricular arrhythmias.

METHODS

Ethics

The study protocol was approved by Yozgat Bozok University Rectorate Non-interventional Clinical Researches Ethics Committee (Date: 04.12.2024, Decision No: 2024-GOKAEK-2414_2024.12.04_204). All procedures were conducted in line with ethical guidelines and the principles outlined in the Declaration of Helsinki. Participants' formal informed permission was not acquired because the work was a retrospective plan.

Patient Population and Study Protocol

Between January 2018 and June 2024, we examined 131 patients with stable coronary artery disorder subsequent to performing coronary angiography at our hospital and documenting total occlusion in one of their major coronary arteries. Demographic characteristics, laboratory results, and ECG data were collected retrospectively. CTO was characterized as a fully blocked lesion with TIMI 0 flow persisting for over three months, as defined by the Euro CTO Club.[12] Collateral circulation was assessed using the Rentrop classification: grade 0 (no collateral filling), grade 1 (collateral vessels supply side branches of the CTO artery, excluding epicardial segments), grade 2 (collateral vessels partially fill epicardial arteries), and grade 3 (collateral vessels fill epicardial vessel).[13] Participants were separated into two groups based on the Rentrop classification: group 1 (Rentrop 0 and 1) and group 2 (Rentrop 2 and 3). Exclusion criteria included recent myocardial infarction (within three months), arrhythmias other than atrial fibrillation, sinus rhythm, any level of atrioventricular or bundle branch block, diseases related to valve disorders, patients who have undergone bypass surgery, abnormal serum electrolyte concentrations, use of antiarrhythmic medications, or having a permanent pacemaker.

ECG Parameters

A standard 12-lead resting ECG was recorded via the Nihon Kohden recorder (Tokyo, Japan) with a 10 mm/mV amplitude and a 25 mm/s paper speed. Two expert cardiologists reviewed all ECGs without knowing the patient's clinical condition or angiographic results. The QT interval was calculated from the onset of the first negative deflection of the QRS complex to the end of the T wave, considering the TP isoelectric line. The heart ratio QTc interval was calculated via Bazett's formula. QTD was defined as the distinction between the maximum and minimum QT calculations from the precordial leads.[10] fQRS was detected as the emergence of an extra R wave (R'), interruption of the R or S wave, or the occurrence of several R waves (R') in sequential leads correlating with a major coronary artery territory.[11]

Statistical Analysis

As SPSS software, version 26.0 (SPSS Inc., Chicago, IL, USA) was carried out to conduct statistical analyses, the Kolmogorov-Smirnov test was applied to examine variable distribution patterns. While continuous variants were shown as mean±standard deviations or medians with interquartile range (IQR), considering the level of distribution, categorical variables were presented as ratios and percentages. Nonparametric continuous variables were determined via the Mann-Whitney U test, as categorical variants were analyzed via Fisher's exact test or Pearson's chi-square test. The correlation between clinical parameters and Rentrop classification was determined using Spearman's correlation examination. The Hosmer-Lemeshow test was used to evaluate model fit. We also used receiver operating characteristic (ROC) curve analysis to determine optimal cut-off values for QTcD and fQRS in predicting collateral circulation and to assess specificity and sensitivity. A p-value <0.05 was regarded as statistically significant.

RESULTS

131 participants overall were encompassed within the research, with general patient characteristics shown in **Table 1**. Sixty-four patients were allocated to collateral group 1, and 67 patients to collateral group 2. Laboratory analysis revealed that the population of individuals with diabetes mellitus (DM) was remarkably higher in group 1 compared to group 2 [39 (60.9%) vs. 29 (43.3%), p=0.043]. ECG parameters related to the good and poor collateral groups were assessed as presented in **Table 2**. Remarkable distinctions were detected between the groups with respect to QTcD (91.3±21.6 vs. 57.2±26.2, p<0.001), QTD (87.6±21.3 vs. 55.2±26.2, p<0.001), and the existence of fQRS (139.5±8.0 vs. 128.1±13.1, p<0.001).

Correlation analysis revealed substantial relationships between the Rentrop classification and QTcD (r=-0.659, p<0.001), QTD (r=-0.648, p<0.001), fQRS (r=-0.557, p<0.001), DM (r=-0.176, p=0.044), and creatinine (r=0.185, p=0.034), (**Table 3**).

ROC curve tests indicated that a QTcD cut-off rate above 75.5 predicted the Rentrop classification with 84.4% sensitivity and 85.1% specificity (AUC:0.885; 95% CI:0.822-0.949; p<0.001). For fQRS, a cut-off rate above 133.5 estimated the Rentrop classification with 78.1% sensitivity and 79.1% specificity AUC:0.840; 95% CI:0.769-0.912; p<0.001, (**Figure 1, 2**).

DISCUSSION

Our research purposed to detect the connection between the condition of CCC, QTcD, QTD, and fQRS in individuals with CTO. The improvement of CCC can affect the prognosis of CTO individuals. CCC is a response to occlusive damage in the coronary arteries. CTO individuals often have collateral vessels in the distal arteries, and these collaterals may help alleviate ischemic and anginal symptoms, as well as preserve ventricular function.[14] Former works have proved that good collateral circulation is correlated with fewer infarctions,

Table 1. Baseline characteristics and laboratory parameters of the study groups

Variables	Good collateral (n: 67)	Poor collateral (n: 64)	p-value
Baseline characteristics			
Age, years	67.3±11.9	68.0±11.7	0.885
Male gender, n (%)	47 (70.1)	46 (71.9)	0.828
Diabetes mellitus, n (%)	29 (43.3)	39 (60.9)	0.043
Hypertension, n (%)	44 (65.7)	46 (71.9)	0.444
Dyslipidemia, n (%)	20 (29.9)	20 (31.2)	0.862
Smoking status, n (%)	32 (47.8)	31 (48.4)	0.938
History of CAD, n (%)	26 (38.8)	21 (32.8)	0.475
Laboratory parameters			
Glucose, mg/dl	115 (99-171)	134 (106-167)	0.098
Creatinine, mg/dl	1.26±0.96	1.09±0.83	0.064
Sodium, mmol/L	138 (137-140)	138 (135-140)	0.387
Potassium, mmol/L	4.2 (4.0-4.4)	4.1 (3.9-4.5)	0.991
Uric acid, mh/dl	6.3±1.7	6.9±5.6	0.809
Total cholesterol, mg/dl	180±37	181±48	0.885
HDL-C, mg/dl	41 (35-46)	43 (36-47)	0.197
LDL-C, mg/dl	110±35	110±42	0.718
Triglycerides, mg/dl	129 (86-176)	117 (84-177)	0.385
WBC count, x10^3/µl	9.4 (7.1-12.5)	9.5 (6.9-11.8)	0.985
Neutrophil count, x10^3/µl	6.3 (4.2-8.0)	6.0 (4.4-8.4)	0.883
Lymphocyte count, x10^3/µl	1.5 (1.0-2.6)	1.7 (1.1-2.5)	0.471
Monocyte count, x10^3/µl	0.7 (0.4-0.9)	0.7 (0.5-0.9)	0.195
Hemoglobin, g/dl	13.2±1.9	13.1±1.9	0.816
Platelet count, x10^3/µl	222 (184-282)	230 (175-294)	0.950

Data are given as mean± standard deviation, n (%), or median (interquartile range). CAD: Coronary artery disease, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, WBC: White blood cell

Table 2. Electrocardiographic and angiographic findings of the study population

Variables	Good collateral (n: 67)	Poor collateral (n: 64)	p-value
QTc dispersion (ms)	57.2±26.2	91.3±21.6	<0.001
QT dispersion (ms)	55.2±26.2	87.6±21.3	<0.001
Fragmante QRS (ms)	128.1±13.1	139.5±8.0	<0.001
Occluded artery, n (%)			
LAD	7 (10.4)	12 (18.8)	0.177
LCX	13 (19.4)	10 (15.6)	0.570
RCA	49 (73.1)	45 (70.3)	0.720

Data are given as mean±standard deviation or n (%), QT: The time from the beginning of the Q wave to the end of the T wave, QRS: Electrical activity of ventricular muscles, LAD: Left anterior descending artery, LCx: Left circumflex artery, RCA: Right coronary artery

Table 3. Correlation between the Rentrop classification and clinical variables

Parameters	r-value	p-value
Diabetes mellitus	-0.176	0.044
Creatinin	0.185	0.034
QT dispersion	-0.648	<0.001
QTc dispersion	-0.659	<0.001
Fragmented QRS	-0.557	<0.001

QT: The time from the beginning of the Q wave to the end of the T wave, QRS: Electrical activity of ventricular muscles

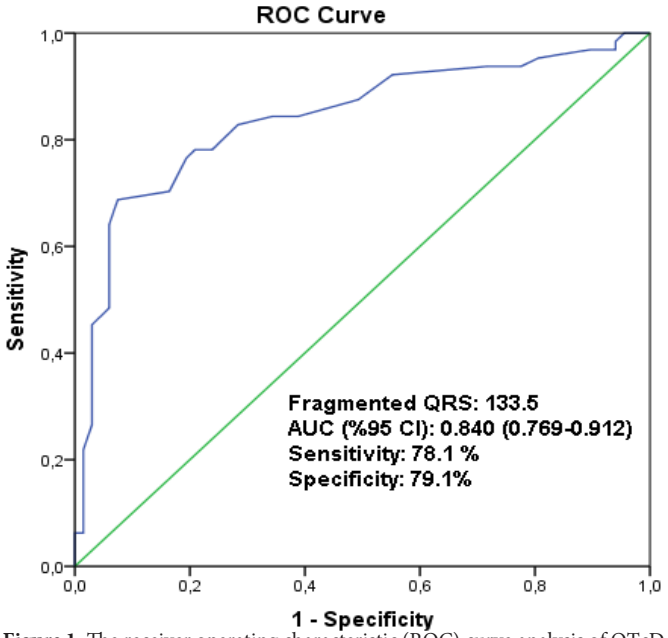

Figure 1. The receiver operating characteristic (ROC) curve analysis of QTcD for the Rentrop classification

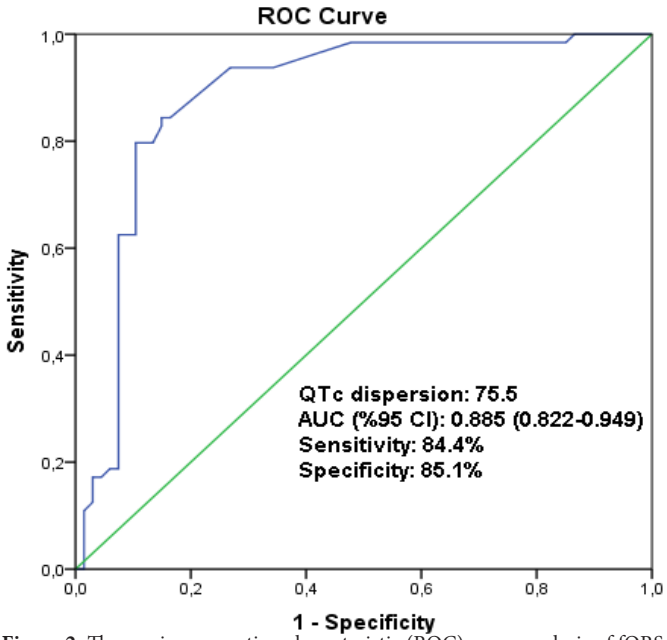

Figure 2. The receiver operating characteristic (ROC) curve analysis of fQRS for the Rentrop classification

fewer ventricular aneurysms, enhanced cardiac function, fewer future cardiac incidents, and better survival.[15] Enhanced repolarization dispersion is regarded as a contributing factor to the development of lethal ventricular arrhythmias.[16]

The intervals measured during the QT period reflect the duration from the start of the Q wave to the end of the T wave on an ECG, reflecting the duration of the activation wave's depolarization and the electrical recovery or repolarization of the cells. The shortest QT interval on a standard ECG represents the early repolarization region, while the lead showing the longest QT interval corresponds to the region of the ventricular myocardium that repolarizes last. The difference between these intervals is defined as QTD, which is considered an indicator of ventricular repolarization

variability and electrical instability.[17] Thus, as the QTD gets larger, the variability in the timing of electrical recovery in the heart becomes greater, which features a clinical significance, especially in the case of cardiac ischemia, as experimental studies have revealed that larger dispersion of repolarization is a crucial agent underlying severe and, fatal arrhythmias.[18] Various works have suggested that QTD also increases extraordinarily in conditions such as heart failure and ventricular hypertrophy, beyond coronary artery stenosis.[19,20]

In individuals with CAD, regional differences in repolarization, particularly in determining QTD, are prominent. The increased dispersion of ventricular repolarization at rest in CAD individuals may result in regional myocardial ischemia since ventricular repolarization can act more sensitively compared to other myocardial functions. Therefore, ischemic regions and/or fibrosis that may develop due to chronic ischemia can lead to electrical instability and heterogeneous repolarization in the myocardium, contributing to the high level of QTD.[21] It is believed that the increased QTD seen in CAD individuals is parallel to the severity of ischemia, partially caused by impaired responses to catecholamines or abnormal calcium ion flux in the ischemic myocardium.[22]

It has been stated in different works that ECG changes are often seen after stroke and that intracardiac sympathetic activity increases after intracranial ischemia and this triggers cardiac arrhythmias.[23] This situation is one of the cardiac causes of death in stroke individuals in the long term.[23] High troponin values and ECG changes can be seen in patients with acute ischemic stroke even if structural and ischemic heart diseases are excluded.[24]

Another parameter related to inflammation and ischemic changes is fQRS. fQRS is characterized by unpredicted deviations in the QRS morphology, but the exact reason of fragmentation in surface ECGs is not yet wholly understood. It has been argued that fQRS can prognosticate cardiac incidents in various cases. From a pathophysiological perspective, fQRS is usually considered to result from cardiac fibrosis, scarring, and ischemia, which lead to inhomogeneous cardiac electrical activation.[25] It has been demonstrated that fQRS is connected with cardiac fibrosis in individuals with ischemic or non-ischemic left ventricular dysfunction.[26] In previous studies that used gadolinium-enhanced delayed contrast cardiac magnetic resonance imaging to assess myocardial structure, fQRS was detected to be related to large cardiac scars.[27] fQRS complexes can be highly sensitive with a negative predictive rate than the Q wave in predicting prior myocardial infarction, as shown by scintigraphic assessments of regional perfusion abnormalities.[11] Regional fQRS complexes indicate the presence of larger regional myocardial scars, as demonstrated by stress myocardial perfusion imaging.[28] A work by Çetin et al.[29] identified a relationship between coronary atherosclerotic burden, CAD prevalence, and fQRS.

Another study suggested that fQRS may be present even if myocardial fibrosis cannot be detected with any technique. They stated that this could be due to the technique being inadequate to show low-grade fibrosis, depolarization abnormality, or a normal variant.[30] Çetin et al.[29] reported that fQRS may be associated with ventricular systolic functions in individuals with stable CAD or normal coronary arteries. In the same study, they reported that fQRS is causally related to inflammation.[29] It has also been proposed that both prolonged or shortened QRS duration and left ventricular systolic dysfunction serve as independent predictors of fQRS complexes on an ECG.[29]

The specified ECG parameters will provide useful information about CCC in CTO patients. Thus, we will have the opportunity to evaluate the treatment strategy in CTO patients more accurately.

Limitations

Some major limitations encountered during the study were the insufficient sample size, the use of manual measurements instead of computer software for calculating ECG parameters, and the inability to obtain evidence of ischemia. Other limitations include the complexity of local ischemia models and the fact that their contribution to the overall distribution of repolarization in patients has not been systematically investigated. The fact that the JT interval, which truly reflects ventricular repolarization, has not been evaluated.

CONCLUSION

This study suggests that increased QTcD, QTD, and the presence of fQRS are correlated with poor coronary collateral circulation in CAD individuals. Additional research should be conveyed to explain the connection between ECG parameters and collateral recovery.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Yozgat Bozok University Rectorate Non-interventional Clinical Researches Ethics Committee (Date: 04.12.2024, Decision No: 2024-GOKAEK-2414_2024.12.04_204).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Buxton AE, Ellison KE, Kirk MM, et al. Primary prevention of sudden cardiac death: trials in patients with coronary artery disease. *J Interv Card Electrophysiol*. 2003;9(2):203-206. doi:10.1023/a:1026236524273

2. Meissner MD, Akhtar M, Lehmann MH. Nonischemic sudden tachyarrhythmic death in atherosclerotic heart disease. *Circulation*. 1991;84(2):905-912. doi:10.1161/01.cir.84.2.905
3. Virmani R, Burke AP, Farb A. Sudden cardiac death. *Cardiovasc Pathol*. 2001;10(5):211-218. doi:10.1016/s1054-8807(01)00091-6
4. Das MK, Suradi H, Maskoun W, et al. Fragmented wide QRS on a 12-lead ECG: a sign of myocardial scar and poor prognosis. *Circ Arrhythm Electrophysiol*. 2008;1(4):258-268. doi:10.1161/CIRCEP.107.763284
5. Das MK, Michael MA, Suradi H, et al. Usefulness of fragmented QRS on a 12-lead electrocardiogram in acute coronary syndrome for predicting mortality. *Am J Cardiol*. 2009;104(12):1631-1637. doi:10.1016/j.amjcard.2009.07.046
6. Das MK, Saha C, El Masry H, et al. Fragmented QRS on a 12-lead ECG: a predictor of mortality and cardiac events in patients with coronary artery disease. *Heart Rhythm*. 2007;4(11):1385-1392. doi:10.1016/j.hrthm.2007.06.024
7. van Dongen IM, Elias J, Meijborg VMF, et al. Electrocardiographic changes after successful recanalization of a chronic total coronary occlusion. A systematic review and meta-analysis. *Cardiovasc Revasc Med*. 2018;19(2):221-228. doi:10.1016/j.carrev.2017.09.004
8. Goodhart DM, Hubacek J, Anderson TJ, et al. Effect of percutaneous coronary intervention of nonacute total coronary artery occlusions on QT dispersion. *Am Heart J*. 2006;151(2):529.e1-529.e6. doi:10.1016/j.ahj.2005.08.010
9. Kadı H, Ceyhan K, Koç F, Celik A, Onalan O. Relation between fragmented QRS and collateral circulation in patients with chronic total occlusion without prior myocardial infarction. *Anad Kardiyol Derg*. 2011;11(4):300-304. doi:10.5152/akd.2011.079
10. Suzuki M, Nishizaki M, Arita M, et al. Increased QT dispersion in patients with vasospastic angina. *Circulation*. 1998;98(5):435-440. doi:10.1161/01.cir.98.5.435
11. 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):2495-2501. doi:10.1161/CIRCULATIONAHA. 105.595892
12. Sianos G, Werner GS, Galassi AR, et al. Recanalisation of chronic total coronary occlusions: 2012 consensus document from the EuroCTO club. *EuroIntervention*. 2012;8(1):139-145. doi:10.4244/EIJV8I1A21
13. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. *J Am Coll Cardiol*. 1985;5(3):587-592. doi:10.1016/s0735-1097(85)80380-6
14. Seiler C, Stoller M, Pitt B, Meier P. The human coronary collateral circulation: development and clinical importance. *Eur Heart J*. 2013;34(34):2674-2682. doi:10.1093/eurheartj/eht195
15. Meier P, Hemingway H, Lansky AJ, Knapp G, Pitt B, Seiler C. The impact of the coronary collateral circulation on mortality: a meta-analysis. *Eur Heart J*. 2012;33(5):614-621. doi:10.1093/eurheartj/ehr308
16. Okin PM, Devereux RB, Howard BV, Fabsitz RR, Lee ET, Welty TK. Assessment of QT interval and QT dispersion for prediction of all-cause and cardiovascular mortality in American Indians: the strong heart study. *Circulation*. 2000;101(1):61-66. doi:10.1161/01.cir.101.1.61
17. Day CP, McComb JM, Campbell RW. QT dispersion: an indication of arrhythmia risk in patients with long QT intervals. *Br Heart J*. 1990;63(6):342-344. doi:10.1136/hrt.63.6.342
18. Janse MJ, Wit AL. Electrophysiological mechanisms of ventricular arrhythmias resulting from myocardial ischemia and infarction. *Physiol Rev*. 1989;69(4):1049-1169. doi:10.1152/physrev.1989.69.4.1049
19. Buja G, Miorelli M, Turrini P, Melacini P, Nava A. Comparison of QT dispersion in hypertrophic cardiomyopathy between patients with and without ventricular arrhythmias and sudden death. *Am J Cardiol*. 1993;72(12):973-976. doi:10.1016/0002-9149(93)91118-2
20. Barr CS, Naas A, Freeman M, Lang CC, Struthers AD. QT dispersion and sudden unexpected death in chronic heart failure. *Lancet*. 1994;343(8893):327-329. doi:10.1016/s0140-6736(94)91164-9
21. Elsässer A, Schlepper M, Klövekorn WP, et al. Hibernating myocardium: an incomplete adaptation to ischemia. *Circulation*. 1997;96(9):2920-2931. doi:10.1161/01.cir.96.9.2920
22. Arab D, Valeti V, Schünemann HJ, López-Candales A. Usefulness of the QTc interval in predicting myocardial ischemia in patients undergoing exercise stress testing. *Am J Cardiol*. 2000;85(6):764-A8. doi:10.1016/s0002-9149(99)00858-9
23. Oppenheimer SM. Neurogenic cardiac effects of cerebrovascular disease. *Curr Opin Neurol*. 1994;7(1):20-24. doi:10.1097/00019052-199402000-00005
24. Yıldırım S, Kaşıkcı MT. Examination of troponin levels and electrocardiographic changes in patients with acute ischemic stroke. *Muğla Sıtkı Koçman Uni Med J*. 2020; 7(2): 65-69.
25. Basaran Y, Tigen K, Karaahmet T, et al. Fragmented QRS complexes are associated with cardiac fibrosis and significant intraventricular systolic dyssynchrony in nonischemic dilated cardiomyopathy patients with a narrow QRS interval. *Echocardiography*. 2011;28(1):62-68. doi:10.1111/j.1540-8175.2010.01242.x
26. Calore C, Cacciavillani L, Boffa GM, et al. Contrast-enhanced cardiovascular magnetic resonance in primary and ischemic dilated cardiomyopathy. *J Cardiovasc Med (Hagerstown)*. 2007;8(10):821-829. doi:10.2459/JCM.0b013e3280101e3c
27. Chatterjee S, Changawala N. Fragmented QRS complex: a novel marker of cardiovascular disease. *Clin Cardiol*. 2010;33(2):68-71. doi:10.1002/clc.20709
28. Mahenthiran J, Khan BR, Sawada SG, Das MK. Fragmented QRS complexes not typical of a bundle branch block: a marker of greater myocardial perfusion tomography abnormalities in coronary artery disease. *J Nucl Cardiol*. 2007;14(3):347-353. doi:10.1016/j.nuclcard.2007.02.003
29. Cetin M, Kocaman SA, Canga A, et al. The independent relationship between systemic inflammation and fragmented QRS complexes in patients with stable angina pectoris. *Kardiol Pol*. 2012;70(7):668-675.
30. MacAlpin RN. The fragmented QRS: does it really indicate a ventricular abnormality? *J Cardiovasc Med (Hagerstown)*. 2010;11(11):801-809. doi:10.2459/JCM.0b013e32833b9816