

COVID-19 survivors may exhibit deterioration in frontal plane QRS-T angle and other electrocardiogram parameters

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Abstract. – OBJECTIVE: COVID-19 infection is known to injure myocardial tissue and increase arrhythmic events. However, data on the subject is limited in the literature. In our study, our aim was to investigate possible arrhythmic damages in COVID-19 survivors using the frontal plane QRS-T [f(QRS)-T] angle and a few other ECG parameters.

PATIENTS AND METHODS: 269 patients who recovered from COVID-19 between April 2020 and January 2021 were included into the study. Pre-admission electrocardiograms and first-month outpatient clinic control ECGs of the patients were compared.

RESULTS: After COVID-19, left bundle branch block ($p<0.001$), right bundle branch block ($p<0.001$), right bundle branch block ($p<0.001$), and atrial fibrillation ($p<0.001$) rates had increased. Prolongation was detected in QRS duration ($p<0.001$), QT interval ($p=0.014$), adjusted QT interval ($p=0.007$) and Tpe interval ($p=0.012$). F(QRS)-T angle ($p<0.001$) and fragmented QRS rate ($p<0.001$) were increased.

CONCLUSIONS: It was observed in our study that even if patients survived COVID-19, permanent deterioration in ECG parameters may occur.

Key Words:

COVID-19, Frontal plane QRS-T angle, Electrocardiogram.

suffer from acute myocardial or elevation of cardiac troponin^{2,3}. Around 6-17% of these patients have also developed malignant ventricular arrhythmias, more prevalent among those in the intensive care unit^{4,5}. The prevalence of cardiac arrhythmia is conversely correlated with clinical stability⁶. Thus, critical cases pose a higher risk of developing cardiac arrhythmia⁷. Although most arrhythmic events are temporary, data are limited regarding permanent arrhythmic injury in survivors⁶. Frontal plane QRS-T [f(QRS-T)] angle is a novel marker for measuring heterogeneity of ventricular repolarization⁸. It is defined as the angle between the ventricular depolarization (QRS axis) and repolarization (T axis). Most of the 12-lead ECG devices report the QRS and T-wave axes automatically, so electrocardiography (ECG) allows the f(QRS-T) angle to be measured quite simply⁸. Previous research^{9,10} has emphasized that the f(QRS-T) angle has prognostic value among various patient populations. Additionally, one study¹¹ has demonstrated the distinguishing value of wider f(QRS-T) angles ($> 90^\circ$) in terms of long-term mortality in cases with left ventricular systolic dysfunction following acute myocardial infarction.

Here, we aimed at examining the possibility of permanent arrhythmic effects in COVID-19 survivors using f(QRS-T) and other repolarization parameters.

Introduction

First reported in December 2019 in Wuhan, China, COVID-19 is an infectious disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Since then, the disease has rapidly spread around the globe, resulting in the ongoing COVID-19 pandemic¹. Recent data show that 20-36% of COVID-19 patients

Patients and Methods

269 patients who had been hospitalized due to COVID-19, had recovered from the disease, and then had come to the cardiology outpatient clinic one month after hospitalization in the Training

and Research Hospital in the Faculty of Medicine at Suleyman Demirel University between April 2020 and January 2021 were included in the study. Pre-admission electrocardiograms and first-month outpatient clinic control ECGs of the patients were compared. To detect SARS-CoV-2 RNA, a reverse-transcription polymerase chain reaction (RT-PCR) test was used. The criteria for hospitalization in the study were consistent with those developed by the Turkish Ministry of Health, including age > 50 years, moderate or severe symptoms, at least one risk factor on presentation (HT, DM, chronic pulmonary disease, chronic heart disease, chronic kidney failure, or immune-deficiency), at least one poor prognostic factor (lymphopenia, high levels of C-reactive protein, ferritin, or D-dimer) and a social indication, such as inappropriate home conditions or a problem of compliance with isolation rules. Criteria for discharge were as follows: absence of fever for at least 3 days, improved lung condition (both lungs) on chest computed tomography, clinical remission of respiratory symptoms, and a negative throat-swab sample. The study was performed according to the Helsinki Declaration and approved by an institutional ethics committee and the Ministry of Health.

Detailed medical history was collected, and a complete physical examination was performed. 12-lead ECG was used to evaluate the patients, and hypertension was diagnosed as systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg in at least 3 measurements, or anti-hypertensive drug use. Diabetes mellitus was diagnosed as fasting blood glucose ≥ 7.0 mmol/L or anti-diabetic drug use. Hyperlipidemia was defined as total cholesterol ≥ 5.2 mmol/L or statin use history except for the previous 3 months. Individuals who had been smoking prior to their hospitalization were defined as smokers.

Electrocardiography

A 12-lead ECG was used in the supine position and at 50 mm/s paper speed (Nihon Kohden, Tokyo, Japan). All ECGs were scanned and magnified by $\times 400\%$ on a computer using Adobe Photoshop to reduce errors. QT and Tp-e intervals on ECGs were measured by two cardiologists blinded to patients' data. Subjects with U waves on ECGs were excluded. QT intervals were measured from the beginning of the QRS complex to the end of the T-wave and then corrected for heart rate using Bazett's formula: $cQT = QT\sqrt{(R-R \text{ interval})}$. Tp-e intervals were measured from pre-

cordial leads as the peak to end T-wave¹². Since the ECG device automatically gives the frontal QRS and T-wave axes, the f(QRS-T) angle was calculated as the absolute difference between them (subtracted from 360° if $> 180^\circ$)^{8,9,13}. The f(QRS-T) angle was calculated based on the automatic report of the ECG device to rule out the subjective component.

Statistical Analysis

Data analyses were carried out using the Statistical Package for Social Sciences (SPSS) for Windows 20 software (IBM Corp., Armonk, NY, USA). Conformity to normal distribution was tested using the Kolmogorov-Smirnov test. Normally distributed variables were given as mean \pm standard deviation, those with skewed distribution as median (interquartile range) and categorical variables as a percentage (%). Differences between the serial ECG parameters of patients were evaluated using One Way Analysis of Variance (ANOVA), and if sphericity was violated, a Greenhouse-Geisser correction was performed. Comparisons between two groups were assessed using the unpaired *t*-test for normally distributed variables and the nonparametric Mann-Whitney U test for non-normally distributed variables. The level of statistical significance was set at *p*-values < 0.05 for all measurements.

Results

Among the patient group, the mean age was 66.3 ± 10.1 , the ratio of the female patients was 40.9%, the mean body mass index was 26.58 ± 3.89 , the diabetes' rate was 27.9%, the hypertension rate was 48.3%, the smoking rate was 42.8%, the congestive heart failure rate was 20.4%. Admission rate to the intensive care unit was 13.0%, and the intubation rate was 2.6%. Favipiravir usage rate was 98.1%, hydroxychloroquine usage rate was 94.4% (Table I).

When ECG parameters were compared between hospitalization and polyclinic control, no statistically significant difference was found in *p* wave duration ($p < 0.001$). Left bundle branch block ($p < 0.001$), right bundle branch block ($p < 0.001$) atrial fibrillation ($p < 0.001$) rates had increased. Prolongation was detected in QRS duration ($p < 0.001$), QT interval ($p = 0.014$), adjusted QT interval ($p = 0.007$) and Tp-e interval ($p = 0.012$). F(QRS)-T angle ($p < 0.001$) and fragmented QRS rate ($p < 0.001$) were increased (Table II).

Table I. Demographic, echocardiographic and surgical characteristics of the patients.

Variables	(N = 269)
Age	66.3 ± 10.1
Female n (%)	110 (40.9%)
Body mass index, kg/m ²	26.58 ± 3.89
Diabetes Mellitus, n (%)	75 (27.9%)
Hypertension, n (%)	130 (48.3%)
Hyperlipidemia, n (%)	63 (23.4%)
Smoking, n (%)	115 (42.8%)
Congestive heart failure, n (%)	55 (20.4%)
Chronic obstructive pulmonary disease, n (%)	51 (19.0%)
Chronic kidney disease, n (%)	40 (14.9%)
Intensive care unit hospitalization, (%)	35 (13.0%)
Intubation, (%)	7 (2.6%)
Favipiravir usage, (%)	264 (98.1%)
Hydroxychloroquine, (%)	254 (94.4%)

Data are given as mean ± standard deviation or percentage [n (%)].

Discussion

In this study, we observed that permanent deterioration in ECG parameters might occur even if patients survive after COVID-19.

f(QRS-T) is defined as the angle between ventricular depolarization and repolarization as a novel marker for heterogeneity^{8,13}. Compared to more traditional parameters for myocardial repolarization on ECG, the QRS-T angle has been reported to provide better reliability and to be less susceptible to noise and definition issues^{13,14}. It can be calculated on a 3D space as spatial QRS-T angle or on a 12-lead ECG as frontal QRS-T angle^{8,9}. The first remains too complex to allow routine calculation using ECG devices^{8,9}. Yet, the f(QRS-T) is easier to

measure as the absolute difference between the QRS and T axes on ECG¹⁵. Previous research also suggests f(QRS-T) as a suitable substitute for spatial QRS-T angle in predicting coronary artery diseases and total mortality¹⁶. Due to the similar orientation of myocardial depolarization and repolarization axes, the f(QRS-T) angle tends to be narrow (<45°)¹⁷. A wider f(QRS-T) angle indicates inconsistency between ventricular depolarization and repolarization phases. This allows physicians to predict poor outcomes in ST elevation myocardial infarction (STEMI) cases¹¹.

The pathophysiological mechanisms leading to heart injury as a result of COVID-19 remain unclear and require further research. Oudit et al¹⁸ found SARS-CoV viral RNA in human heart samples, suggesting direct myocyte invasion by the virus, and reported a down-regulation of ACE-2 protein. The authors hypothesized the contribution of the interaction between SARS-CoV and ACE-2 to myocardial inflammation and damage. They also highlighted significant myocardial and Purkinje system macrophage infiltration in heart samples. This down-regulation of ACE-2 would hamper the protective effects of angiotensin 1-7, resulting in increased production of TNFα¹⁹. Many studies have shown that an excessive inflammatory response may be responsible for damage to the heart's conduction system. Guo et al¹⁹ reported increased levels of C-reactive protein and TnT in patients with an underlying cardiovascular disease and poor outcomes. Their findings have supported the hypothesis that severe inflammatory response could mediate Purkinje system damage. Since interferons cause a shift to adaptive protective immunity, interferon-mediated responses have

Table II. Electrocardiographic features of the groups.

Variables	Hospitalization ECG	Day 30, ECG	p-value
Left bundle branch block, n (%)	32 (11.9%)	43 (16.0%)	< 0.001
Right bundle branch block, n (%)	38 (14.1%)	53 (19.7%)	< 0.001
Atrial fibrillation, n (%)	30 (11.2%)	38 (14.1%)	< 0.001
QRS, ms	110 ± 22	119 ± 21	< 0.001
P wave, ms	94 ± 18	95 ± 19	0.731
QT, ms	346 ± 29	368 ± 30	0.014
QTc, ms	390 ± 36	404 ± 23	0.007
Tpe, ms	81 ± 10	84 ± 15	0.012
f(QRS)-T (°)	56.1 ± 30.5	74.1 ± 39.3	< 0.001
Fragmented QRS, n (%)	21 (7.8%)	30 (11.2%)	< 0.001

Data are given as mean ± standard deviation or percentage [n (%)]. ECG: electrocardiogram; QTc - corrected QT interval; f(QRS) - T; frontal QRS-T angle.

been reported to possibly contribute to myocardial dysfunction in SARS patients. Exaggerated cytokine response by Type 1 and 2 helper T cells is another mechanism that may play a role²⁰. Considering the significant resemblance between SARS infection and COVID-19, these mechanisms may be involved in system damage in the latter, aside from systemic inflammation, myocardial interstitial fibrosis, coronary plaque destabilization, and hypoxia.

It is well known that autonomic imbalance is involved in arrhythmogenicity²¹. Inflammatory cytokines can activate the vagus nerve to control inflammation through acetylcholine and modulate the immune system by inhibiting inflammatory cytokines in macrophages and the spleen²². In this regard, reduced cardiac vagal activity in pulmonary and cardiometabolic diseases negatively affects critically ill cases, like COVID-19²³. However, increased cardiac vagal activity can protect against this cytokine release syndrome²⁴. Research emphasizes the importance of the cholinergic anti-inflammatory pathway in COVID-19. This explains why neuromodulation treatment is suggested for controlling inflammation and loss of organ function in COVID-19²⁴. As a key element of autonomic dysregulation in infections and hyper-inflammatory syndromes, hyperactivation of sympathetic nervous system, and reduced vagal activity due to the virus may result in a severe loss of autonomic balance, contributing to arrhythmogenicity.

There is limited reliable data on arrhythmias associated with COVID-19, and the current knowledge fails to support any discrimination between the direct myocardial effect of the virus and arrhythmias due to hypoxemia, metabolic or electrolyte abnormality, inflammatory syndrome, or drug use. One study on 234 patients with COVID-19 reported cardiac arrhythmia in 10 (4.3%) patients, 3 of them had pre-existing hypertension, 2 had coronary disease, and 8 were critically ill. The authors associated this finding and the lack of a history of cardiovascular disease in most of these patients with COVID-19²⁵. Creatine kinase levels at admission were found to have the highest predictive value in terms of arrhythmia²⁵. This data is consistent with other research²⁶ showing cardiac injury as a critical risk factor for arrhythmogenesis and mortality. Evident by increased troponin levels, myocardial injury is frequent in COVID-19 cases and is connected to poor outcomes. These

include arrhythmias and mortality, both being higher in prevalence in patients with a chronic cardiovascular status or severe progression of COVID-19^{27,28}. Recent findings on 36 pediatric patients highlight a rare occurrence of significant arrhythmias, although still higher than what would be expected from the general population. Of these 6 cases of arrhythmias, 5 had non-sustained ventricular tachycardia (NS-VT), and one had sustained atrial tachycardia, all of which resolved spontaneously²⁹. Despite the association between noncardiac comorbidities and significant arrhythmias in this patient group, the findings were similar in cases with no arrhythmia ($p=1$). The use of HCQ with or without AZM was significantly correlated with prolonged QTc ($p<0.0001$). Yet again, QTc did not differ between the patient groups with and without arrhythmias ($p=1$).

Our knowledge on the arrhythmogenic mechanisms in COVID-19 remains limited. Thus, further research should focus on the prevalence of arrhythmias, associated mechanisms, cautionary measures, safe treatment methods, and suitable monitoring in COVID-19 patients.

Limitations of the Study

The present study is a cross-sectional study with a relatively small sample size. Data on major adverse cardiovascular events during the follow-up was not available for the patient population studied. Therefore, the results of the current study should be verified in multi-center prospective longitudinal studies on larger sample sizes. The limitations of this study should be considered when interpreting the results.

Conclusions

We observed a significant deterioration in ECG parameters in COVID-19 survivors. Our study demonstrates the importance of preventive cardiological strategies in COVID-19 patients. However, further studies are needed to better establish arrhythmic events in COVID-19.

Conflict of Interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and publication of this article.

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Ethical Approval

This study was approved by the Suleyman Demirel University Faculty of Medicine Ethics Committee (date: 12/02/2021, No. 80).

Informed Consent

The patients signed a written informed consent for the study.

References

- 1) Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, Zhao X, Huang B, Shi W, Lu R, Niu P, Zhan F, Ma X, Wang D, Xu W, Wu G, Gao GF and Tan W. A Novel Coronavirus from Patients with Pneumonia in China, 2019. *N Engl J Med* 2020; 382: 727-733.
- 2) Parohan M, Yaghoubi S and Seraji A. Cardiac injury is associated with severe outcome and death in patients with Coronavirus disease 2019 (COVID-19) infection: A systematic review and meta-analysis of observational studies. *Eur Heart J Acute Cardiovasc Care* 2020; 9: 665-677.
- 3) Lala A, Johnson KW, Januzzi JL, Russak AJ, Paranjpe I, Richter F, Zhao S, Somani S, Van Vleck T, Vaid A, Chaudhry F, De Freitas JK, Fayad ZA, Pinney SP, Levin M, Charney A, Bagiella E, Narula J, Glicksberg BS, Nadkarni G, Mancini DM and Fuster V. Prevalence and Impact of Myocardial Injury in Patients Hospitalized With COVID-19 Infection. *J Am Coll Cardiol* 2020; 76: 533-546.
- 4) Bhatla A, Mayer MM, Adusumalli S, Hyman MC, Oh E, Tierney A, Moss J, Chahal AA, Anesi G, Denduluri S, Domenico CM, Arkles J, Abella BS, Bullinga JR, Callans DJ, Dixit S, Epstein AE, Frankel DS, Garcia FC, Kumareswaram R, Nazarian S, Riley MP, Santangeli P, Schaller RD, Supple GE, Lin D, Marchlinski F and Deo R. COVID-19 and cardiac arrhythmias. *Heart Rhythm* 2020; 17: 1439-1444.
- 5) Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, Wang B, Xiang H, Cheng Z, Xiong Y, Zhao Y, Li Y, Wang X and Peng Z. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. *JAMA* 2020; 323: 1061-1069.
- 6) Sala S, Peretto G, De Luca G, Farina N, Campochiaro C, Tresoldi M, Dagna L, Zangrillo A, Gulletta S and Della Bella P. Low prevalence of arrhythmias in clinically stable COVID-19 patients. *Pacing Clin Electrophysiol* 2020; 43: 891-893.
- 7) Lazaridis C, Vlachogiannis NI, Bakogiannis C, Spyridopoulos I, Stamatelopoulos K, Kanakakis I, Vassilikos V and Stellos K. Involvement of cardiovascular system as the critical point in coronavirus disease 2019 (COVID-19) prognosis and recovery. *Hellenic J Cardiol* 2020; 61: 381-395.
- 8) Macfarlane PW. The frontal plane QRS-T angle. *EP Europace* 2012; 14: 773-775.
- 9) Andrew O, Trevor F, A. HC and G. TL. QRS-T Angle: A Review. *Ann Noninvasive Electrocardiol* 2014; 19: 534-542.
- 10) Aro AL, Huikuri HV, Tikkanen JT, Junttila MJ, Rissanen HA, Reunanen A and Anttonen O. QRS-T angle as a predictor of sudden cardiac death in a middle-aged general population. *Europace* 2012; 14: 872-876.
- 11) Raposeiras-Roubin S, Virgos-Lamela A, Bouzas-Cruz N, Lopez-Lopez A, Castineira-Busto M, Fernandez-Garda R, Garcia-Castelo A, Rodriguez-Manero M, Garcia-Acuna JM, Abu-Assi E and Gonzalez-Juanatey JR. Usefulness of the QRS-T angle to improve long-term risk stratification of patients with acute myocardial infarction and depressed left ventricular ejection fraction. *Am J Cardiol* 2014; 113: 1312-1319.
- 12) Hevia JC, Antzelevitch C, Bárzaga FT, Sánchez MD, Balea FD, Molina RZ, Quiñones Pérez MA and Rodríguez YF. Tpeak-Tend and Tpeak-Tend Dispersion as Risk Factors for Ventricular Tachycardia/Ventricular Fibrillation in Patients With the Brugada Syndrome. *J Am Coll Cardiol* 2006; 47: 1828-1834.
- 13) Zhang Z-m, Rautaharju PM, Prineas RJ, Tereshchenko L and Soliman EZ. Electrocardiographic QRS-T angle and the risk of incident silent myocardial infarction in the Atherosclerosis Risk in Communities study. *J Electrocardiol* 2017; 50: 661-666.
- 14) Dilaveris P, Antoniou CK, Gatzoulis K and Tousoulis D. T wave axis deviation and QRS-T angle - Controversial indicators of incident coronary heart events. *J Electrocardiol* 2017; 50: 466-475.
- 15) Li YH, Ren XJ, Han ZH, Wang YL, Wang Y, Zhang JR and Chen F. Value of the frontal planar QRS-T angle on cardiac dysfunction in patients with old myocardial infarction. *Int J Clin Exp Med* 2013; 6: 688-692.
- 16) Zhang ZM, Prineas RJ, Case D, Soliman EZ and Rautaharju PM. 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: 844-849.
- 17) Gungor M, Celik M, Yalcinkaya E, Polat AT, Yuksel UC, Yildirim E, Firtina S, Bugan B and Ozer AC. The Value of Frontal Planar QRS-T Angle in Patients without Angiographically Apparent Atherosclerosis. *Med Princ Pract* 2017; 26: 125-131.
- 18) Oudit GY, Kassiri Z, Jiang C, Liu PP, Poutanen SM, Penninger JM and Butany J. SARS-corona-

- virus modulation of myocardial ACE2 expression and inflammation in patients with SARS. *Eur J Clin Invest* 2009; 39: 618-625.
- 19) Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, Wang H, Wan J, Wang X and Lu Z. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19). *JAMA Cardiol* 2020; 5: 811-818.
 - 20) Cameron MJ, Bermejo-Martin JF, Danesh A, Muller MP and Kelvin DJ. Human immunopathogenesis of severe acute respiratory syndrome (SARS). *Virus Res* 2008; 133: 13-19.
 - 21) Manolis AA, Manolis TA, Apostolopoulos EJ, Apostolaki NE, Melita H and Manolis AS. The role of the autonomic nervous system in cardiac arrhythmias: The neuro-cardiac axis, more foe than friend? *Trends Cardiovasc Med* 2021; 31: 290-302.
 - 22) Tracey KJ. The inflammatory reflex. *Nature* 2002; 420: 853-859.
 - 23) Ackland GL, Whittle J, Toner A, Machhada A, Del Arroyo AG, Sciuso A, Jenkins N, Dyson A, Struthers R, Sneyd JR, Minto G, Singer M, Shah AM and Gourine AV. Molecular Mechanisms Linking Autonomic Dysfunction and Impaired Cardiac Contractility in Critical Illness. *Crit Care Med* 2016; 44: e614-624.
 - 24) Fudim M, Qadri YJ, Ghadimi K, MacLeod DB, Mollinger J, Piccini JP, Whittle J, Wischmeyer PE, Patel MR and Ulloa L. Implications for Neuromodulation Therapy to Control Inflammation and Related Organ Dysfunction in COVID-19. *J Cardiovasc Transl Res* 2020; 13: 894-899.
 - 25) Liu Q, Chen H and Zeng Q. Clinical characteristics of COVID-19 patients with complication of cardiac arrhythmia. *J Infect* 2020; 81: e6-e8.
 - 26) Si D, Du B, Ni L, Yang B, Sun H, Jiang N, Liu G, Massé S, Jin L, Nanthakumar J, Bhaskaran A, Yang P and Nanthakumar K. Death, discharge and arrhythmias among patients with COVID-19 and cardiac injury. *CMAJ* 2020; 192: E791.
 - 27) Sandoval Y, Januzzi JL and Jaffe AS. Cardiac Troponin for Assessment of Myocardial Injury in COVID-19: JACC Review Topic of the Week. *J Am Coll Cardiol* 2020; 76: 1244-1258.
 - 28) Zaman S, Maclsaac AI, Jennings GL, Schlaich MP, Inglis SC, Arnold R, Kumar S, Thomas L, Wahi S, Lo S, Naismith C, Duffy SJ, Nicholls SJ, Newcomb A, Almeida AA, Wong S, Lund M, Chew DP, Kritharides L, Chow CK and Bhindi R. Cardiovascular disease and COVID-19: Australian and New Zealand consensus statement. *Med J Aust* 2020; 213: 182-187.
 - 29) Samuel S, Friedman RA, Sharma C, Ganigara M, Mitchell E, Schleien C and Blafox AD. Incidence of arrhythmias and electrocardiographic abnormalities in symptomatic pediatric patients with PCR-positive SARS-CoV-2 infection, including drug-induced changes in the corrected QT interval. *Heart Rhythm* 2020; 17: 1960-1966.