Erciyes Med J 2021; 43(6): 574–8 • DOI: 10.14744/etd.2021.93902
ORIGINAL ARTICLE – OPEN ACCESS

Ventricular Repolarization Parameters in Hospitalized Patients with COVID-19

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ABSTRACT

Objective: Coronavirus disease-2019 (COVID-19) may cause atrial and/or ventricular arrhythmias and sudden cardiac deaths, especially in severe cases. In this study, we aimed to investigate whether COVID-19 affects the ventricular repolarization parameters.

Materials and Methods: In this study, 152 hospitalized patients with COVID-19 were included retrospectively. Age and sex-matched 151 healthy subjects were assigned as the control group. QT, QTc, Tpe, Tpe/QT, and Tpe/QTc values of all participants were determined and compared between both groups. The relationship between high sensitive-troponin I (hsTnI) and the parameters mentioned was analyzed using the Spearman correlation test.

Results: Mean age, male gender and comorbidity rates were similar in both groups ($p>0.05$ for each). Heart rate, QTc, Tpe, Tpe/QT, and Tpe/QTc values were higher in the study group ($p<0.001$ for each). However, QT and QRS values were comparable in both groups ($p>0.05$ for each). There were positive correlations between hsTnI and QT (rho=0.218, p=0.008), QTc (rho=0.308, $p<0.001$), Tpe (rho=0.646, $p<0.001$), Tpe/QT (rho=0.571, $p<0.001$), and Tpe/QTc (rho=0.608, $p<0.001$).

Conclusion: In patients with COVID-19, QTc, Tpe, Tpe/QT, and Tpe/QTc values are higher than the control group and these parameters correlate positively with hsTnI. Therefore, ECG follow-up may be beneficial in preventing arrhythmic events, especially in patients with acute cardiac injuries.

Keywords: COVID-19, acute cardiac injury, malignant ventricular arrhythmia, T peak-to-end, T peak-to-end/QT

Cite this article as:
Sivri S, Kıvrak A, Hızmalı L. Ventricular Repolarization Parameters in Hospitalized Patients with COVID-19. Erciyes Med J 2021; 43(6): 574-8.

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Submitted
11.01.2021

Accepted
23.02.2021

Available Online
02.04.2021

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INTRODUCTION

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a viral pathogen that appeared in Wuhan, Hubei Province, China, at the end of 2019 and spread to the whole world in a short time. Full-genome sequencing and phylogenic analysis have shown that SARS-CoV-2 is a member of the RNA betacoronaviruses, such as human severe acute respiratory syndrome (SARS) and the Middle East respiratory syndrome (MERS) (1). The disease caused by this pathogen is called the coronavirus disease-2019 (COVID-19). Patients with COVID-19 may be asymptomatic or presented with symptoms, such as fever, nonproductive cough, dyspnea, myalgia, fatigue, pneumonia and gastrointestinal tract symptoms, such as nausea and diarrhea. Especially elderly patients and patients with comorbidity can be complicated with shock, acute respiratory distress syndrome (ARDS), acute kidney injury, secondary infection, and death (2, 3). Additively, major cardiovascular complications are not uncommon, especially in severe cases requiring intensive care units (ICU) (3–6). In a study conducted by Shi et al. (4), 19.7% of 416 hospitalized patients diagnosed with COVID-19 had acute cardiac injuries, defined by high sensitive-troponin I (hsTnI) >99th percentile. Wang et al. (5) reported the incidence of shock, arrhythmia, and acute cardiac injury in patients with 2019 novel coronavirus-infected pneumonia requiring ICU, as 30.6%, 22.2%, and 44.4%, respectively. However, there was no detailed information about the definition and classification of the arrhythmias mentioned in that study. In another study, Guo et al. (6) showed that 27.8% of 187 patients with COVID-19 had myocardial damage detected with elevated troponin T levels. In that study, malignant ventricular arrhythmias, such as ventricular fibrillation (VF) and ventricular tachycardia (VT), occurred in 5.9% of the total study population and 17.3% of a subgroup with elevated cardiac injury markers. In addition, cases first presenting with myopericarditis have been reported, which creates a substrate for outcomes, such as malignant ventricular arrhythmia and sudden cardiac death (2, 7, 8).

Ventricular repolarization features can be assessed and interpreted by QT interval and T wave changes on electrocardiogram (ECG). The QT interval (or corrected QT) (the total duration of ventricular depolarization and repolarization), the T peak-to-end (Tpe) interval, and the Tpe/QT ratio predict the spatial dispersion of ventricular repolarization, and they are parameters predicting the risk of malignant ventricular arrhythmia and arrhythmic death (9–11). In our study, we investigated whether QT, QTc, Tpe, Tpe QT, and Tpe/QTc values were different on hospital admission in these patients than the control group and whether there was a relationship between these parameters and hsTnI.

MATERIALS and METHODS

Study Population

We analyzed retrospectively 152 consecutive cases of COVID-19, treated with hospitalization between March-June 2020, in the department of infectious diseases and clinical microbiology of our hospital. COVID-19 diagnosis was made based on 'Clinical management of severe acute respiratory infection when novel coronavirus (2019-nCoV) infection was suspected: interim guidance' by the World Health Organization (12). Moreover, age and sex-matched 151 healthy subjects were included in this case-control study as the control group. The patients with atrial fibrillation, pacemaker, bundle branch block, unmeasurable T waves, antiarrhythmic drugs that could affect the QT interval, heart failure, active or chronic infection except from COVID-19, and inflammatory diseases were excluded from this study.

Blood samples were taken from each patient on admission to evaluate the complete blood count, liver and kidney function tests, cardiac injury markers, inflammatory status markers and bleeding profile. Access hsTnI Assay kits (reference range: 0–17.5 pg/mL) (Beckman Coulter Inc., İstanbul, TURKEY) were used for measurement of cardiac troponin I levels.

Our study was planned in accordance with the Helsinki Declaration criteria and approved by Ahi Evran University ethical committee (No: 2020-09/74). All participants signed the written consent form before conducting this study.

ECG Examination

12-leads standard ECG records were obtained using a MAC 2000 (GE Medical Systems Information Technologies, USA). The ECG recordings were scanned and then transferred to the personal computer. The measurements were made using Adobe Photoshop program by making 400% magnification. Ventricular repolarization parameters were measured on precordial lead 5 (V_5) and the mean value of the consecutive three beats were recorded (13, 14).

The QT interval was measured from the beginning of the QRS complex to the end of the T wave, and then QTc interval was obtained using Bazett's formula ($QTc=QT/RR^{-2}$). The Tpe interval was measured as the duration between the T wave's peak point and down slope tangent intersecting with the isoelectric line (the tangent method) (15).

ECG assessments were made by two cardiologists blinded to the study data. The interobserver and intraobserver coefficients of variation were 4.4% and 4.1%, respectively.

Statistical Analysis

The statistical analysis of this study was performed using Statistical Package for Social Sciences version 21.0 software for Windows (IBM SPSS Statistics for Windows, USA). The Kolmogorov-Smirnov and Shapiro-Wilk tests were used for the normality examination. Descriptive data of the variables were expressed as mean±standard deviation, or median (25^{th} percentile–75^{th} percentile), or frequency (%). Univariate analysis of the study parameters was performed using the chi-square test, continuity correction test, independent t-test, and Mann-Whitney U tests. Correlations between variables were analyzed by Spearman correlation test. P-value <0.05 was considered statistically significant.

RESULTS

Table 1 shows clinical, demographic, biochemical, and electrocardiographic features of the study population. Mean age (49.54±14.43 vs. 51.47±18.04), male gender and comorbidities, such as diabetes mellitus (DM), hypertension (HT), coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD), and malignancy, were comparable in both groups (p>0.05 for each).

In biochemical data comparison, lymphocyte, platelet, potassium, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total bilirubin, creatine kinase (CK) and lactate dehydrogenase (LDH) values were similar in both groups (p>0.05 for each). However, white blood cell count (WBC) (8.06±2.04 vs. 9.24±4.53, p=0.004), neutrophile (5.33±1.92 vs. 6.56±4.06, p=0.034), glucose (111.32±39.16 vs. 137.82±61.21, p<0.001), urea (29.84±9.38 vs. 41.59±10.64, p=0.001), creatinine (0.85±0.21 vs. 1.07±0.65, p=0.007), and C-reactive protein (0.48±0.53 vs. 7.12±2.56, p<0.001) values were higher in the study group.

Bleeding profile indicators, such as prothrombin time (PT) (10.12 [9.02–11.64] vs. 10.35 [9.31–12.80], p=0.506) and activated partial thromboplastine time (aPTT) (28.73±2.22 vs. 34.62±20.79, p=0.626) values were comparable in both groups. Mean values of procalcitonin, D-dimer, fibrinogen, and hsTnI, which were not tested for the control group, were 1.11 (0.02–100.00), 0.85±1.17, 440.44±137.42, and 189.40 (0.80–2940.00) respectively, in the study group. Acute cardiac injury (hsTnI >17.5 pg/mL) was observed 36.1% of the study group (55 patients).

Heart rate was higher in the study group than the control group (76.37±11.76 vs. 83.40±14.52, p<0.001). While QT and QRS values were similar in both groups (p>0.05 for each); QTc (392.29±24.35 vs. 407.45±28.84, p<0.001), Tpe (64.87±9.52 vs. 77.97±16.10, p<0.001), Tpe/QT (0.18±0.02 vs. 0.22±0.04, p<0.001), Tpe/QTc (0.16±0.02 vs. 0.19±0.03, p<0.001) values were higher in the study group than the control group.

Moreover, weak positive correlations between hs-troponin I and QT (rho=0.218, p=0.008), and QTc (rho=0.308, p<0.001), and strong positive correlations between hs-troponin I and Tpe (rho=0.646, p<0.001), Tpe/QT (rho=0.571, p<0.001), and Tpe/QTc (rho=0.608, p<0.001) were observed (Table 2, Fig. 1a–e).

DISCUSSION

Our study results can be summarized as follows: 1) Heart rate, QTc, Tpe, Tpe/QT, and Tpe/QTc values were higher in hospitalized patients with COVID-19 than the control grup, 2) QT, QTc, Tpe, Tpe/QT, and Tpe/QTc values correlated positively with hsTnI levels.

In patients with COVID-19, four mechanisms can be emphasized in the pathophysiology of the development of malignant ventricular arrhythmia: 1) Acute cardiac injury, 2) Hypokalemia, 3) Side effects of the drugs used in treatment, such as hydroxychloroquine and azithromycin, and 4) Proarrhythmic effects of vasopressors and inotropic agents used in supportive therapy. However, we will discuss only the first two causes since the arrhythmic risks of the patients at hospital admission were inves-

Table 1. Comparative analysis of demographic, biochemical, and electrocardiographic data of the study and the control groups

Variables	Control group (n=151)	Study group (n=152)	p
Age (years)	49.54±14.43	51.47±18.04	0.305
Sex (males, %)	90 (48.1)	97 (51.9)	0.451
DM, n (%)	25 (46.3)	29 (53.7)	0.566
HT, n (%)	52 (55.3)	42 (44.7)	0.200
CAD, n (%)	24 (39.3)	37 (60.7)	0.067
COPD, n (%)	19 (59.4)	13 (40.6)	0.340
CKD, n (%)	20 (52.6)	18 (47.4)	0.845
Malignancy, n (%)	5 (38.5)	8 (61.5)	0.579
WBC (K/uL)	8.06±2.04	9.24±4.53	**0.004**
Neutrophile (K/uL)	5.33±1.92	6.56±4.06	**0.034**
Lymphocyte (K/uL)	2.12±0.85	1.81±0.43	0.137
Platelet (K/uL)	266.82±58.35	256.23±107.34	0.301
Glucose (mg/dL)	111.32±39.16	137.82±61.21	**<0.001**
Urea (mg/dL)	29.84±9.38	41.59±10.64	**0.001**
Creatinine (mg/dL)	0.85±0.21	1.07±0.65	**0.007**
Potassium (mmol/L)	4.37±0.46	4.31±0.50	0.645
AST (U/L)	23 (19.25–25.0)	24 (19.0–31.0)	0.130
ALT (U/L)	23.5 (17.75–26.75)	20 (13.0–32.0)	0.434
Total bilirubin (mg/dL)	0.44 (0.33–0.69)	0.56 (0.39–0.79)	0.335
Creatinekinase (U/L)	108.75 (78.48–139.01)	186.74 (131.18–242.29)	0.437
LDH (U/L)	173.33±16.95	249.23±125.75	0.144
C-reactive protein (mg/dL)	0.48±0.53	7.12±2.56	**<0.001**
Procalcitonin (ng/mL)	–	1.11 (0.02–100.00)	–
PT (sec)	10.12 (9.02–11.64)	10.35 (9.31–12.80)	0.506
aPTT (sec)	28.73±2.22	34.62±20.79	0.626
D-dimer (mg/L)	–	0.85±1.17	–
Fibrinojen (mg/dL)	–	440.44±137.42	–
Hs-troponin I (pg/mL)	–	189.40 (0.80–2940.00)	–
Heart rate (beat/min)	76.37±11.76	83.40±14.52	**<0.001**
QT (msec)	350.38±28.76	349.26±32.89	0.753
QTc (msec)	392.29±24.35	407.45±28.84	**<0.001**
Tpe (msec)	64.87±9.52	77.97±16.10	**<0.001**
QRS (msec)	91.89±11.20	91.05±14.31	0.571
Tpe/QT	0.18±0.02	0.22±0.04	**<0.001**
Tpe/QTc	0.16±0.02	0.19±0.03	**<0.001**

ALT: Alanine aminotransferase; aPTT: Activated partial thromboplastine time; AST: Aspartate aminotransferase; CAD: Coronary artery disease; CKD: Chronic kidney disease; COPD: Chronic obstructive pulmonary disease; DM: Diabetes mellitus; Hs-troponin I: High sensitive troponin I; HT: Hypertension; LDH: Lactate dehydrogenase; PT: Prothrombin time; WBC: White blood cell; Tpe: T peak-to-end

tigated in our study. Acute cardiac injury may occur due to 1) myocarditis caused by the direct effects of the viral pathogen or 2) relative ischemia (type 2 acute myocardial infarction [AMI]) (16). The causes of type 2 AMI in these patients may include hypoxia, hypotension, increased inflammatory status triggered by cytokine storm, angiotensin converting enzyme 2 (ACE2) receptor downregulation, and increased catecholamine discharge (2, 5, 17, 18). Both the increased inflammatory status observed during acute cardiac injury and atrial and/or ventricular fibrosis and scar tissue are likely to develop after acute cardiac injury may trigger the development of malignant ventricular arrhythmia. Recent studies have shown that in patients with AMI, life-threatening arrhythmias can only be reduced to less than 3% despite advances in current treatment (19, 20). Concordantly, in the retrospective cohort study of Si et al. (21), arrhythmia was reported in 44 of 170 COVID-19 patients with elevated cardiac

troponin I, of which 6 were VT or VF. In their study, Guo et al. (6) observed that incidence of malignant ventricular arrhythmia in COVID-19 patients with elevated troponin T levels is higher than COVID-19 patients with normal troponin T levels. On the other hand, increased wall thickness with diffuse biventricular hypokinesis and marked biventricular myocardial interstitial edema were demonstrated in cardiac magnetic resonance imaging (CMR) of SARS-CoV-2 myocarditis presented by Inciardi et al. (7) This extensive myocardial involvement can be a substrate for ventricular arrhythmias. In parallel, in a study conducted by Ucar et al. (22), ventricular repolarization dispersions were compared between the patients with acute myocarditis (AM) and the control grup. As a result of the study, while QT and QTc were similar in both groups; Tpe, Tpe/QT, and Tpe/QTc were higher in the patient group. Additively, the aforementioned parameters showed positive correlation with troponin I, as in our study. Similarly, Kucuk et al. (23) showed higher Tpe interval, Tpe/QT, and QT dispersion values in children with acute rheumatic carditis compared to the healthy controls. In addition, they hypothesized that higher Tpe and Tp-e/QT values can predict myocardial involvement in these patients.

Table 2. The correlations of hs-troponin I with ventricular repolarization parameters

	Hs-troponin I	p
QT	0.218*	**0.008**
QTc	0.308*	<**0.001**
Tpe	0.646*	<**0.001**
Tpe/QT	0.571*	<**0.001**
Tpe/QTc	0.608*	<**0.001**

Hs-troponin I; High sensitive troponin I; Tpe: T peak-to-end

As another cause of arrhythmia in patients with COVID-19, hypokalemia is expected to develop as a result of the Renin-Angiotensin System, which deteriorates due to the interaction between SARS-CoV-2 and ACE2 (24). In this context, Chen et al. (25) reported that 18% of the patients with COVID-19 had severe hypokalemia (plasma K^+<3 mmol/L) and 37% had hypokalemia (plasma K^+=3–3.5 mmol/L). Hypokalemia is associated with QT interval prolongation and increased risk of Torsades de pointes-associated polymorphic VT. In our study, although no significant difference in potassium levels was found between the study group and the control group, nonetheless, patients with COVID-19 should be followed up closely for potassium levels.

In a recent study published by Öztürk et al. (26), similar to our study, the ECG data of patients hospitalized with the diagnosis of COVID-19 than the control group. However, the study had a very small study population (51 patients with COVID-19 and 40 controls). As a result of the study, in accordance with our results, QTc, Tpe/QTc, and QT dispersion values were higher in the COVID-19 patient group than in the control group. Furthermore, it was shown that the heart rate correlated positively with troponin levels, but this time, unlike our results, ECG parameters did not correlate with troponin levels.

Our study has several limitations. First, this study was a single-center study comprising relatively small sample size. Another limitation was that NT-proBNP and IL-6 levels could not be evaluated because their kits were not available in our facility. Moreover, the relationship between ventricular repolarization parameters and the development of malignant ventricular arrhythmia or arrhythmic death was not followed up because the medications which can affect the ventricular repolarization parameters, such as hydroxychloroquine and azithromycin, were administered to the patients. In addition, some of the patients were administered positive inotropic agents. Lastly, the ECG measurements were obtained manually because of absence of relevant digital sofware in our hospital.

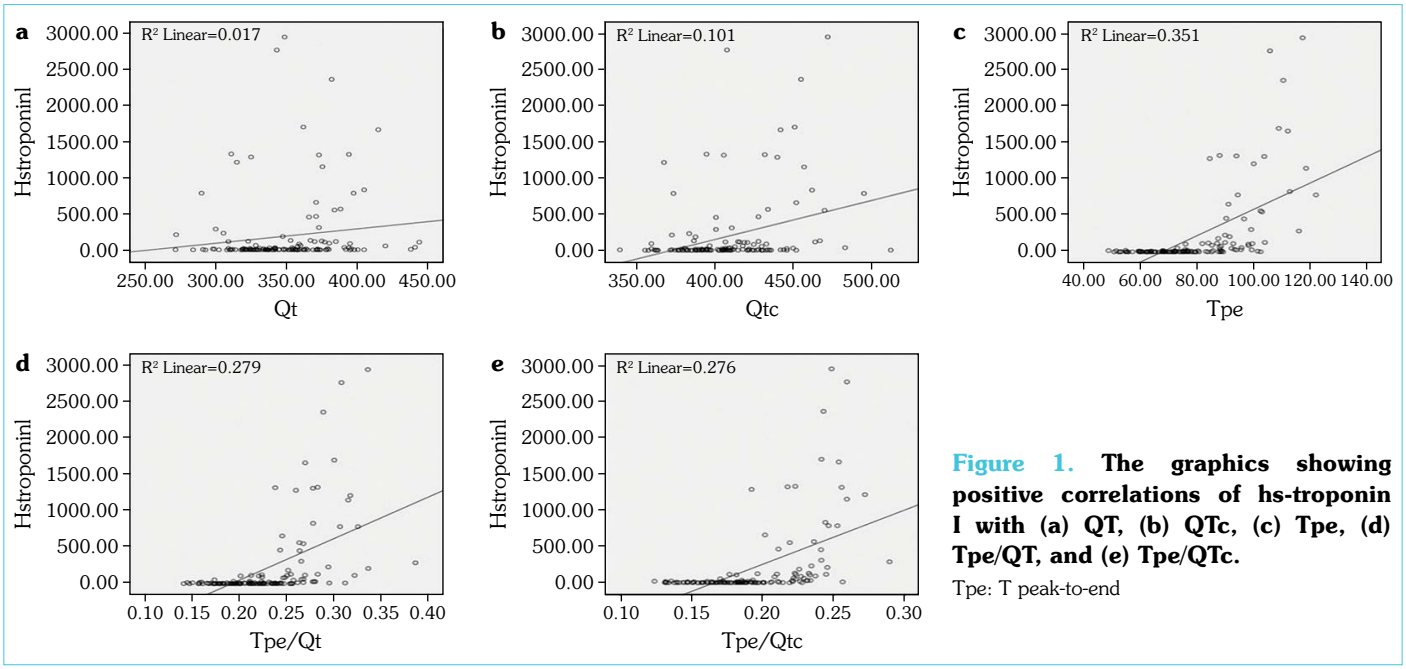

Figure 1. The graphics showing positive correlations of hs-troponin I with (a) QT, (b) QTc, (c) Tpe, (d) Tpe/QT, and (e) Tpe/QTc.

Tpe: T peak-to-end

CONCLUSION

The increased risk of arrhythmia in patients with COVID-19, especially those requiring ICU, has been demonstrated in previous studies. In our study, we showed that patients with COVID-19 have higher QTc, Tpe, Tpe/QT, and Tpe/QTc values, than the control group. These parameters also showed a positive correlation with hsTnI, a marker of cardiac injury. Therefore, although it may be say that close ECG monitoring can be beneficial to prevent arrhythmic events in patients with COVID 19, especially in the presence of acute cardiac injuries, larger scale studies are required to support our arguments.

Ethics Committee Approval: The Ahi Evran University Ethics Committee granted approval for this study (date: 24.06.2020, number: 2020-09/74).

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

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – SS, AK; Design – SS, LH; Supervision – SS, LH; Resource – None; Materials – None; Data Collection and/or Processing – AK, LH; Analysis and/or Interpretation – SS, AK; Literature Search – SS, AK; Writing – SS; Critical Reviews – SS, LH.

Conflict of Interest: The authors have no conflict of interest to declare.

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

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