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Acute ECG changes and post-COVID arrhythmia incidence in patients with acute COVID-19 infection

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ABSTRACT

Objective: During the COVID-19 pandemic, many patients have experienced cardiovascular complications, including a variety of arrhythmias. The aim of our study was to evaluate the acute electrocardiography (ECG) changes and post-COVID arrhythmia incidence in patients with acute COVID-19 infection.

Patients and Methods: One hundred hospitalized COVID-19 patients were consecutively included. Patients were divided into two groups according to their troponin levels. Thirty subjects were included as controls. All patients underwent daily 12-lead ECG during hospitalization and were followed up for at least 12 months, by performing ECG and ambulatory ECG monitoring and questioning their symptoms at 3-month intervals.

Results: Thirty-one patients had elevated high sensitive cardiac troponin I (hs-cTnI). These patients had significantly longer QT dispersion compared to COVID-19 patients with normal troponin levels and controls. Regardless of troponin elevation, COVID-19 patients had significantly longer Tp-e intervals and P wave (PW) durations compared to controls. During the follow-up period; palpitation, beta-blocker usage, and inappropriate sinus tachycardia were more common in the COVID-19 group with hs-cTnI than control group.

Conclusion: COVID-19 causes prolongation in PW durations, Tp-e intervals, and QT dispersion during acute infection, which may lead to arrhythmias in these patients. The higher incidence of inappropriate sinus tachycardia in COVID-19 patients with elevated troponin levels may be a sign of myocardial involvement.

Keywords: Post-COVID, Arrhythmia, Electrocardiography, Troponin

1. INTRODUCTION

Since the beginning of the coronavirus disease 2019 (COVID-19) pandemic, cardiovascular complications have emerged as the predominant extrapulmonary manifestations of COVID-19 infection. Cardiac involvement associated with acute COVID-19 manifests in various patterns, from asymptomatic elevations in cardiac biomarkers to serious conditions such as cardiogenic shock and sudden cardiac death [1,2].

The arrhythmia that develops in acute COVID-19 infection has a broad spectrum. Although, the exact cause of these arrhythmias has not been fully elucidated, direct cell damage by the virus, cardiomyocyte damage due to hyperactivation of the immune system, and direct arrhythmogenic effect of inflammatory cytokines are some of the underlying mechanisms [3]. On the other hand, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus uses the angiotensin converting enzyme-2 (ACE-2) receptor for entry into cells and down-regulates ACE-2 expression [4]. Downregulation of ACE-2, which converts the proinflammatory and prooxidant angiotensin II to angiotensin 1-7, creates a proinflammatory environment that can lead to arrhythmias [5].

Cardiac arrhythmias, usually presenting with palpitations, may endure for several months following acute COVID-19 infection [6]. The range of COVID-19-associated arrhythmias is diverse and likely due to different underlying pathomechanisms. Although, the relationship between COVID-19 and arrhythmia has been demonstrated in the long term after COVID-19, the underlying mechanism has still not been fully explained. Cardiac manifestations are frequently seen in post-COVID syndrome and a new entity has been named as post-COVID

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tachycardia syndrome [7,8]. However, their relationship with cardiac manifestations in acute infection is not clear.

The aim of this study was to evaluate acute electrocardiography (ECG) changes and post-COVID arrhythmia incidence in patients with acute COVID-19 infection.

2. PATIENTS and METHODS

Study population

One hundred consecutive hospitalized COVID-19 patients between March 24, 2020 and May 4, 2020 constituted our study population. And these patients were followed for 12 months. Thirty subjects with no symptoms or signs of active COVID-19 infection were included as a control group. Patients with reduced ejection fraction, left bundle branch blocks were excluded from the study. The usage of beta-blockers, calcium antagonists, or any other antiarrhythmic drugs was also an exclusion criterion of our study. The troponin levels of the patients were noted and patients were divided into two groups according to their troponin levels.

Electrocardiographic Measurements

The standard 12-lead ECG of all patients was obtained using a recorder (Schiller AT-2 plus, Switzerland) set at a 25 mm/sec paper speed and 1 mV/cm standardization. EPCalipers program (Version 2.4 (40) for Windows) was used for online analysis of scanned ECGs. ECGs of the patients were taken before starting the drug treatment. A single physician who was blinded to patient characteristics measured ECG parameters. Basal measurements of all patients such as PR intervals, QRS intervals, and RR intervals were recorded. A mean value of three readings was calculated for each lead and included in the analysis. Only recordings with more than eight leads that can be analyzed were included.

PW dispersion measurements: P wave (PW) duration was measured as between the initial and final deflection of the PW that crossed the isoelectric line. The maximum PW (Pmax.) duration was accepted as the longest PW, that is the longest atrial conduction time, while the minimum PW (Pmin.) duration was accepted as the shortest PW, which is the shortest atrial conduction time. PW dispersion was defined as the difference between Pmax. and Pmin.

QT and QT dispersion measurements: The QT interval was measured from the onset of the QRS complex to the end of the T wave. The end of the T wave was defined as the intersection of the T wave downslope with the isoelectric line when not followed by the U wave. If a U wave was present, this was not included in the measurement of the interval. Corrected QTd (QTcd) was defined as the difference between maximum QT (QT max.) and minimum QT (QT min.) interval and corrected for heart rate with Bazett's (QTc = QT$\sqrt{RR}$) [9], and Framingham formula (QTcFra=QT+0.154 (1−RR)) [10].

The Tp–e interval measurements: The Tp-e interval was measured from the peak of the T wave to the end of the T wave. Measurements of the Tp–e interval were performed by using the standard tangential method from V_5 preferentially [11]. If the T waves were isoelectric or of very low amplitude at V_5, V_2 derivation was used instead. The Tp–e/QT ratio was calculated from these measurements.

Arrhythmia assessment: The ECG was recorded using a Philips Digitrak XT Holter recorder (Philips Healthcare, Andover, Massachusetts) for 24 hours in three channels. During the 12-month follow-up, records were repeated every three months.

This study was approved by the Ethics Committee of the University of Health Sciences, Umraniye Research and Training Hospital (protocol code: 107 and date of approval 4 April 2020). The investigation conformed to the principles outlined in the Declaration of Helsinki. All participants gave written informed consent.

Statistical Analysis

All statistical tests were performed by a statistical analysis program (SPSS 21.0 for Windows, Chicago, IL). The distribution of data was tested using a one-sample Kolmogorov–Smirnov test. Categorical variables were defined as a percentage, and comparisons were made using the Chi-square test. Continuous data were expressed as mean ± standard deviation and Student's *t* test or ANOVA were used to compare the normally distributed continuous variables while the Mann-Whitney *U* test or Kruskal-Wallis test were used to compare the nonparametric continuous variables. Post hoc analyses were performed using Bonferroni test when an overall statistical significance was determined. A significance level was set at P<0.05.

Table I. *The general characteristics and laboratory parameters of the patients according to high sensitive cardiac troponin I level*

	COVID-19 patients with elevated troponin (n= 31)	COVID-19 patients with normal troponin (n= 69)	Controls (n= 30)	P value
Age (years)	60.9 ± 16.7	53.4 ± 16.3	55.6 ± 17.0	0.118
Male sex (n –%)	20 (64.5%)	39 (56.5)	16 (53.3)	0.649
Hypertension (n–%)	13 (41.9)	18 (26.1)	12(40)	0.195
Diabetes mellitus (n–%)	8 (25.8)	13 (18.8)	6 (20)	0.724
Coronary heart disease (n –%)	9 (29)	15 (21.7)	8 (26.7)	0.704
Heart failure (n –%)	4 (12.9)	3 (4.3)	5 (16.7)	0.108
COPD (n –%)	6 (19.4)	5 (7.2)	4 (13.3)	0.202
hs-cTnI (ng/mL)	0.061 ± 0.049 *,+	0.007 ± 0.018	0.012 ± 0.025	**<0.001**
hs-CRP (mg/L)	13.8 ± 8.7 *,+	7.2 ± 7.8 *	0.5 ± 0.9	**<0.001**
Lymphocyte (/µl)	1465 ± 1022 *	1553 ± 778 *	2463 ± 886	**<0.001**
D-dimer (µg/l)	1467 ± 1699 *,+	890 ± 873*	260 ± 188	**<0.001**

COPD: Chronic obstructive pulmonary disease, hs-cTnI: high sensitive cardiac troponin , hs-CRP: High sensitive C reactive protein, ***PostHoc analysis****: * denotes statistical significance versus control group, + denotes statistical significance versus patients with normal troponin levels*

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3. RESULTS

One hundred patients (mean age: 55 ±16 years, 59 male) with acute COVID-19 infection were consecutively included in the study. Thirty-one patients (31%) had elevated cardiac troponin. The general characteristics and laboratory parameters of the patients according to troponin levels are shown in Table I. Although, there were not any significant differences in the general characteristics of the patients, the COVID-19 patients had significantly higher D-dimer and hs-CRP levels, and lower lymphocyte counts compared to controls, while those patients with high hs-cTnI had also significantly higher D-dimer and hs-CRP levels, compared to COVID-19 patients with normal troponin levels.

Table II. *Electrocardiographic parameters of the study population during acute COVID-19 infection*

	COVID-19 patients with elevated troponin (n= 31)	COVID-19 patients with normal troponin (n= 69)	Controls (n= 30)	P value
AF (n–%)	2 (6.5)	3 (4.3)	1 (3.3)	0.835
RBBB (n%)	4 (12.9)	9 (13)	3 (10)	0.231
ST segment depression (n–%)	4 (12.9)	12 ((17.4)	3 (10)	0.741
T wave changes (n–%)	7 (22.6)	22 (31.9)	3 (10)	0.064
PVC (n–%)	3 (9.7)	3 (4.3)	1 (3.3)	0.469
PAC (n–%)	1 (3.2)	2 (2.9)	1 (3.3)	0.990
PVC ablation (n–%)	0	0	0	
SVT ablation (n–%)	0	0	0	

AF: Atrial fibrillation, RBBB: Right bundle branch block, PVC: Premature ventricular contraction, PAC: Premature atrial contraction, SVT: Supraventricular tachycardia

The conventional electrocardiographic parameters are listed in Table II. There were no significant differences in frequencies of atrial fibrillation, right bundle branch block, ST segment, and T wave changes among patients with different troponin levels.

The arrhythmogenic electrocardiographic parameters are listed in Table III. The COVID-19 patients had significantly higher heart rates, PW max., PW min., QTc max., and Tp-e intervals compared to controls. There were no significant differences in the PW dispersion among groups. The COVID-19 patients with high hs-cTnI had significantly higher QTc dispersion than the COVID-19 patients with normal hs-cTnI and the control group.

During the follow-up period; palpitation, beta-blocker usage, and inappropriate sinus tachycardia were more common in the COVID-19 patients with hs-cTnI than the control group (Table IV). The COVID-19 patients with elevated hs-cTnI had higher frequencies of ventricular extrasystole and three patients had premature ventricular contraction (PVC) ablation procedure at this period. There were no significant differences in frequencies of AF and PAC among patients. Only one COVID-19 patient had an SVT ablation and there was no difference among the groups.

Table III. *The arrhythmogenic electrocardiographic parameters during acute COVID-19 infection*

	COVID-19 patients with elevated troponin (n= 31)	COVID-19 patients with normal troponin (n= 69)	Controls (n= 30)	P value
Heart rate (beats/min)	83 ± 3*	84 ± 8*	76 ± 11	**0.011**
QRS duration (msec)	94 ± 17	94 ± 20	93 ± 24	0.983
PR interval (msec)	154 ± 25	157 ± 29	156 ± 28	0.832
PW max. (msec)	113 ± 16*	112 ± 14*	96 ± 16	**<0.001**
PW min. (msec)	75 ± 13*	76 ± 11*	65 ± 10	**<0.001**
PW dispersion (msec)	38 ± 10	36 ± 12	32 ± 13	0.189
QT max. (msec)	389 ± 43	385 ± 35	381 ± 31	0.688
QT min. (msec)	344 ± 44	349 ± 32	347 ± 29	0.811
QT disp (msec)	45 ± 15*,+	36 ± 16	34 ± 18	**0.015**
QTc max. (Bazett) (msec)	451 ± 34*	452 ± 37*	420 ± 33	**<0.001**
QTc min. (Bazett) (msec)	398 ±37	409 ± 33*	382 ± 34	**0.002**
QTc disp. (Bazett) (msec)	52 ±16*,+	43 ± 20	38 ± 19	**0.012**
QTc max. (Framingham) (msec)	428 ±33*	425 ± 28.5*	407 ± 27	**0.007**
QTc min. (Framingham) (msec)	382 ±35	388 ± 25*	372 ± 28	**0.035**
QTc disp. (Framingham) (msec)	45 ±14*+	37 ±17	35 ± 17	**0.037**
Tp-e interval (msec)	81 ± 16*	78 ± 16*	69 ± 11	**0.006**
Tp-e / QTc ratio	0.17 ±0.3	0.17 ±0.3	0.16 ± 0.2	0.286

*PW: P wave, QTc disp: corrected QT dispersion, PostHoc analysis: * denotes statistical significance versus control group, + denotes statistical significance versus patients with normal troponin levels*

Table IV. *The incidence and types of arrhythmias in patients during the one-year follow-up period*

	COVID-19 patients with elevated troponin (n= 31)	COVID-19 patients with normal troponin (n= 69)	Controls (n= 30)	P value
Palpitations (n–%)	12 (38.7) *	18 (26.1)	3 (10)	**0.036**
Beta blocker usage (n–%)	13 (41.9) *	8 (11.6)	2 (6.7)	**<0.001**
IST (n–%)	7 (22.6) *	4 (5.8)	0	**0.003**
AF (n–%)	1(3.2)	2 (2.9)	1 (3.3)	0.992
PVC (n–%)	5 (16.1) *	1 (1.4)	1 (3.3)	**0.025**
PAC (n–%)	1(3.2)	3 (4.3)	1 (3.3)	0.951
PVC ablation (n–%)	3 (9.6) *	0	0	**0.007**
SVT ablation (n–%)	0	1 (1.4)	0	0.641

*IST: Inappropriate sinus tachycardia, AF: Atrial fibrillation, PVC: Premature ventricular contraction, PAC: Premature atrial contraction, SVT: Supraventricular tachycardia, **PostHoc analysis**: * denotes statistical significance versus control group*

4. DISCUSSION

In our study, we evaluated the acute ECG changes and post-COVID arrhythmia incidence in patients with acute COVID-19 infection We found that during acute infection; all COVID-19 patients had significantly higher PWmax., PWmin., QTc max., and Tp-e intervals compared to controls. The COVID-19 patients with high hs-cTnI had significantly higher QTc dispersion, which is regarded as a marker for succeptibilty for development of cardiac arrhythmias. In parallel, we noted that during the follow-up, inappropriate sinus tachycardia and PVC were more frequent in the COVID-19 patients with elevated troponin levels.

The frequency of arrhythmias in acute COVID-19 infection is observed at a rate of 20% [12]. Although, ECG differences vary according to the clinical status of COVID-19 patients, AF, and all arrhythmias occur more frequently in intensive care patients [13]. In our study, heart rate, PW durations, corrected QT interval and Tp-e interval were significantly higher in the group with high hs-cTnI and there was no difference between the groups in terms of the frequency of AF and PVC. Elevation of troponin, an indirect indicator of myocardial damage, has been observed in approximately 20% of COVID-19 patients in the acute period. High troponin levels were associated with higher in-hospital mortality [14].

We showed at 1-year follow-up, palpitations, beta-blocker usage, and PVC frequency were more common in the troponin-positive group than in the troponin-negative group and the control group. Cardiovascular symptoms may emerge after the acute infection and endure for an extended period [15]. Several large studies have reported persistent cardiac arrhythmias after COVID-19 [16]. The overall prevalence of cardiac arrhythmias ranges from 10 to 20% in post-COVID [17]. Similar to our study, in a study in which COVID-19 patients were followed for 6 months, 154 patients (9.3%) had palpitations [18]. In addition, another study reported that 13.7% of patients had a consistently high heart rate until about 4 months after acute infection [19]. Also, in a study in which patients with a history of hospitalization due to COVID-19 were followed up for 3 months, cardiac arrhythmia was found to be 27%. The most common anomaly detected is PVC, seen in 18% of patients. In our study, PVC was observed in 6 people in the entire COVID-19 patient group during acute infection, while this number increased in the high troponin group and decreased in the troponin-negative group in the follow-up. PVC ablation was applied to 3 patients from the troponin-positive group [20].

We also showed inappropriate sinus tachycardia (IST) in 11% of the entire COVID-19 patients during the follow-up period. IST, a rather common observation in post-COVID patients, is defined by a sinus heart rate of >100 bpm at rest (with an unexplained mean 24-h heart rate> 90 bpm) and is associated with distressing symptoms of palpitations [21]. In a study on post-COVID patients, approximately 20% of them met the criteria of the IST [22]. In our study, IST was significantly more common in the high troponin group.

Atrial fibrillation (AF) occurs in hospitalized acute COVID-19 patients with severe disease progression, often accompanied by hyperinflammatory laboratory indicators [23]. But there are publications stating that the incidence of AF is 1.7 times higher in 6-month follow-up after acute infection in patients who are not hospitalized due to COVID-19 [24]. However, in our study, there was no difference in the incidence of AF between the groups at one-year follow-up.

In a cardiovascular magnetic resonance (CMR) study of 26 people who recovered but still experienced cardiac symptoms, 58% of patients had myocardial edema and scar tissue [25]. In another CMR study, which included 100 patients 3 months after acute infection, it was reported that fibrosis in 78% of the patients and myocardial inflammation in 60% of patients persisted [26]. Fibrosis can potentially disrupt the spread of electrical signals and create pathways for re-entry circuits, thereby playing a role in the development of arrhythmias. The connection between fibrosis and arrhythmias has been observed across various cardiac pathological conditions, with CMR being used to evaluate fibrotic remodeling in nearly all cases [27].

In the long term, the frequency of arrhythmias in the troponin-positive group may have been caused by fibrosis or prolonged myocardial inflammation due to cardiac involvement. There is a need for studies that integrate clinical, laboratory and imaging methods to predict the arrhythmia potential of long-term post – COVID-19 patients.

Limitations

The first limitation was a small sample size, and the study was a single-center study. The absence of previous ambulatory ECG monitoring recordings of the patients was another limitation of the study. Coronary artery disease diagnosis was based on patient-reported medical history in our study which was another limitation of our study. Finally, coronary angiography was not performed in the group with elevated troponin, so ischemia, a possible source of arrhythmia, may have been underdiagnosed.

Conclusions

COVID-19 infection induces prolongation of PW durations, Tp-e intervals, and QT dispersion during the acute infection phase. Relatively increased QT dispersion especially in COVID-19 patients with high troponin levels may serve as an indicator of higher arrhythmia risk in this subgroup during acute infection or follow. The higher frequency of inappropriate sinus tachycardia in COVID-19 patients with elevated troponin levels may be a sign for cardiac involvement in these patients and may bear the potential for arrhythmias. These findings emphasize the importance of closely monitoring cardiac parameters in COVID-19 patients, particularly those with elevated troponin levels, to identify potential arrhythmias and implement appropriate management strategies for improved long-term outcomes. Further research is needed to elucidate the underlying mechanisms of these cardiac changes and their implications for patient care.

Compliance with the Ethical Standards

Ethics approval:. The study was approved by the Ethics Committee of the University of Health Sciences, Umraniye Research and Training Hospital (protocol code: 107 and date of approval 4 April 2020). The investigation conformed to the principles outlined in the Declaration of Helsinki. All participants gave written informed consent.

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Authors contributions: ZD : Concept, design, data collection and analysis, CI: Supervision, ZD and CI: Literature search and writing, CI: Critical review. Both authors critically revised the manuscript, approved the final version to be published, and agreed to be accountable for all aspects of the work.

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