

## ECG INTERPRETATION AMONG COVID-19 PATIENTS

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### Abstract

**Background:** The goal was to use surface electrocardiographic (ECG) indicators to detect COVID-19's malignant arrhythmic potential.

**Methodology:** In 51 COVID-19 patients and 40 control subjects, the ECG parameters PR, QT, QTc, QTd, TPe, and Tpe/QTc were assessed. COVID-19 patients had substantially higher mean QTc (410.8 24.3 msec vs. 394.6 20.3 msec,  $p=0.001$ ), Tpe/QTc (0.19 0.02 vs. 0.18 0.04,  $p=0.036$ ), and median QTd (47.52 vs. 46.5) values than the control group. Troponin levels were linked with heart rate ( $r=0.387$ ,  $p=0.006$ ) but not with ECG parameters ( $r=0.387$ ,  $p=0.006$ ).

**Conclusion:** In Covid19 patients, several ventricular arrhythmia surface ECG predictors, such as QTc, QTd, and Tpe/QTc, are elevated. Because drugs used in COVID-19 patients have the potential to impact these parameters.

**Keywords:** Malignant arrhythmia, COVID-19, QT prolongation ECG.

### Introduction

From Wuhan, Hubei Province, China, the Coronavirus 2019 disease (COVID-19) spread over the world, causing pandemic [1]. For persons with chronic conditions and the elderly, this disease has high mortality and morbidity rates [2]. Because we are still learning about COVID-19, the majority of scholarly studies focus on its epidemiological, demographical, and clinical consequences [3,4]. Myocardial injury, thromboembolic events, and deadly arrhythmias are among its cardiovascular consequences [5]. In fact, comparable viruses have arrhythmogenic potential, and the drugs used to treat COVID-19 have a higher risk of causing arrhythmia [6]. Several surface electrocardiographic (ECG) markers have been used to predict the risk of ventricular arrhythmias, including the QT interval and QT dispersion (QTd), T wave peak-to-end interval (Tp-e), and Tp-e/QTc ratio [7–9]. The goal of this cross-sectional study was to see how COVID-19 affected surface ECG arrhythmia parameters like the QT interval, QTd, Tp-e, and Tp-e/QTc ratio.

### Material and Methods

#### Participants and study design

The study included 51 adult patients hospitalized with COVID-19 in March and April 2020, as well as 40 participants who were age and gender matched.

The study was authorized by Van Yuzuncu Yil University's Local Medical Ethics Committee.

Polymerase chain reaction (PCR) and computed tomography were used to confirm the diagnosis of COVID-19 (CT).

The study excluded patients with a recent infectious condition, pregnancy, chronic kidney failure, chronic liver failure, known coronary artery disease, malignancies, or inflammatory diseases. Exclusion criteria included the use of beta blockers, calcium antagonists, or antiarrhythmic medicines, as well as the presence of atrial fibrillation, frequent ventricular premature beats, or bundle branch blockages. The 12-lead ECGs were taken at the time of admission, 3 hours after the first dosage of medicine, and on subsequent days (if QTc is normal, daily, if not, once every 3 days). The patients' admission ECG was compared to that of a healthy control group. After 30 minutes of rest, 12-lead ECG records (Nihon Kohden brand) were received at room temperature (20–24 °C). Two independent clinicians were blinded to the study design and clinical data and assessed all ECGs (filter range 0.5–150 Hz, AC filter 60 Hz, 25 mm/s, 10 mm/mV). All ECG records were scanned, the data was transferred to a personal computer to reduce probable inaccuracies, and 400 percent

magnification was achieved using Adobe Photoshop software. The participants whose ECGs showed a U wave were excluded from the research. The QT interval was estimated using the Bazett formula ( $QTc = QT(R-R \text{ interval})$ ) and measured from the beginning of the QRS complex to the conclusion of the T wave. The difference between the highest and minimum QT intervals in 12 leads was used to calculate the QT dispersion (QTd). The Tp-e interval is the time between a T wave's peak and its end. Precordial leads were used to determine the Tp-e interval [10,11]. The distance between the start of the P wave and the start of the Q wave was designated as the PR interval.

### Statistical Analysis

SPSS version 22.0 was used to analyze the data (SPSS 16.0; SPSS Inc., Chicago, IL). The Kolmogorov-Smirnov method

was used to determine whether the variables were normal. For the comparison of normally distributed variables, the Student t test was utilized, and the results were reported as mean standard deviation (SD). Mann-Whitney For the comparison of non-normally distributed variables, the U test was utilized, and the results were reported as median (min-max). The categorical variables were compared using the Chi-square test. The Spearman test was used to assess the correlations between variables. Statistical significance was defined as p.05 values.

### Results

Between the patients and the control group, baseline demographic factors such as age, sex, hypertension, and diabetes were not substantially different (Table 1).

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Table 1. General characteristics of the study groups.

Baseline characteristics	COVID 19 patients (n = 51)	Control group (n = 40)	p value
Age (years)	49.2 ± 16.7	47.9 ± 14.9	.390
Male/female	29/22	26/14	.431
HT	6 (11.8%)	4 (10.0%)	.789
DM	6 (11.7%)	3(7.5%)	.951
Smoking	10 (19.6%)	5 (12.5%)	.364
LVEF (%)	58.5 ± 5.4	60 ± 4.3	.210
Heart rate (bpm)	83.7 ± 17.3	82.5 ± 16.7	.741

DM: diabetes mellitus; HL: hyperlipidemia; HT: hypertension; LVEF: left ventricular ejection fraction.

In comparison to the control group, Covid19 patients had substantially higher mean QTc (410.8 24.3 msec vs. 394.6 20.3 msec, p.001), Tpe/QTc (0.19 0.02 vs. 0.18 0.04, p =.036), and median QTd (vs. 46.5) values (Table 2).

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Table 2. Electrocardiographic measurements of the study groups.

	COVID 19 patients (n = 51)	Control group (n = 40)	p value
PR, msec	150.4 ± 22.9	148.3 ± 19.1	.635
QT, msec	352.5 ± 31.5	342.3 ± 36.5	.153
QTc, msec	410.4 ± 24.5	394.6 ± 20.3	<.001
QTd, msec	47.52 ± 1.7	46.15 ± 1.5	<.001
	48 (40-51)	46.5 (41-48)	
Tpe, msec	81.6 ± 9.4	76.6 ± 9.7	.156
	80 (60-100)	80 (60-90)	
Tpe/QTc	0.19 ± 0.02	0.18 ± 0.04	.036

Troponin levels were shown to have a significant relationship with heart rate ( $r = 0.387$ ,  $p = .006$ ). However, no significant relationships were found between ECG characteristics and troponin levels, as well as other clinical factors such as age. During follow-up, three individuals died. Table 3 shows the ECG values of deceased patients.

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Table 3. ECG values of died patients.

	PR msec	QT msec	QTc msec	QTd msec	Tpe msec	Tpe/QTc
Patient 1th	120	340,00	421,00	46	98,00	0,23
Patient 2nd	100	290,00	419,00	50	72,00	0,17
Patient 3rd	170	420,00	430,00	51	80,00	0,19

## Discussion

We discovered that possible ECG arrhythmia indicators QTc, QTd, and Tpe/QTc were significantly elevated in Covid19 patients in our investigation. This discovery could have significant ramifications for the treatment of Covid19-related arrhythmias. COVID-19 has been linked to myocardial damage and life-threatening arrhythmias [12]. In a recent research from Wuhan, China [13], malignant arrhythmias were found in 16.7% of COVID-19 hospitalized patients and 44.4 percent of COVID-19 critical care patients. Several 12-lead ECG characteristics have been proposed as possible markers for malignant arrhythmia. A prolonged QT interval, which is dependent on ventricular repolarization fluctuation, is one of the risk factors that can enhance malignant arrhythmia [14]. QTd is also useful for predicting ventricular repolarization problems and ventricular arrhythmias [15]. Shocking cardiac mortality induced by cardiac arrhythmias has been linked to increased QT and QTd values [9,14]. The Tp-e interval and Tp-e/QT ratio are now widely accepted as primary markers of ventricular repolarization and dispersion [16]. The Tp-e interval corresponds to epicardium and myocardial repolarization, however some cells in the subendothelial tissue are vulnerable to early depolarization, which can cause arrhythmia. As a result of aberrant activation of these cells, malignant arrhythmias and cardiac death can occur [17,18]. Despite changes in heart rate, these relatively recent measures of ventricular repolarization stay constant. As a result, an elevated Tp-e/QT ratio has been linked to arrhythmogenic events [19]. The difference in ECG (QTc, QTd, and Tpe/QTc) between the control and patient groups in our investigation suggests that the virus may impact ventricular repolarization as well. This theory is strengthened by the fact that the control group is identical to the sick group in terms of age and concomitant disorders. Troponin levels beyond a certain threshold were linked to a bad prognosis. Troponin levels above a certain threshold have been linked to an increased incidence of arrhythmia in Covid19 patients [5,13,20]. However, no correlation was found between the studied ECG parameters and troponin in our research. According to studies, the elderly with comorbidities, as well as the male population, are disproportionately affected [21]. Two of the three patients who died in our study also had hypertension and diabetes mellitus. They were 69 and 74 years old, respectively. The three guys who died were all men. When

the QTC values of deceased patients were evaluated, they were greater than those of the control group, but not to the point of causing malignant arrhythmia. Our patients did not die of arrhythmia; instead, they died of respiratory failure. It is unknown whether they would develop arrhythmias if they lived longer.

## Limitations

One of the most significant drawbacks of this study is the limited sample size. Patients' arrhythmia potential was assessed via ECG, thus it's uncertain if long-term rhythm data in this patient group would have yielded similar results. Many factors influence ventricular repolarization, including coronary artery disease and hormones [22]. We lack sufficient evidence to resolve this issue because we did not perform coronary angiography or assess hormone levels in our patients.

**Conclusion** In Covid19 patients, several ventricular arrhythmia surface ECG predictors, such as QTc, QTd, and Tpe/QTc, are elevated. This situation can induce the condition to develop malignant arrhythmias. Because drugs used in COVID-19 patients have the potential to impact these parameters, paying attention to these ECG markers and taking measures sooner could help to reduce the disease's arrhythmia-related mortality. For potential impact of this discovery on Covid19-related arrhythmias, larger and longer-term follow-up investigations are required.

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