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Characteristic Electrocardiography Findings of COVID-19 Patients

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Abstract
Background: Determining a relationship between coronavirus disease 2019 (COVID-19) and the ECG findings of the patients with this disease can assist in early diagnosis and patient management based on these findings. This study aimed to investigate whether COVID-19 patients had characteristic ECG findings in the acute period.
Methods: A total of 124 patients were divided into two groups as those diagnosed with COVID-19 and controls. The ECGs of these patients were evaluated in terms of rate, rhythm, presence of ST changes, PR interval, QRS width, QTc and QT interval, and presence of right and left bundle branch blocks.
Results: On the ECG, the median heart rate of the COVID-19 patients was 104/min (IQR: 99–114), and there was a significant difference compared to the control group ($P < 0.001$). The median PR interval was 157/ms, the QRS width was 86 ± 9/ms in the COVID-19 patients, with no significant difference compared to the controls ($P = 0.161$ and $P = 0.631$, respectively). The median QT interval of the COVID-19 patients was normal (400/ms), but a significant difference was detected compared to the controls ($P = 0.005$). The QTc, ST change, AF, and presence of right and left bundle branch blocks were not significantly different between the two groups.
Conclusion: Considering the importance of ECG findings in order to diagnose COVID-19 disease early, we can state that sinus tachycardia is very common in COVID-19 patients, but there is no characteristic ECG finding for COVID-19, including tachycardia.
Keywords: COVID-19, Electrocardiography, Myocardial injury, Pandemic, Sinus tachycardia
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Introduction

The coronavirus disease 2019 (COVID-19) is a clinical disease caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), which emerged in Wuhan, China and spread across the world in a very short time.[1] Although COVID-19 generally affects the lungs, it is also reported as often causing acute respiratory distress and pneumonia and has cardiac side effects,[2] such as myocarditis, acute coronary syndrome, decompensated heart failure, sudden cardiac death, and arrhythmia. In addition, associated with these side effects, COVID-19 patients present with findings on electrocardiography (ECG).[3-6]

It is interesting to determine whether COVID-19 has effects on the heart during the acute period of the disease and the type of findings it presents on ECG. The cardiac effects of COVID-19 are not known yet; therefore, an evaluation of ECG changes in these patients can help detect cardiac adversities quickly and early. Determining a relationship between COVID-19 and the ECG findings of these patients would be beneficial for early diagnosis of the disease and patient management based on these findings. In this study, we aimed to investigate whether COVID-19 patients had characteristic ECG findings in

the acute phase.

Materials and Methods

The study was initiated after obtaining the ethics committee approval (number: 06/09) from the Ethics Committee of Atatürk University. In the G*Power analysis, a total of 116 patients were calculated as the sample size, including at least 58 patients for each group at 80% power and 95% confidence level. The study included a total of 124 patients who presented to the emergency service of the university hospital. Of these patients, 62 who were positive for COVID-19 were evaluated as the COVID-19 group and 62 without COVID-19 as the control group. The patients included in the COVID-19 group consisted of those who presented to the emergency service with related symptoms, such as fever, headache, joint pain, shortness of breath, and sore throat. After the patients were evaluated by a general systemic examination, a chest computer tomography (CT) was undertaken. Based on the chest CT findings, the presence of ground glass opacity (GGO), paving-stone appearance, and consolidation areas were considered significant for the diagnosis of COVID-19. Then, the ECGs of the patients were taken and recorded before starting any treatment.

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Nasopharyngeal swaps were taken from the patients and the diagnosis of COVID-19 was made using the reverse-transcriptase polymerase chain reaction (RT-PCR) test. The control group consisted of patients who presented to the emergency service with any symptom other than those related to COVID-19, showed no evidence of COVID-19 on the chest CT, had a negative reverse transcription polymerase chain reaction (RT-PCR) test and had no history of cardiac disease.

Patients with trauma, those under 18 years of age, those with coronary artery disease, and pregnant women were excluded from the study. The patients' demographic characteristics, presenting complaints, CT findings, PCR results, vital signs, treatments they had received, and chronic diseases, if any, were recorded. Then, the recorded ECGs were assessed by the cardiologist in terms of heart rate, rhythm, presence of ST changes, PR interval, QRS width, QT interval, corrected QT (QTc), and presence of right bundle branch block (RBBB) and left bundle branch block (LBBB).

The number of beats per minute (bpm) was calculated by dividing the number of large squares between two R waves (R-R interval = one beat) by 300. In ECG, every 1 mm is 0.04 seconds and every 5 mm is 0.2 seconds in the horizontal plane. The PR interval was obtained by measuring the time from the onset of the P wave and the onset of the QRS complex (the beginning of the R wave was taken when the Q wave was not observed). The duration of the QRS complex was calculated by measuring the time between the onset of the Q wave and the termination of the S wave. The QT interval was determined by measuring the time from the beginning of the QRS complex to the end of the T wave. The QTc was calculated using the Bazett formula ($QTc = QT/\sqrt{RR}$) on patients with heart rates between 60/min and 100/min and if the heart rate was 100 and above the Framingham formula ($QTc = QT + 0.154 (1-RR)$) was used for QTc calculating. In ECG, a normal P wave before each QRS complex and a regular rhythm at 60–100 bpm was interpreted as normal sinus rhythm. In ECGs with normal sinus rhythm, the P waves were positive in lead II and negative in lead aVR. A heart rate below 60 bpm was interpreted as sinus bradycardia, and a heart rate above 100 bpm as sinus tachycardia. Using a 12-lead or single-channel ECG of 30 seconds or more, the records with no noticeable and repetitive P waves and irregular R-R intervals (in cases where AV conduction was not impaired) were evaluated as atrial fibrillation. The normal ranges for the PR interval, QRS width, QT interval and QTc interval in male and female were accepted as 120–200 ms, 70–110 ms, and QT distance 350–440 ms, 350–440 ms and 350–460 ms respectively. In accordance with the diagnostic criteria of LBBB, the QRS duration being 0.12 seconds or more, appearance of wide, notched or distorted R waves in I, aVL and V5-V6, presence of secondary ST-T wave disturbances in I, aVL and V5-V6, absence of Q waves in I and V5-V6, and the time for the R waves to reach the peak (intrinsicoid deflection time)

being 0.06 s or more were evaluated as ECG indicators of LBBB. RBBB was identified based on the QRS duration being 0.12 seconds or longer, presence of a second R' wave in the right precordial derivations and the last R' wave being greater than the initial R, and secondary ST-T-wave disturbances in the right precordial derivations. These findings were recorded for each patient, and analyzed and interpreted using a statistics program.

Statistical Analysis

Analyses were undertaken using IBM SPSS v. 20. Data were presented as mean, standard deviation, median, minimum, maximum, percentage, and numbers or medians [interquartile range (IQR percentiles 25 and 75)]. Normal distribution of continuous variables was checked with the Shapiro-Wilk W-test when the sample size was < 50, and the Kolmogorov-Smirnov test when the sample size was ≥50. In addition, normal distribution of continuous variables was assessed using graphical methods. In the comparison between two independent groups, independent samples *t* test was conducted when the distribution was normal, and Mann-Whitney U test was used when data were not normally distributed. In 2×2 comparisons between categorical variables, the Pearson chi-square test was performed if expected value was > 5, the chi-square Yates test if 3–5, and Fisher's exact test is < 3. For comparisons greater than 2x2 between categorical variables, the Pearson chi-square test was used when the expected value was > 5 and the Fisher-Freeman-Halton test if it was < 5. A multivariable model was created between medication use and significant variables. The statistical significance level was taken at $P < 0.05$.

Results

The sample consisted of 124 patients with 21 males and 41 females in the COVID-19 group, and 29 males and 33 females in the control group (Table 1). The median age of the patients was 58 (IQR: 39-71) years in the COVID-19 group and 60 (IQR: 45–70) years in the control group (Table 2).

When the patients in the COVID-19 group were evaluated according to their presenting complaints, 31 (50%) had fever, 15 (24.2%) had a sore throat, 14 (22.6%) had shortness of breath, and 2 (3.2%) had loss of smell. In the control group, 7 (11.3%) patients had headache, 10 (16.19%) had abdominal pain, 12 (19.4%) had nausea and vomiting, 4 (6.5%) had back pain, 4 (6.5%) had dizziness, and 25 (40.3%) patients had other symptoms not associated with COVID-19 (Table 1). While all the patients in the COVID-19 group (n = 62, 100%) had findings in favor of COVID-19 on chest CT, none of the patients in the control group had chest CT pathological findings (Table 1).

When the patients in the COVID-19 group were evaluated according to the history of chronic disease, 5 (8.1%) had chronic obstructive pulmonary disease, 4 (6.5%) had diabetes mellitus, and 1 (1.6%) had chronic

Table 1. Comparative Evaluation of the Demographic and Clinical Characteristics of the Study Groups

Characteristics		COVID-19 Group (n = 62, 100%)	Control Group (n = 62, 100%)	P*
Gender	Male	21 (33.9%)	29 (46.8%)	0.143
	Female	41 (66.1%)	33 (53.2%)	
Presenting complaint	Fever	31 (50%)	0 (0%)	—
	Sore throat	15 (24.2%)	0 (0%)	
	Shortness of breath	14 (22.6%)	0 (0%)	
	Loss of smell	2 (3.2%)	0 (0%)	
	Headache	0 (0%)	7 (11.3%)	
	Abdominal pain	0 (0%)	10 (16.1%)	
	Nausea-vomiting	0 (0%)	12 (19.4%)	
	Back pain	0 (0%)	4 (6.5%)	
	Dizziness	0 (0%)	4 (6.5%)	
	Other	0 (0%)	25 (40.3%)	
Abnormal tomography findings	Present	62 (100%)	0 (0%)	0.001
	Absent	0 (0%)	62 (100%)	
RT-PCR result	Positive	62 (100%)	0 (0%)	0.001
	Negative	0 (0%)	62 (100%)	
Chronic disease history	COPD	5 (8.1%)	0 (0%)	—
	None	52 (83.9%)	62 (100%)	
	DM	4 (6.5%)	0 (0%)	
	CRF	1 (1.6%)	0 (0%)	
Medication use	Present	18 (29%)	8 (12.9%)	0.027
	Absent	44 (71%)	54 (87.1%)	
Mortality	Discharged	60 (96.8%)	62 (100%)	0.496
	Died	2 (3.2%)	0 (0%)	

COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; CRF, chronic renal failure.
*Pearson chi-square.

Table 2. Age, Electrolyte Status and ECG Parameters of the Study Groups

Parameters	COVID-19 Group	Control Group	P	Mean Difference	95% Confidence İntervals (Lower-Upper)
Age	58 (IQR 39–71)	60 (IQR: 45–70)	0.546*	-1.984	-8.478–4.510
Rate/msn	104 (IQR 99–114)	84 (IQR: 76–95)	0.001*	17.581	12.772–22.390
PR interval/ms	157 (IQR 143–173)	169 (IQR: 142–181)	0.161*	-4.815	-12.644–3.014
QRS width/ms	86 ± 9	85 ± 7	0.631**	0.710	-2.207–3.627
QT interval/ms	398 (IQR 381–409)	411 (IQR: 397–420)	0.005*	-8.548	-15.764– -1.332
QTc interval/ms	Male 437 (IQR 429–450)	441(IQR 432–462)	0.085*	0.780	-6.351–8.447
	Female 451(IQR 442–461	452 (IQR 431–460)	0.466*	0.670	-6.353–8.450
Sodium (mmol/L)	140 (IQR 138–142)	140 (IQR 139–141)	0.704*	0.524	-1.058–0.542
Potassium (mmol/L)	4.1 (IQR 3.8–4.3)	4.2 (IQR 3.9–4.4)	0.290*	0.251	-0.183–0.048
Magnesium (mmol/L)	2 (IQR 1.8– 2.1)	2 (IQR 1.8–2.1)	0.727*	0.755	-0.059–0.043
Calcium (mg/dL)	9.8 (IQR 9.4–10.6)	9.8 (IQR 9.4–10.6)	0.906*	0.855	-0.206–0.248
Chlorine (mg/dL)	106 (IQR 104–109)	106 (IQR 105–109)	0.793*	0.807	-1.172–0.914

*Mann-Whitney U test; **Student's t test.

renal failure (Table 1). While 18 (29%) patients in the COVID-19 group were using medication, 44 (71%) did not use any medication. In the control group, medication use was found in 8 (12.9%) patients and absent in 54 (87.1%). The chronic medication use in the COVID-19 group consisted of levofloxacin in 1 (1.6%) patient, paracetamol in 3 (4.8%) patients, salmeterol in 4 (6.4%) patients, terbutaline in 1 (1.6%) patient, insulin glargine in 4 (6.4%) patients, sodium hydrogen carbonate in one (1.6%) patient, Fluoxetine in one (1.6%) patient, proton pump inhibitors in 2 (3.2%) patients and nimesulide in one (1.6%) patient. The medication use in the control

group consisted of proton pump inhibitors in three (4.8%) patients, levofloxacin in one (1.6%) patient, citalopram in one (1.6%) patient, paracetamol in two (3.2%) patients and dexketoprofen trometamol in one (1.6%) patient. The heart rate of medication use was higher in the COVID-19 patients and statistically significant compared to the control group ($P = 0.027$) (Table 1).

When the patients were evaluated according to electrolyte status, the median sodium level was 140 mmol/L (IQR138–142), the median potassium level was 4.1 mmol/L (IQR 3.8–4.3), the median magnesium level was 2 mmol/L (IQR 1.8–2.1), the median calcium level was 9.8 mg/dL (IQR 9.4–10.6) and the median chloride level was 106 mg/dL (IQR 104–109) in the COVID-19 group and the median sodium level was 140 mmol/L (IQR139-141), the median potassium level was 4.2 mmol/L (IQR 3.9–4.4), the median magnesium level was 2 mmol/L (IQR 1.8–2.1), the median calcium level was 9.8 mg/dL (IQR 9.4-10.6) and the median chloride level was 106 mg/dL (IQR 105-109) in the control group. There was no significant difference between the two groups ($P = 0.704$, $P = 0.290$, $P = 0.727$, $P = 0.906$ and $P = 0.793$, respectively) (Table 2).

When the ECGs of the patients were evaluated, the median heart rate was 104 bpm (IQR: 99–114) in the COVID-19 group and 84 bpm (IQR: 76–95) in the control group, with a significant difference between the two groups ($P = 0.001$). The heart rate of the COVID-19 group was higher. While the median PR interval of the patients in the COVID-19 group was 157/ms (IQR: 143–173), that of the patients in the control group was 169/ms (IQR: 142–181), and there was no significant difference between the two groups ($P = 0.161$). The mean QRS width was 86 ± 9/ms in the COVID-19 group and 85 ± 7/ms in the control group, and there was no significant difference between the two groups ($P = 0.631$). The median QT distance was 398/ms (IQR: 381–409) and 411/ms (IQR: 397–420) in the COVID-19 and control groups, respectively, with a significant difference between the two groups ($P = 0.005$). Although the QT interval was within normal limits in the control group, it was found to be longer than the patients with COVID-19. The median QTc distance in males was

437/ms (IQR: 429–450) and 441/ms (IQR:432–462) in the control group, and there was no significant difference between the two groups ($P = 0.085$). The median QTc distance in females was 451/ms (IQR: 442–461) and 452/ms (IQR: 431–460) in the control group, and there was no significant difference between the two groups ($P = 0.466$) (Table 2).

Evaluating the COVID-19 patients according to rhythm types, 40 (64.5%) had sinus tachycardia, 17 (27.4%) had normal sinus rhythm, and 5 (8.1%) had atrial fibrillation (AF). In the control group, 62 patients (100%) had sinus rhythm. When the COVID-19 patients were evaluated in terms of rhythm within their group, the rate of sinus tachycardia was significantly higher compared to other rhythms ($P = 0.001$) (Table 3).

In the COVID-19 group, 3 (4.8%) patients had ST segment changes, while no patient in the control group presented with these changes. There was no statistically significant difference in ST segment changes between the two groups and within the COVID-19 group ($P = 0.432$) (Table 3). While 2 (3.2%) COVID-19 patients had LBBB, there was no patient with LBBB in the control group, but no significant difference was detected between the two groups ($P = 0.496$). Similarly, 1 (1.6%) COVID-19 patient had RBBB, with no significant difference compared to the control group ($P = 1.0$) (Table 3).

When the rhythms were evaluated between medication using patients, 14 (22.5%) patients had sinus tachycardia, and 4 (6.4%) patients had normal sinus rhythm in the COVID-19 group. Three (4.8%) patients had sinus tachycardia, and 5 (8.2%) patients had normal sinus rhythm in the control group. There was no significant difference between the rhythms of the patients using drugs in both groups ($P = 0.078$). When the heart rate was evaluated between medication using patients, the median heart rate was 102/min (IQR 100-106) in the COVID-19 group and 86/min (IQR77-108) in the control group. There was no significant difference between the heart rate of the patients using drugs in both groups ($P = 0.231$) (Table 4).

All the patients were hospitalized in the COVID-19 group. Twenty-five patients (40.3%) were hospitalized

Table 3. ECG Evaluations of the Study Groups

ECG Evaluations		COVID-19 Group (n = 62, 100%)	Control Group (n = 62, 100%)	P*
Rhythm	Sinus tachycardia	40 (64.5%)	11 (17.7%)	0.001**
	Normal sinus	17 (27.4%)	48 (77.4%)	
	Atrial fibrillation	5 (8.1%)	3 (4.8%)	
ST-segment changes	Present	3 (4.8%)	0 (0%)	0.432
	Absent	159 (95.2%)	62 (100%)	
Right bundle branch block	Present	2 (3.2%)	0 (0%)	0.496
	Absent	60 (96.8%)	62 (100%)	
Left bundle branch block	Present	1 (1.6%)	0 (0%)	1.000
	Absent	61 (98.4%)	62 (100%)	

*Pearson chi-square; **Between sinus tachycardia and normal sinus rhythm.

Table 4. Effects of Using Medications on Rhythm and Heart Rate Between Groups

Medications		COVID-19 Group (%, Median (IQR))	Control Group (%, median (IQR))	P*
Medication using present	Sinus tachycardia	14 (22.5%)	3 (4.8%)	
	Normal sinus	4 (6.4%)	5 (8.2%)	0.078
	Atrial fibrillation	0 (0%)	0 (0%)	
Medication using absent	Sinus tachycardia	26 (41.9%)	8 (12.9%)	
	Normal sinus	13 (21%)	43 (69.3%)	0.002
	Atrial fibrillation	5 (8.2%)	3(4.8%)	
Medication using present	Rate	102 (IQR 100-106)	86 (IQR77-108)	0.231
Medication using absent	Rate	106 (IQR 99-114)	83 (IQR75-93)	0.001

*Multivariable test.

in the intensive care unit and thirty-seven patients (59.7%) were hospitalized in the clinics. In terms of in-hospital mortality, two (3.2%) patients in the COVID-19 group died at hospital while 96.8% were discharged after recovery. In the control group, all patients were discharged from the hospital with full recovery. There was no significant difference between the two groups (*P* = 0.496) (Table 1).

Discussion

At the point of writing, approximately nine months had passed since the beginning of the COVID-19 pandemic in the world, with the emergency services continuing to struggle against the disease. Cardiometabolic and many organ functions are reported to be affected by COVID-19 in most studies.[7] Acute cardiac injury is a cardiac complication seen in approximately 8%-12% of COVID-19 patients.[8] In some studies, ECG abnormalities, such as QT changes, atrioventricular block, tachycardia, and ST-segment elevation myocardial infarction (STEMI) have been reported in approximately 15% of COVID-19 patients.[9] In our study, we examined the heart rate, QT, PR and QRS intervals, rhythms, block presence, and ST-segment changes in patients with a diagnosis of COVID-19.

Considering that COVID-19 patients may have hypoxia and electrolyte disturbances, cardiac arrhythmias can be seen in their ECGs.[10] Some case reports have described arrhythmias in COVID-19 patients.[3] In a study conducted by Mccullough et al on COVID-19 patients, it was found that 5.6% of the patients had AF and 94.4% had normal sinus rhythm.[11] In our study, sinus tachycardia was observed in 64.5% of the COVID-19 patients, normal sinus rhythm in 27.4%, and AF in 8.1%. When these rates were compared with the control group, the ECG of the COVID-19 patients was significantly different in terms of sinus tachycardia. However, when the patients were evaluated together with their clinical complaints, fever was present in 50% of the patients while there was no complaint of fever in the control group, and therefore, we consider that the tachycardia seen in the COVID-19 patients might be due to fever. Although this rate was

statistically significantly higher in the COVID-19 group, we cannot consider it a characteristic ECG finding for this disease.

The QT interval has been presented as an issue that needs to be investigated in COVID-19 patients since it was reported that hydroxychloroquine or chloroquine drugs used in the treatment of COVID-19 can cause QT prolongation.[12] In addition, sepsis, hypokalemia and some medications can cause myocardial damage and QTc prolongation.[13] Therefore, due to the possibility of arrhythmia development, it is necessary to measure the QT intervals in patients who are planned to start COVID-19 treatment and to monitor these measurements during the treatment process.[8] In our study, we found that the both QT and QTc interval of the COVID-19 patients were shorter than the controls, but the values of the former were within the normal ranges for this parameter. It can be said that there are genetic factors underlying prolonged QTc in some patients.[14]

In a previous study, it was found that a patient with COVID-19 had wider QRS and atrial arrhythmia.[15] In our study, there was no difference in the QRS width between the evaluated COVID-19 patients and the control group. Therefore, we can state that the QRS width is not a definitive ECG finding in COVID-19 patients.

In some studies, STEMI has been reported in some COVID-19 patients.[16] In our study, STEMI was detected in 4.8% of the patients with COVID-19 while it was not observed in any of the patients in the control group. Since there was no significant difference between the two groups, it can be concluded that ST-segment elevation is not a characteristic ECG finding for COVID-19.

Potassium and calcium ions in the plasma are responsible for cardiac electrical activity. Disturbances in these electrolytes can cause ECG changes.[17] In case of changes in the concentrations of these ions, changes such as speed and rhythm can be seen on the ECG.[18] In our study, electrolyte levels were normal in both groups and electrolyte values had no effect on ECG findings.

A number of cardioactive drugs can cause changes in the ECG. Fluoroquinolones, proton pump inhibitors and some drugs may affect the QT distance at ECG. Smoking,

insomnia, alcohol, excessive caffeine consumption, stress, fever and some drugs can cause tachycardia.[19] In our study, no difference was found between the heart rate and rhythm of patients using drugs in both the COVID-19 group and the control group. We think that the drugs used did not affect the ECG data of the patients in our study group.

In a study conducted by Mccullough et al on COVID-19 cases, 7.8% of the patients had RBBB and 1.5% had LBBB.[11] In our study, LBBB was detected in 3.2% of the COVID-19 patients while RBBB was detected in only 1.6% of the patients in this group. When these rates were compared to the control group, it was observed that there was no difference in terms of the presence of either block. Therefore, we consider that neither RBBB nor LBBB is a characteristic finding for COVID-19. In the abovementioned study,[11] a prolonged PR interval was detected in 2.5% of the COVID-19 patients. In our study, when the PR interval of COVID-19 patients was compared with the controls, it was not found to be a specific parameter for COVID-19.

The first limitation of our study is that some patients in our study were using drugs that could affect ECG. Another limitation is the low number of patients included in our study.

In conclusion, considering the importance of ECG findings in the early diagnosis of COVID-19, we can state that sinus tachycardia is very common in COVID-19 patients, but there is no characteristic ECG finding for COVID-19, including tachycardia.

Authors' Contribution

AG contributed to study conception and design, interpretation of data, and drafting of the manuscript. ZU contributed to study conception, study design and critical revision.

Conflict of Interest Disclosures

The authors declare no conflicts of interest.

Ethical Statement

The study was approved by the Ataturk University Ethics Committee (No: 06/09).

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References

1. Cheng ZJ, Shan J. 2019 Novel coronavirus: where we are and what we know. Infection. 2020;48(2):155-63. doi: 10.1007/s15010-020-01401-y.
2. Fried JA, Ramasubbu K, Bhatt R, Topkara VK, Clerkin KJ, Horn E, et al. The variety of cardiovascular presentations of COVID-19. Circulation. 2020;141(23):1930-6. doi: 10.1161/circulationaha.120.047164.
3. Kochav SM, Coromilas E, Nalbandian A, Ranard LS, Gupta A, Chung MK, et al. Cardiac arrhythmias in COVID-19 infection. Circ Arrhythm Electrophysiol. 2020;13(6):e008719. doi: 10.1161/circep.120.008719 .
4. Lakkireddy DR, Chung MK, Gopinathannair R, Patton KK, Gluckman TJ, Turagam M, et al. Guidance for Cardiac Electrophysiology During the COVID-19 Pandemic from the Heart Rhythm Society COVID-19 Task Force; Electrophysiology Section of the American College of Cardiology; and the Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, American Heart Association. Circulation. 2020;141(21):e823-e31. doi: 10.1161/circulationaha.120.047063.
5. Akhmerov A, Marbán E. COVID-19 and the heart. Circ Res. 2020;126(10):1443-55. doi: 10.1161/circresaha.120.317055.
6. Clerkin KJ, Fried JA, Raikhelkar J, Sayer G, Griffin JM, Masoumi A, et al. COVID-19 and cardiovascular disease. Circulation. 2020;141(20):1648-55. doi: 10.1161/circulationaha.120.046941.
7. Inciardi RM, Lupi L, Zaccone G, Italia L, Raffo M, Tomasoni D, et al. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). JAMA Cardiol. 2020;5(7):819-24. doi: 10.1001/jamacardio.2020.1096.
8. Bansal M. Cardiovascular disease and COVID-19. Diabetes Metab Syndr. 2020;14(3):247-50. doi: 10.1016/j.dsx.2020.03.013.
9. Ma KL, Liu ZH, Cao CF, Liu MK, Liao J, Zou JB, et al. COVID-19 myocarditis and severity factors: an adult cohort study. medRxiv. 2020. doi: 10.1101/2020.03.19.20034124.
10. Lakkireddy DR, Chung MK, Gopinathannair R, Patton KK, Gluckman TJ, Turagam M, et al. Guidance for cardiac electrophysiology during the COVID-19 pandemic from the Heart Rhythm Society COVID-19 Task Force; Electrophysiology Section of the American College of Cardiology; and the Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, American Heart Association. Heart Rhythm. 2020;17(9):e233-e41. doi: 10.1016/j.hrthm.2020.03.028.
11. McCullough SA, Goyal P, Krishnan U, Choi JJ, Safford MM, Okin PM. Electrocardiographic findings in coronavirus disease-19: insights on mortality and underlying myocardial processes. J Card Fail. 2020;26(7):626-32. doi: 10.1016/j.cardfail.2020.06.005.
12. Wang M, Cao R, Zhang L, Yang X, Liu J, Xu M, et al. Remdesivir and chloroquine effectively inhibit the recently emerged novel coronavirus (2019-nCoV) in vitro. Cell Res. 2020;30(3):269-71. doi: 10.1038/s41422-020-0282-0.
13. Yang P, Kanki H, Drolet B, Yang T, Wei J, Viswanathan PC, et al. Allelic variants in long-QT disease genes in patients with drug-associated torsades de pointes. Circulation. 2002;105(16):1943-8. doi: 10.1161/01.cir.0000014448.19052.4c.
14. Vink AS, Neumann B, Lieve KVV, Sinner MF, Hofman N, El Kadi S, et al. Determination and Interpretation of the QT Interval. Circulation. 2018;138(21):2345-58. doi: 10.1161/circulationaha.118.033943.
15. He J, Wu B, Chen Y, Tang J, Liu Q, Zhou S, et al. Characteristic electrocardiographic manifestations in patients with COVID-19. Can J Cardiol. 2020;36(6):966.

e1-966.e4. doi: 10.1016/j.cjca.2020.03.028.

16. Reddy V, Reddy V, Mangat S, Shokr M, Kundumadam S, Laharwani H. Wide complex tachycardia in a COVID-19 patient: what is the mechanism? J Electrocardiol. 2020;60:200-2. doi: 10.1016/j.jelectrocard.2020.05.001.

17. Diercks DB, Shumaik GM, Harrigan RA, Brady WJ, Chan TC. Electrocardiographic manifestations: electrolyte abnormalities. J Emerg Med. 2004;27(2):153-60. doi: 10.1016/j.jemermed.2004.04.006.

18. Surawicz B. Electrolytes and the electrocardiogram. Am J Cardiol. 1963;12(5):656-62. doi: 10.1016/0002-9149(63)90255-8.

19. Romanò M. Electrocardiographic changes caused by drugs and electrolyte abnormalities. In: Text Atlas of Practical Electrocardiography. Milano: Springer; 2015. p. 217-21. doi: 10.1007/978-88-470-5741-8_15.