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Electrocardiographic findings in non-critical patients with coronavirus disease-2019

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Ethics Committee Approval
The study protocol was approved by Kanuni Sultan Suleyman Training and Research Hospital's Ethical Committee (Number: KAEK/2020.05.57)
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

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Abstract

Background/Aim: Cardiovascular involvement in patients with coronavirus disease 2019 (COVID-19) is closely related to the course of the disease; however, this issue has not been adequately studied in Turkey. Thus, we aimed to investigate the electrocardiography (ECG) findings in noncritical patients with COVID-19 in Turkey.

Methods: This retrospective cohort study was conducted on non-critical patients with COVID-19 with no history of any cardiac disease. The laboratory parameters and ECG findings of the patients at the time of admission were analyzed.

Results: In total, 100 patients with a mean age of 56.8 (16.7) years were included in the study, among which 54 were males. The rate of patients having at least one abnormal ECG finding, ST segment pathology, and elevated troponin level were 58%, 26%, and 48%, respectively. The respiratory rate and mean troponin level were higher and the mean lymphocyte count was lower in patients with ST segment pathology than in patients without. The respiratory rate, fibrinogen level, and incidence of T negativity and abnormal ECG findings were higher, and the lymphocyte count was lower in patients with elevated troponin levels than in patients with normal troponin levels ($P<0.05$ for all). Troponin level was significantly negatively correlated with lymphocyte count and significantly positively correlated with respiratory rate and C-reactive protein ($P=0.003$, $r = -0.298$; $P=0.031$, $r = 0.215$; and $P=0.02$, $r = 0.233$, respectively). In the receiver operating characteristic analysis, it was found that ST segment pathologies were more common in patients with a troponin level >0.03 mg/mL ($P<0.001$, area under the curve: 0.763, sensitivity: 61.5%, and specificity: 90.5%).

Conclusion: Cardiac involvement is very common in patients with COVID-19, and elevated cardiac troponin levels and pathological ECG findings are observed.

Keywords: Electrocardiography, Troponin, ST segment pathology, COVID-19

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Introduction

Coronavirus disease 2019 (COVID-19) is an infection that emerged as atypical cases of pneumonia in the Hubei province of China in December 2019 and later became a pandemic [1]. According to the reports of the World Health Organization, as of February 12, 2021, >107 million cases and >2 million deaths were reported worldwide, with a mortality rate of approximately 2.1% [2].

Although pulmonary findings constitute the basic clinical presentation of the disease in patients with COVID-19, cardiac involvement is also common and has an impact on mortality rate in this patient group. In patients presenting with cardiac involvement, the possibility of severe disease progression, need for intensive care, and mortality are higher [3-5]. The mechanisms affecting the occurrence of cardiac symptoms in COVID-19 are uncertain, and various pathophysiological mechanisms have been reported as explanations. According to one hypothesis, the virus can bind to the angiotensin converting enzyme-2 receptor, an aminopeptidase that is highly expressed in the heart and lungs, causing alterations in angiotensin converting enzyme-2 signaling pathways and myocardial damage [6, 7]. Another mechanism could be the occurrence of acute systemic inflammatory response and cytokine storm in severe cases, causing multiorgan failure and cardiac damage due to an increase in circulating proinflammatory cytokine levels [8, 9]. In addition, it is considered that hypoxia and systemic inflammation, which affect myocardial oxygen requirement, increase the risk of plaque rupture due to elevated coronary blood flow rate and that the presence of various electrolyte imbalances that facilitate the occurrence of arrhythmias might cause cardiac damage in this patient group [3, 10]. In addition to the effects of the disease itself, drugs such as chloroquine, hydroxychloroquine, and azithromycin, which are used for its treatment, are known to have various cardiac side effects.

In this study, we aimed to identify any pathological findings detected by electrocardiogram (ECG) analysis at admission before treatment in patients with COVID-19 and examine the relationship of these findings with the severity of the disease and its associated parameters.

Materials and methods

In this retrospective study, we examined patients who were hospitalized in isolation wards with the diagnosis of COVID-19 at a tertiary training and research hospital between January 4, 2020 and May 31, 2020. Individuals younger than 18 years of age and pregnant and breastfeeding women were not included in the study. In addition, patients with any cardiovascular diseases, history of arrhythmia, or anti arrhythmic drug use in the period before the diagnosis of COVID-19 or those who had previously undergone coronary imaging for any reason were excluded from the study. The data of 156 patients were obtained, but 56 were excluded because of the following criteria: Having cardiovascular disease (n = 42), using anti arrhythmic drugs (n = 9), and verbally expressing a history of arrhythmia despite not using anti arrhythmic drugs (n = 7). Demographic data, vital signs, laboratory analysis results, ECG images, and pulmonary imaging findings were obtained through the data stored in the online system. The demographic data of the patients (age, sex, complaints at presentation, and presence of comorbid diseases) and their vital signs at presentation (blood pressure, heart rate, percentage of oxygen saturation at room air, and respiratory rate per minute) were recorded. The results of complete blood count analysis and C-reactive protein (CRP), procalcitonin, cardiac troponin, creatine kinase (CK), ferritin, fibrinogen, D-dimer, and electrolyte levels were recorded from the laboratory tests performed at the time of admission. The patients were grouped as mild, moderate, and severe cases according to the severity of the disease. The clinical presentation was considered severe in the presence of at least one of the following conditions: Respiratory rate ≥30/min, mean oxygen saturation at room air ≤90%, >50% pneumonia involvement detected on imaging and severe respiratory distress symptoms. Patients with a respiratory rate of <24/min, a mean oxygen saturation at room air of >93%, and normal lung imaging were included in the mild disease group. Patients who did not match the criteria for any of the two groups were defined as those with moderate disease.

ECG data of the patients were obtained from the results of 12-lead ECG analysis performed at a speed of 25 mm/s and saved in the system. There were small squares of 1 × 1 mm and large squares of 5 × 5 mm on the electrocardiography sheet, and each small square was considered to represent 0.04 s. In the vertical plane, a 0.1-mV stimulus was adjusted to create a 1-mm deflection. The following parameters were calculated and recorded using ECG:

Heart rate: It was calculated with the formulas of the number of large squares between R-R as 300/R-R, or the number of small squares between R-R, as 1500/R-R. The reference range is between 60–100/min, and a heart rate of <60/min was defined as bradycardia, whereas HR >100/min was defined as tachycardia.

ST segment: The area from the end of the QRS complex to the beginning of the T wave was evaluated. An ST elevation of ≥0.1 mV (1 small square) relative to the point J, where the S wave ends, and an ST depression of ≥0.05 mV (1/2 small square) in two consecutive anatomical leads were considered significant. A T-wave depression of ≥0.1 mV in two consecutive anatomical leads was accepted as T negativity and the presence of sharp T waves, as T sharpness.

P wave: It was obtained by measuring the distance from the beginning to the end of the P wave.

PR range: It was obtained by measuring the segment between the beginning of the P wave and the beginning of the QRS complex. Its normal value was 0.12–0.20 s, and values of >20 s were defined as PR prolongation.

QRS range: It was calculated as the time between the beginning of the Q wave and the end of the S wave.

QT interval: It was obtained by measuring the time from the beginning of the QRS complex to the end of the T wave. The heart rate-corrected QT interval (QTc) was calculated by dividing the QT distance by the square root of R-R interval (Bazett formula). It was defined as long QT for values greater than 460 ms in women and 440 ms in men.

Pathological Q wave: It was considered pathological if the Q wave was wider than 1 mm (0.04 s) or deeper than 2 mm, or if it was greater than one-fourth of the R wave in the same lead.

Axis: The electrical axis of the heart was obtained by measuring the QRS wave amplitudes. The vector was considered normal if the cardiac axis was between 30° and 90°, as right axis deviation if it was between 90° and 180°, and as left axis deviation if it was between −30° and −90°.

In addition to these measurements, ECG was evaluated as a whole, and if there was any specific ECG finding (left bundle-branch block, right bundle-branch block, and atrial fibrillation), it was noted.

Statistical analysis

SPSS version 15.0 for Windows (IBM Corporation, Chicago, IL, USA) was used for statistical analyses. Categorical variables were presented as numbers and percentages and numerical variables, as mean ± standard deviation. The Shapiro–Wilk test was used to determine how the variables were distributed. The Chi-square test was used to compare categorical data between groups. In the comparison of numerical variables, the student's T test was used when the variables showed normal distribution, and the Mann–Whitney U test was used when they showed non-normal distribution. When a numerical variable was compared among more than two groups, analysis of variance test was used for parameters with a normal distribution and the Kruskal–Wallis test was used for parameters with a non-normal distribution. Pearson and Spearman correlation analysis were performed for variables with normal and non-normal distribution, respectively. Multivariate binary regression analysis was performed to analyze whether certain variables had a role in the presence of pathological findings on ECG and whether ECG findings had a role in mortality. *P*-values of <0.05 were considered significant.

Ethical approval: This study was approved by the Turkish Ministry of Health Scientific Research Platform (İskender Ekinci-2020-05-06T18_35_20) and the Clinical Research Ethics Committee of Health Sciences University, Kanuni Sultan Suleyman Training and Research Hospital, Istanbul, Turkey (Number: KAEK/2020.05.57). Study procedures were performed in accordance with the 2009 Helsinki Declaration.

Results

A total of 100 patients, 54 of which were male, diagnosed with COVID-19, were included in the study. The mean age of the patients was 56.8(16.7) (20–92) years, and 63 were over 50 years old. Among presentation complaints, 66% of the patients had cough, 52% had weakness, 50% had shortness of breath, and 45% had high fever, followed by myalgia, sore throat, and headache, which were less common. Signs of pneumonia were detected in 89 patients on imaging, whereas 11 had no pneumonia. Fifty-two patients had at least one comorbid disease, and the most common among these were hypertension (n = 32) and type-2 diabetes mellitus (n = 24). The mean hospitalization period of the patients was 8.19 (3.5) (3–19) days, the mean systolic and diastolic blood pressures were 117.8 (10.9) mmHg (100–150), and 71.9 (8.1) (50–95) mmHg, respectively. The mean oxygen saturation rate at room air was 93.7 (4.2) % (75–98), and the mean respiratory rate per minute was 19.2 (2.1) (14–28). Twenty-five patients had mild, 53 had moderate, and 22 had severe disease. Six of the patients included in the study died, and the mortality rate was 6% within the study group.

The number of patients with at least one abnormal ECG finding was 58, and the number of patients with ST segment pathology was 26. Patients were grouped as those with and without ST segment pathology, and their results were compared (Table 1). Findings of sex distribution, incidence of pneumonia, distribution of patients according to disease severity, prevalence of type-2 diabetes mellitus and hypertension, and mean systolic and diastolic blood pressures were similar in patients with and without ST segment pathology (*P*>0.05). The respiratory rate, mean troponin value, and proportion of patients with elevated troponin value were higher, whereas mean lymphocyte count was lower in the group with ST segment pathology than in the group without (Table 1).

Table 1: Laboratory parameters in patients with ST segment pathology and those without ST segment pathology

	All patients	Patients with ST segment pathology	Patients without ST segment pathology	*P*-value
n	100	26	74	
Age, year	56.8(16.7)	54.69(19.10)	57.64(15.89)	0.441
Respiration rate, min	19.2(2.1)	20.03(2.47)	19.01(2.01)	0.038
Saturation, %	93.7(4.2)	91.5(6.66)	94.55(2.59)	0.128
Heart rate, bpm	82(13.2)	81.38(15.06)	82.32(12.62)	0.757
Hemoglobin, g/dL	12.71(1.69)	12.72(1.74)	12.71(1.69)	0.975
Leukocyte, 10^3µ/L	6.74(2.44)	6.48(2.29)	6.83(2.5)	0.621
Lymphocyte, 10^3µ/L	1.52(0.79)	1.18(0.65)	1.63(0.81)	0.004
C-reactive protein, mg/L	52.5(58.93)	67.07(80.16)	47.39(49.06)	0.527
Procalcitonin, mg/mL	0.18(0.63)	0.16(0.2)	0.19(0.73)	0.078
Troponin, mg/mL	0.64(1.94)	2.33(3.27)	0.05(0.24)	<0.001
Creatine kinase, U/L	109.14(165.79)	167.88(285.71)	87.89(86.6)9	0.933
D-dimer, mg/L	1.68(4.73)	3.15(8.82)	1.17(1.63)	0.305
Calcium, mg/dL	8.9(0.81)	8.63(1.42)	8.99(0.45)	0.756
Potassium, mg/dL	4.36(0.58)	4.26(0.49)	4.4(0.61)	0.435
Magnesium, mg/dL	2.14(0.47)	2.09(0.43)	2.16(0.48)	0.936
Ferritin, mg/mL	443.79(671.76)	427.05(511.06)	449.67(722.78)	0.747
Fibrinogen, mg/dL	400.41(168.93)	429(155.12	390.36(173.40)	0.318
Elevated troponin, n	48	19 (%73)	29 (%39)	0.003

ST segment pathology was detected in 16%, 22.6%, and 45.4% of patients with mild, moderate, and severe disease, respectively (*P*>0.05). Other pathological ECG findings were similar between the groups (*P*>0.05). Five of the six patients with mortality had ST segment pathology, and the incidence of ST segment pathology was higher than that among survivors (*P*=0.004).

The troponin level was elevated in approximately half of the patients (48%). Sex distribution, incidence of pneumonia, distribution of patients according to disease severity, prevalence of type-2 diabetes mellitus and hypertension, and mean systolic and diastolic blood pressure were similar in patients with elevated and normal troponin levels (*P*>0.05). In patients with elevated troponin levels, the lymphocyte count was lower (*P*=0.014), fibrinogen level was higher (*P*=0.021), and other laboratory parameters were similar compared to those with normal serum troponin (*P*>0.05). Patients with elevated troponin

levels were compared with those with normal troponin levels in terms of ECG findings, and the results obtained are presented in Table 2. The respiratory rate per minute, incidence of T negativity, and abnormal ECG findings were higher in patients with elevated troponin levels than in those with normal troponin levels. Furthermore, troponin levels were above the normal range in all six patients who died.

Table 2: Electrocardiography findings in patients with elevated troponin level and those with normal troponin level

	All patients	Patients with elevated troponin level	Patients with normal troponin level	*P*-value
n	100	48	52	
Age, year	56.8(16.7)	56.3(19.3)	57.3(14)	0.764
Respiration rate, min	19.2(2.1)	19.7(2.2)	18.8(2)	0.03
Saturation, %	93.7(4.2)	93.5(4.9)	93.9(3.4)	0.626
Heart rate, bpm	82(13.2)	83.1(14.8)	81.1(11.6)	0.451
Tachycardia, n	12	7	5	0.544
ST depression, n	7	5	2	0.256
ST elevation, n	4	2	2	1
T wave negativity, n	24	17	7	0.018
T wave sharpness, n	2	2	0	0.228
Long QT, n	24	13	11	0.640
LBBB, n	3	0	3	0.244
RBBB, n	4	1	5	0.207
Atrial fibrillation, n	3	3	0	0.107
Pathologic Q wave, n	7	3	4	1
Normal axis, n	86	41	45	
Left axis deviation, n	9	6	3	0.243
Right axis deviation, n	5	1	4	
Abnormal ECG findings, n	58	33	25	0.044
P duration, s	0.081(0.022)	0.082(0.024)	0.080(0.021)	0.620
PR duration, s	0.168(0.031)	0.171(0.034)	0.166(0.027)	0.515
QRS duration, s	0.08(0.022)	0.079(0.016)	0.081(0.027)	0.679
QT duration, s	0.366(0.044)	0.365(0.047)	0.367(0.042)	0.793
QTc duration, ms	422.51(41.12)	422.68(43.19)	422.34(39.54)	0.788

The troponin level was significantly negatively correlated with lymphocyte count (r: −0.298, *P*=0.003) and significantly positively correlated with respiratory rate (r: 0.215, *P*=0.031) and CRP level (r: 0.233, *P*=0.02). The troponin level did not have any significant correlation with other laboratory parameters (leukocyte count, levels of hemoglobin, procalcitonin, CK, calcium, magnesium, D-dimer, ferritin, and fibrinogen levels) and distances measured on the ECG (P duration, PR duration, QRS duration, QT duration, and QTc).

Respiratory rate, mean oxygen saturation at room air, lymphocyte count, serum troponin level, and elevated troponin level in the univariate regression analysis and only the mean oxygen saturation value in multivariate regression analysis were found to play significant roles in ST segment pathology (Table 3). Similarly, in the univariate regression analyses in which the role of ECG pathologies in the development of mortality was analyzed, ST depression, T negativity, T-wave sharpness, and ST segment pathology were significant parameters (Table 4).

Based on Receiver Operating Characteristics analysis, the cut-off level for serum troponin in showing ST segment pathology was 0.03. As observed in Figure 1, ST segment pathology was significantly more common in patients with troponin value >0.03 mg/mL (*P*<0.001, area under the curve: 0.763, sensitivity: 61.5%, and specificity: 90.5%).

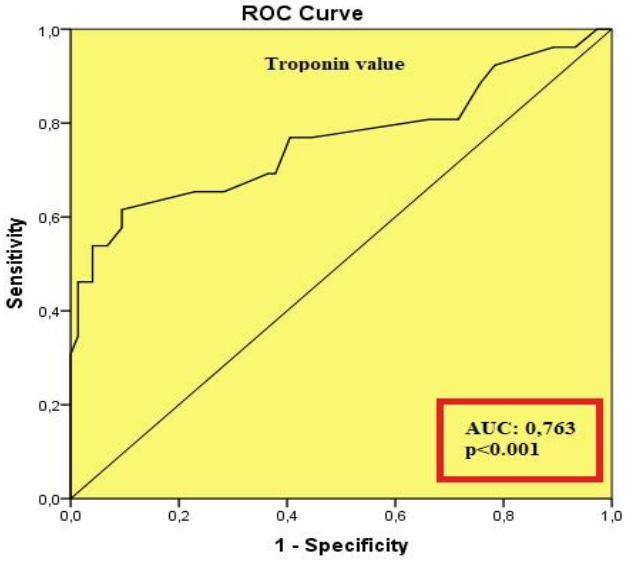
Table 3: Univariate and multivariate logistic regression analysis on the presence of ST segment pathology in patients with Covid-19

Variable	Univariate OR	Univariate 95% Cl (Lower-Upper)	Univariate *P*-value	Multivariate OR	Multivariate 95% Cl (Lower-Upper)	Multivariate *P*-value
Age	1.011	0.984-1.038	0.437	1.030	0.989-1.074	0.157
Male gender	1.242	0.508-3.041	0.635	1.960	0.567-6.769	0.287
Presence of pneumonia	1.076	0.263-4.404	0.919			
Respiratory rate, min	0.796	0.635-0.997	0.047	0.882	0.636-1.224	0.454
Saturation, %	1.175	1.048-1.316	0.006	1.198	1.011-1.419	0.037
Lymphocyte, 10^3µ/L	2.668	1.201-5.924	0.016	1.666	0.656-4.231	0.283
C-reactive protein, mg/mL	0.995	0.988-1.002	0.152			
D-dimer, mg/L	0.924	0.834-1.025	0.136			
Ferritin, mg/mL	1	0.999-1.001	0.882	1.001	0.999-1.003	0.230
Troponin, mg/mL	0.183	0.055-0.609	0.006	0.295	0.099-0.878	0.028
Elevated troponin	0.237	0.089-0.635	0.004			

Table 4: Univariate and multivariate logistic regression analysis on the mortality by electrocardiographic findings in patients with Covid-19

Variable	Univariate OR	Univariate 95% Cl (Lower-Upper)	Univariate *P*-value	Multivariate OR	Multivariate 95% Cl (Lower-Upper)	Multivariate *P*-value
Tachycardia	0.238	0.039-1.469	0.122			
ST depression	8.9	1.303-60.8	0.026	1.635	0.165-16.214	0.674
ST elevation	0	0 -	0.999			
T wave negativity	7.4	1.263-43.348	0.026	0.296	0.008-10.937	0.509
ST pathology	17.381	1.924-157	0.011	39.027	0.592-2573.7	0.086
T wave sharpness	18.6	1-342.9	0.049	2.255	0.054-93.314	0.669
Pathologic Q wave	2.93	0.294-29.28	0.359	1.497	0.064-35.23	0.802
PR duration	7.80	0.013-4.54	0.09			
Prolonged PR	6.067	0.531-69.3	0.147			
QT duration	0	0-6.837	0.073			
QTc duration	0.983	0.960-1.006	0.140			

Figure 1: Receiver operating characteristic analysis for serum troponin level on occurrence ST segment pathology



Discussion

In this study, ECG pathologies were analyzed in patients with COVID-19 and more than half of the patients had at least one pathological finding on ECG and approximately quarter of the patients had ST segment pathologies. In addition, the serum troponin level was elevated as a marker of cardiac damage in approximately half of the patients. Furthermore, we found that respiratory rate, mean oxygen saturation, troponin levels, and lymphocyte count were significant predictors of ST segment pathologies detected on ECG, whereas ST depression, T negativity, T sharpness, and ST segment pathology were significant predictors of mortality. The data of six non-survivor patients revealed that troponin levels were elevated in all, and five patients had ST segment pathology.

In the present study, the most common ECG pathologies were T-wave negativity and long QT (24%), followed by tachycardia (12%), left axis deviation (9%), ST depression (7%), pathological Q wave (7%), short QT (6%), right axis deviation (5%), ST elevation (4%), right bundle branch block (4%), left bundle branch block (3%), atrial fibrillation (3%), and T sharpness (2%), respectively. In a similar study, abnormal ECG findings were reported in 63% of the cases, including ST–T changes (32.6%), sinus tachycardia (12.5%), atrial fibrillation (6.6%), abnormal Q wave (5.6%), right bundle branch block (9.7%), left bundle branch block (0.9%), sinus bradycardia (6%), and long QT (1.3%) [11]. In another study, most patients had ECG pathology, including atrial fibrillation / flutter (5.6%), AV block (2.6%), premature atrial contractions (7.7%), premature ventricular contractions (3.4%), left-axis deviation (13.8%), right-axis deviation (5.5%), right-bundle branch block (7.8%), left-bundle branch block (1.5%), left ventricular hypertrophy (15.5%), right ventricular hypertrophy (4%), pathological Q wave (13.9%), localized ST elevation (0.7%), and localized T wave inversion (10.5%) [12]. In a study conducted in our country, tachycardia was found in 31%, ST segment pathology in 28%, T negativity in 22%, ST depression in 20%, and long QT in 5% of the patients [13]. In yet another study, ST–T abnormalities were reported in 30% and left ventricular hypertrophy, in 33% of the patients [14]. Considering both our own study and previous similar studies, although pathological ECG findings vary, these pathologies are very common in patients with COVID-19.

In the present study, the incidence of ST segment pathologies detected on ECG increased in parallel with the worsening of the disease presentation and led to a difference between the groups. The incidence of ST segment pathology was 45% in patients with severe disease and 16% in patients with mild disease. Other pathological ECG findings in the present study were similar among the groups in terms of disease severity. Deng Q et al. reported that myocarditis, segmental wall motion abnormality on ECG, pulmonary hypertension, and pericardial effusion were more common and the mean ejection fraction was lower in patients with severe disease; however, tachycardia and ST segment pathologies were not different between patients with and without severe disease [15]. McCullugh et al. reported that many pathological ECG findings were more common in non-surviving patients, and in a study conducted in Turkey, ST segment pathologies, ST depression, and T negativity were reported more commonly in patients with severe disease [12, 13]. In a COVID-19 case series of 18 patients with ST elevation, this ECG pathology occurred at the time of admission in 10 patients, and the outcome was mortality in 13 patients [16]. In the present study, five of 26 patients with ST segment pathology died.

In our study, the respiratory rate, troponin value, percentage of patients with elevated troponin value, and mortality rate were higher, and the lymphocyte count was lower in patients with ST segment pathology than in those without. The respiratory rate, mean oxygen saturation, lymphocyte count, and troponin level were significant predictors for the development of ST segment pathology. In a study by Wang et al., troponin and N-terminal pro-brain natriuretic peptide levels, and in a study by Barman et al., presence of hypertension, severe disease presentation, myocardial damage, and elevated D-dimer levels were reported as the parameters that play a role in the emergence of ST segment changes on ECG [11, 13].

In this study, approximately half of the patients had elevated cardiac troponin levels. The respiratory rate was higher and T negativity and abnormal ECG findings were more common in patients with elevated troponin levels. Elevated cardiac troponin level in all patients with mortality was another remarkable finding. In many previous studies, similar to the present study, the cardiac troponin level was higher in patients with severe disease, which required intensive care unit follow-up, or resulted in mortality [9, 13, 17]. Deng Q et al. reported that troponin levels were comparatively higher in patients with severe disease, whereas the respiratory rate was higher and saturation was lower in patients with elevated troponin levels [15]. Zhou et al. found the rate of patients with elevated troponin levels to be 17%, which increased to 46% in a subgroup with mortality: They suggested that cardiac troponin level was a significant marker for mortality rate [9]. In different studies of a similar nature, the rate of cardiac involvement varied between 7.2% to 27.8% in patients with COVID-19 disease [11, 15, 17, 18]. Barman et al. demonstrated that QRS segment duration was longer and long QRS, long QT, ST depression, T negativity, and ST segment pathologies were more common in patients with cardiac damage than in those without [13]. In the present study, only the incidence of T negativity was different, whereas other ECG pathologies were similar in patients with elevated and normal troponin levels. In addition, PR, QRS, QT, and QTc distances were not different between the two groups in the present study.

Studies have shown that patients with cardiac damage have older age, a longer period of hospitalization, more comorbid diseases such as type 2 diabetes mellitus and hypertension, need mechanical ventilation more often, have more disease-related complications, and higher mortality rates than patients without cardiac damage [5, 18]. In these studies, higher leukocyte and neutrophil counts as well as higher CRP, procalcitonin, CK myocardial band, troponin, and D-dimer levels and lower lymphocyte count and calcium levels were reported in patients with cardiac damage. In addition, it has been reported in a case series that cardiac damage markers are closely associated with inflammation parameters [16]. In the present study, the lymphocyte count was lower and the fibrinogen level was higher in patients with elevated troponin levels, whereas age, sex distribution, prevalence of comorbid diseases, mean blood pressure, and incidence of pneumonia were similar. We also observed that the troponin level had a significant negative correlation with lymphocyte count and a significant positive correlation with respiratory rate and CRP level.

Limitations

The limitations of this study include the relatively smaller sample size, especially in terms on non-surviving patients, and the fact that only basal ECG and laboratory data were analyzed. The reason for this is that cardiac pathologies that may arise secondary to the drugs used were intended to be excluded from the scope of this study. This is because our aim in this study was to examine only the cardiac pathologies caused by the presentation of COVID-19. Another limitation is that we did not have former ECG records of these patients before COVID-19

infection. We tried to eliminate this limitation by excluding patients with any known cardiac disease, history of arrhythmia or anti arrhythmic drug use, and those who had previously undergone coronary imaging due to cardiac complaints. Moreover, we believe that prospective studies in which surviving patients who develop cardiac damage are followed up periodically in terms of cardiac signs and symptoms will shed light on the long-term consequences of cardiac involvement.

Conclusions

Considering both findings of the present study and the results of previously published articles, it can be concluded that cardiac involvement is very common in patients with COVID-19 and manifests as both elevated cardiac troponin levels and pathological ECG findings. Moreover, the mortality rate is higher in patients with cardiac damage. Pre-existing cardiac history as well as cardiac involvement during the COVID-19 disease period are important factors for assessing the course of the disease. At this point, we think the present study results are remarkable. In the light of this information, we believe that close monitoring of patients with COVID-19 in terms of the development of cardiac damage will be an appropriate approach, both at an early stage during diagnosis of the disease and while being followed up under treatment.

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