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Wide QRS Complex and Lateral ST-T Segment Abnormality Are Associated With Worse Clinical Outcomes in COVID-19 Patients

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ABSTRACT

Background: The information on electrocardiographic features of patients with coronavirus disease 2019 (COVID-19) is limited. Our aim was to determine if baseline electrocardiographic features of hospitalized COVID-19 patients are associated with markers of myocardial injury and clinical outcomes.

Methods: In this retrospective, single center cohort study, we included 223 hospitalized patients with laboratory-confirmed COVID-19. Clinical, electrocardiographic and laboratory data were collected and analyzed. Primary composite endpoint of mortality, need for invasive mechanical ventilation, or admission to the intensive care unit was assessed.

Results: Forty patients (17.9%) reached the primary composite endpoint. Patients with the primary composite endpoint were more likely to have wide QRS complex (>120 ms) and lateral ST-T segment abnormality. The multivariable Cox regression showed increasing odds of the primary composite endpoint associated with acute respiratory distress syndrome (odds ratio 7.76, 95% CI 2.67−22.59; $p < 0.001$), acute cardiac injury (odds ratio 3.14, 95% CI 1.26−7.99; $p = 0.016$), high flow oxygen therapy (odds ratio 2.43, 95% CI 1.05−5.62; $p = 0.037$) and QRS duration longer than >120 ms (odds ratio 3.62, 95% CI 1.39−9.380; $p = 0.008$) Patients with a wide QRS complex (>120 ms) had significantly higher median level of troponin T and pro-BNP than those without it. Patients with abnormality of lateral ST-T segment had significantly higher median level of troponin T and pro-BNP than patients without.

Conclusions: The presence of QRS duration longer than 120 ms and lateral ST-T segment abnormality were associated with worse clinical outcomes and higher levels of myocardial injury biomarkers.

Keywords: COVID-19; Electrocardiogram; Mortality; Myocardial injury; Wide QRS complex. **[Am J Med Sci 2021;361(5):591–597.]**

INTRODUCTION

Novel coronavirus disease 2019 (COVID-19) has resulted in the deaths of more than 764 000 people worldwide as of August 15, 2020.[1] Recently, cases of severe COVID-19 pneumonia have been independently associated with risk of mortality, and substantial evidence has demonstrated the presence of cardiac injury in patients with COVID-19.[2,3] A recent study showed that 20% of COVID-19 patients with acute cardiac injury have higher mortality than COVID-19 patients without cardiac injury.[4] However, electrocardiography (ECG) data were lacking from clinical examinations of patients in isolation wards or the intensive care units, which hinders the determination of the exact mechanisms of cardiac injury and the potential prediction of clinical outcome. Therefore, the present study retrospectively analyzed data from a tertiary referral university hospital in Istanbul, Turkey, to examine the potential association between baseline electrocardiographic findings and in-hospital outcome among patients with COVID-19 pneumonia.

METHODS

Study design

The study was approved by the Ethics Committee of Istanbul Faculty of Medicine, Istanbul University and the

Turkish Ministry of Health. The requirement of written informed consent was waived by the ethics committee of the designated hospital for patients with emerging infectious diseases. This retrospective cohort study included adult inpatients (≥18 years old) from Istanbul Faculty of Medicine, Istanbul University (Istanbul, Turkey). All adult patients who were diagnosed with having COVID-19 pneumonia according to the World Health Organization's (WHO's) interim guidance were screened, and those who died or were discharged between March 10, 2020, and June 10, 2020, were included in the study. Patients were excluded if they were suspected of having COVID-19 pneumonia but had a negative nasopharyngeal swab for SARS-CoV-2 were excluded from the study.

Data collection

SARS-CoV-2 nucleic acid was detected in all patients by real-time PCR to confirm the viral infection. Computed tomography was performed for the diagnosis of pneumonia. Patients' medical records were carefully reviewed and analyzed by two trained physicians (A.N. and Z.G.D.). The patient data obtained included demographic information, comorbidities, signs and symptoms, laboratory results and complications. Patients' comorbidities were extracted from the electronic health records, including hypertension, diabetes, coronary artery disease, cerebrovascular disease, chronic kidney disease, chronic obstructive pulmonary disease (COPD) and malignancy.

Standard 12-lead electrocardiograms were recorded with a speed of 25 mm/s and a gain of 10 mm/mV on the first day of admission and were manually evaluated by cardiologists (M.Y. and H. O.). ECGs were assessed for sinus rhythm or atrial fibrillation, axis deviation, presence of left ventricular hypertrophy, presence of ST segment changes (localized ST elevation or depression) or inverted T wave (deeper than 0.5 mm in leads -DI, -DΠ, and V_3–V_6), and presence of atrioventricular or bundle branch block. Left bundle branch block (LBBB), right bundle branch block (RBBB), and nonspecific intraventricular conduction disturbance (NICD) were defined according to the criteria of the American College of Cardiology Foundation and Heart Rhythm Society.[5]

Anterior leads were defined as leads V1-V4, the lateral leads were V5, V6, I, aVL and the inferior leads were II, III, and aVF. The QRS duration and QT interval were measured from leads of V1-V6, II, DIII, and aVF in digitized 12 lead ECG recordings using the on-screen digital caliper software Cardio Calipers version 3.3 (Iconico, Inc.). Interobserver correlation for determining QRS duration and QT intervals was assessed using the intraclass correlation coefficient (ICC) test, and a strong correlation between observers was found (ICC:0.94, $p < 0.001$).

The occurrence of complications was confirmed by three physicians (E.A.G., M.T. and D.D.) according to the following criteria. Sepsis and septic shock were defined according to the 2016 Third International Consensus Definition for Sepsis and Septic Shock.[6] Acute kidney injury was defined in accordance with the KDIGO Clinical Practice Guidelines for Acute Kidney Injury as one of the following: an increase in serum creatinine by ≥0.3 mg/dl within 48 h, an increase in serum creatinine to ≥ 1.5 times baseline within the previous 7 days, or urine volume ≤ 0.5 ml/kg/h for 6 hours.[7] Cardiac injury was defined by the serum levels of cardiac troponin T (cTnT) above the 99th percentile up of the reference limit. ARDS was defined using the parameters of timing, chest imaging, origin of edema and oxygenation in accordance with the Berlin definition.[8] The clinical outcomes in hospital (i.e., discharges, mortality, and length of stay) were monitored up to June 10, 2020 (the final date follow-up date).

Statistical Analysis

Descriptive statistics were obtained for all study variables. All categorical variables were compared for the study outcome by using the Fisher exact test or $\chi 2$ test, and continuous variables were compared using the t test or the Mann-Whitney U test, as appropriate. The Kruskal-Wallis test was used to compare continuous variables between three or more groups. Continuous data are expressed as the mean (SD) or median (interquartile range [IQR]) values. Categorical data are expressed as proportions.

The clinical and laboratory variables that differed significantly in the comparative statistical analyses were included in a univariate Cox regression analysis. The models used in the univariate analysis were tested by the Likelihood ratio test, Wald test and score (log-rank) test. Multivariate Cox regressions were then performed for comorbidities, complications, laboratory, and ECG parameters. Multivariate Cox regression models were used to determine the independent risk factors for death during hospitalization. Data were analyzed using SPSS version 26.0 (IBM). For all the statistical analyses, $p < 0.05$ was considered significant and all testing was 2-sided.

RESULTS

Patient Characteristics

From March 10, 2020, through June 10, 2020, 759 patients were hospitalized due to possible COVID-19 pneumonia. Of these patients, 626 patients underwent an ECG at or near the time of their admission. Among these patients, 225 were confirmed by PCR to have SARS-CoV-2. Two patients were excluded from the study due to the first presentation as being intracranial hemorrhage. Therefore, 223 patients were enrolled in our study.

The clinical characteristics and comorbidities of the patients are shown in Table 1. The mean age was 57 ± 15 years, 40% were women, 37% of patients had hypertension, 26% had diabetes mellitus, 9% had coronary artery disease 2% had cerebrovascular disease and 2% had chronic heart failure. Other comorbidities were

TABLE 1. Baseline Characteristics, Symptoms and Comorbidities of Patients on Admission

Characteristic	Patients, No. (%)			
	All (n = 223)	Primary Composite Endpoint		*p* Value
		With (n = 40)	Without (n = 183)	
Age, mean (SD)	57 (15)	65 (13)	55 (15)	<0.001
Female	90 (40)	11 (28)	79 (43)	0.067
Signs and symptoms at admission				
Fever	128 (57)	25 (63)	103 (56)	0.702
Cough	163 (72)	23 (58)	140 (77)	0.014
Dyspnea	94 (42)	26 (65)	68 (37)	0.001
Chronic medical illness				
Hypertension	83 (37)	21 (53)	61 (33)	0.023
Diabetes mellitus	58 (26)	10 (25)	48 (26)	0.872
Coronary artery disease	20 (9)	7 (18)	13 (7)	0.037
Cerebrovascular disease	5 (2)	2 (5)	3 (2)	0.193
Chronic heart failure	5 (2)	3 (8)	2 (1)	0.013
Chronic renal failure	14 (6)	6 (15)	8 (4)	0.012
COPD	11 (5)	4 (10)	7 (4)	0.102
Malignancy	18 (8)	7 (18)	11 (6)	0.016

also present among patients: 8% of the patients had a history of malignancy, 6% had chronic renal failure and 5% had chronic obstructive pulmonary disease. The patients' laboratory and radiological findings at admission are shown in Table 2.

Electrocardiographic characteristics

The patients' baseline electrocardiographic features are shown in Table 3. The vast majority of patients were in sinus rhythm (99.1%), and only 1.8% of patients had atrial fibrillation. The median QRS duration was 88 ms (IQR, 80–96 ms), and the median corrected QT interval (using the Bazett formula) was 428 ms (IQR, 407–453 ms). A significant proportion (24.2%) had an abnormal axis (23.8% of patients had left axis deviation, and 0.4% had a right axis deviation). Furthermore, 8.4% had premature contractions (4.9% had atrial, and 3.5% had ventricular premature contractions).

Abnormal intraventricular conduction was found in 11.1%, with RBBB in 5.8%, left bundle branch block in 1.3%, and nonspecific intraventricular conduction delay in 4%. Evidence of a previous Q-wave myocardial infarction was present in 3.5%. Left ventricular hypertrophy (Cornell voltage) was found in 3.6% of patients. A significant proportion (14.6%) had repolarization abnormality: 8.1% had lateral, 3.1% had extensive, 2.2% had inferior and 1.3% had anterior ST-T segment abnormality.

Treatment, complications and clinical outcome

The patients' treatment data are summarized in Supplementary Table 1. During follow-up, a total of 40 patients (17.9%) reached the primary composite endpoint of mortality, need for invasive mechanical ventilation or admission to the intensive care unit. The median time to reach the endpoint was 8 days (IQR, 3–11 days). A total of 25 patients (11.1%) died, and 198 patients (88.8%) were discharged. Complications were more common among patients with the primary composite endpoint than those it and included sepsis (30 [75%] vs. 25 [13.7%]; $p < 0.001$), septic shock (22 [55%] vs. 0 [0%]; $p < 0.001$), ARDS (31 [78%] vs. 1 [0.5%]; $p < 0.001$), acute kidney injury (15 [38%] vs. 6 [3.3%]; $p < 0.001$), acute cardiac injury (32 [80%] vs 32 [17.5%]; $p < 0.001$), acute heart failure (12 [30%] vs. 4 [2.2%]; $p < 0.001$) and arrhythmia (5 [13%] vs. 2 [1.1%]; $p < 0.001$) (Table 3).

The relationship between electrocardiographic parameters and clinical outcomes

ECG findings of a wide QRS complex (>120 ms) and lateral ST-T segment abnormality were more common in patients with the primary composite endpoint than those without it [{9/40 (22%) vs. 12/171 (6.6 %); $p = 0.002$}, {10/40 (25%) vs. 8/171 (4.4%); $p < 0.001$}, respectively]. Wide QRS complex was able to predict worse clinical outcomes with a sensitivity of 22.5%, specificity of 93.4%, positive predictive value of 42.8%, and negative predictive value 84.7%. Lateral ST-T segment abnormality was able to predict the primary composite endpoint with a sensitivity of 25%, specificity of 95.5%, positive predict value of 55.6%, and negative predictive value of 85.3%. The multivariable Cox regression showed increasing odds of the primary composite endpoint associated with acute respiratory distress syndrome (odds ratio 7.76, 95% CI 2.67–22.59; $p < 0.001$), acute cardiac injury (odds ratio 3.14, 95% CI 1.26–7.99; $p = 0.016$), high flow oxygen therapy (odds ratio 2.43, 95% CI 1.05–5.62; $p = 0.037$), and QRS duration longer than >120 ms (odds ratio 3.62, 95% CI 1.39–9.380; $p = 0.008$) (Table 4).

Patients with a wide QRS complex (>120 ms) had a significantly higher median level of troponin T (14,1 pg/mL

TABLE 2. Baseline Laboratory and Radiologic Findings of Patients on Admission

Laboratory findings at admission, median (IQR)	Patients, No. (%)			
	All (n = 223)	Primary Composite Endpoint		*p* Value
		With (n = 40)	Without (n = 183)	
Leukocytes/μL	6100 (4400–7800)	6795 (5237–9575)	5800 (4330–7570)	0.006
Lymphocytes/μL	1160 (850–1610)	970 (592–1287)	1200 (900–1650)	<0.001
Neutrophils/μL	3780 (2830–5770)	5360 (3720–8435)	3470 (2680–5400)	<0.001
Hemoglobin, g/dL	13.2 (12–14.2)	12.7 (12.2–14.1)	13.2 (12.0–14.1)	0.898
Platelets x10^3/μL	194 (154–243)	194 (152–306)	193 (155–240)	0.584
C-reactive protein, mg/dL	49 (21–91)	98 (55–167)	43 (17–77)	<0.001
Procalcitonin, ng/mL	0.09 (0.05–0.185)	0.233 (0.115–0.455)	0.08 (0.05–0.13)	<0.001
High-sensitivity troponin T, pg/mL	6 (3–14)	17 (7–48)	5 (3–11)	<0.001
N-terminal pro-B-type natriuretic peptide, pg/mL	97 (32–340)	658 (195–1746)	67 (25–186)	<0.001
Creatinine, mg/dL	0.88 (0.76–1.06)	1.0 (0.8–1.3)	0.9 (0.7–1.0)	<0.001
Sodium, mEq/L	138 (136–141)	137 (135–140)	138 (136–141)	0.022
Potassium, mEq/L	4.2 (3.9–4.6)	4.3 (3.8–4.7)	4.2 (3.9–4.6)	0.579
Calcium, mg/dL	8.7 (8.4–9.1)	8.5 (8.1–8.8)	8.8 (8.4–9.2)	0.001
Magnesium, mEq/L	0.86 (0.80–0.92)	0.83 (0.75–0.96)	0.87 (0.80–0.91)	0.540
Alanine aminotransferase, U/L	24 (15–38)	28 (15–44)	24 (15–37)	0.475
Aspartate aminotransferase, U/L	28 (21–46)	37 (24–57)	27 (20–40)	0.024
Lactate dehydrogenase, U/L	255 (212–340)	364 (243–475)	248 (209–308)	<0.001
Albumin, g/dL	3.9 (3.5–4.2)	3.6 (3.1–4.0)	4.0 (3.6–4.3)	<0.001
D-dimer, μg/L	725 (460–1340)	1530 (775–385)	660 (435–1070)	<0.001
Ferritin, ng/mL	322 (147–682)	643 (222–1198)	303 (133–584)	0.001
Chest computed tomography findings				0.429
Consolidation	21 (9.4)	2 (5)	19 (10)	
Ground-glass opacity	109 (48.9)	23 (55)	86 (47)	
Bilateral pulmonary infiltration	81 (36.3)	14 (33)	67 (37)	

Abbreviation: COPD, chronic obstructive pulmonary disease; IQR, interquartile range.

[IQR, 7,6–38 pg/mL] vs. 5,8 pg/mL [IQR, 3–12 pg/mL]; $p < 0.001$) and pro-BNP (346 pg/mL [IQR, 58–1363 pg/mL] vs. 87 pg/mL [IQR, 31–261 pg/mL]; $p = 0.033$) than patients without it (Table 5; Figs. 1A, B). Patients with abnormality of the lateral ST segment had a significantly higher median level of troponin T (27 pg/mL [9–49 pg/mL] vs. 5,8 pg/mL [3–11.9 pg/mL]; $p = 0.003$) and pro-BNP (839 pg/mL [237–1720 pg/mL] vs. 73 pg/mL [30–216 pg/mL]; $p = 0.001$) than patients without it (Table 6; Figs. 1C, D).

DISCUSSION

The present study retrospectively analyzed hospitalized COVID-19 patients in terms of the relationship between baseline ECG parameters and the primary composite endpoint of mortality, need for invasive mechanical ventilation or admission to the intensive care unit. We found out that median corrected QT interval and QRS duration were longer in patients with the primary endpoint than in patients without it. In addition, left axis deviation, left ventricular hypertrophy, ventricular premature contractions and lateral ST-T segment abnormality were more common in patients with the primary endpoint.

Recently, in a study on 756-patient, McCullough et al. reported, that intraventricular conduction block, repolarization abnormalities and RBBB in COVID-19 patients were associated with an increased risk of mortality.[9] In comparison, the presence of a wide QRS complex (>120 ms) was associated with 3.43-fold increased odds of worse clinical outcome in our study. Consistently, Ukena et al. reported that a QRS width greater than 120 ms in duration in acute myocarditis is associated with a substantial risk of cardiac death or need for heart transplantation.[10] In our study, patients with a wide QRS complex in the baseline ECG had higher levels of troponin T and pro-BNP than patients without it.

Compared to patients without the primary composite endpoint, those with the endpoint were more likely to have acute cardiac injury in our study. Likewise, cardiac involvement was evident in a retrospective cohort study on 191 adult patients in Wuhan, China. Among the non-survivors (54 patients), 59% had acute cardiac injury.[11] A prospective cohort study in Wuhan, China, analyzed laboratory findings from 416 hospitalized patients diagnosed with COVID-19. Of this cohort, 19.7% of the patients had cardiac injury, and required both invasive and non-invasive mechanical ventilation more frequently than patients without cardiac injury. Complications and mortality were also more common in patients with cardiac injury.[4] It is plausible that cardiac injury related to COVID-19 plays a crucial role in the pathophysiology of the disease.

TABLE 3. Baseline Electrocardiographic Features, Complications and Clinical Outcomes of COVID-19 Patients

Parameter	Patients, No. (%)			
	All (n = 223)	Primary Composite Endpoint		*p* Value
		With (n = 40)	Without (n = 183)	
PR duration, ms (IQR)	160 (140–170)	150 (133–168)	160 (140–171)	0.231
QRS duration, ms (IQR)	88 (80–96)	90 (84–109)	85 (80–95)	0.013
QTc (Bazett), ms (IQR)	428 (407–453)	451 (418–490)	426 (403–449)	<0.001
QTc (Framingham), ms (IQR)	404 (384–449)	419 (392–445)	401 (382–422)	0.011
Wide QRS complex (>120 ms)	21 (9.4)	9 (22)	12 (6.6)	0.002
Rhythm				
Sinus	221 (99.1)	39 (98)	182 (99)	0.908
Atrial fibrillation	4 (1.8)	2 (5)	2 (1)	0.092
Intraventricular conduction delay				0.046
RBBB	13 (5.8)	5 (13)	8 (4)	0.046
LBBB	3 (1.3)	1 (3)	1 (0.5)	0.230
Nonspecific intraventricular conduction delay	9 (4)	3 (8)	5 (2.7)	0.134
Abnormal ST-T segment changes				
Inferior	5 (2.2)	1 (3)	4 (2.2)	0.920
Anterior	3 (1.3)	1 (3)	2 (1.1)	0.484
Lateral	18 (8.1)	10 (25)	8 (4.4)	<0.001
Extensive	7 (3.1)	2 (5)	5 (2.7)	0.424
Pathologic Q wave				0.690
Inferior	6 (2.7)	2 (5)	4 (2.2)	
Anterior	1 (0.4)	0 (0)	1 (0.5)	
Lateral	1 (0.4)	0 (0)	1 (0.5)	
Left ventricular hypertrophy (Cornell voltage)	8 (3.6)	4(10)	4(2.2)	0.025
Axis				
Left axis deviation	53 (23.8)	14 (36)	38 (20.8)	0.046
Right axis deviation	1 (0.4)	0 (0)	1 (0.5)	0.617
Extrasystole				
Atrial	11 (4.9%)	3 (7.5%)	7 (3.8%)	0.317
Ventricular	8 (3.5%)	4 (10%)	4 (2.2%)	0.016
Complications				
Sepsis	55 (24.7)	30 (75)	25 (13.7)	<0.001
Septic shock	22 (9.9)	22 (55)	0 (0)	<0.001
ARDS	32 (14.3)	31 (78)	1 (0.5)	<0.001
Need for intensive care unit	38 (17)	38 (95)	0(0)	<0.001
Acute cardiac injury	64 (28.6)	32 (80)	32 (17.5)	<0.001
Arrhythmia	7 (3.1)	5(13)	2 (1.1)	<0.001
Acute heart failure	16 (7.2)	12 (30)	4 (2.2)	<0.001
Acute kidney injury	21 (9.4)	15 (38)	6 (3.3)	<0.001
Clinical outcome				
Median length of stay, days	10 (7–14)	21 (14–26)	9 (7–12)	<0.001
Discharged	198 (88.8)	15 (38)	183 (100)	<0.001
Died	25 (11.1)	25 (63)	0 (0)	<0.001

Abbreviation: ARDS, acute respiratory distress syndrome; LBBB, left bundle branch block; RBBB, right bundle branch block

Another striking finding of our study was that the patients with lateral ST-T segment abnormality had higher levels of troponin T and pro-BNP than patients without. Lateral ST-T segment abnormality was associated with worse clinical outcomes. Shi et al.[4] recently reported that ECGs performed during a period of elevated cardiac biomarkers showed ST-T segment changes compatible with myocardial ischemia in COVID-19 patients. They concluded that observed cardiac injury may be a result of type two myocardial infarction, secondary to ischemia triggered by a mismatch between oxygen supply and demand caused by the severe respiratory illness. Furthermore, it is also possible that the virus's direct impact on the cells can lead to myocarditis.[4]

It has been hypothesized that indirect inflammatory mechanisms are more likely related to the cardiac complications associated with COVID-19.[12] However, Tavazzi et al. demonstrated the first case of biopsy-proven myocardial localization of coronavirus in a

TABLE 4. Multivariate Cox Regression Analysis on the Risk Factors Associated with the Primary Composite Endpoint in Patients with COVID-19

	Hazard Ratio (95% CI)	*p* Value
Age	1.018 (0.986–1.050)	0.270
Cardiovascular diseases	0.583 (0.192–1.767)	0.340
Chronic kidney failure	1.701 (0.480–6.029)	0.340
Hypertension	1.395 (0.574–3.389)	0.463
Acute cardiac injury	3.141 (1.235–7.991)	0.016
Acute respiratory distress syndrome	7.817 (3.175–19.243)	<0.001
High flow oxygen therapy	2.432 (1.054–5.615)	0.037
Malignancy	1.819 (0.632–5.235)	0.268
Wide QRS complex (>120 ms)	3.428 (1.401–8.385)	0.007

COVID-19 patient with cardiogenic shock.[13] Other possible mechanisms of myocardial injury include myocardial interstitial fibrosis, interferon mediated immune response, increased cytokine response by helper T cells, coronary plaque destabilization, and apoptosis-induced cellular damage.[14,15]

Moreover, Algohbani reported a case of acute myocarditis caused by the Middle East Respiratory Syndrome coronavirus (MERS-CoV), which was characterized by myocardial edema and injury in the apical and lateral segments in cardiac magnetic resonance imaging.[16] Therefore, we hypothesize that the similar regional myocardial damage in infections caused by viruses from the same family may contribute to our understanding of the pathophysiology of myocardial injury in COVID-19 patients. Future imaging studies examining the myocardial damage

TABLE 5. Comparison of Levels of Myocardial Injury Biomarkers between Patients with a Wide QRS complex and Patients without it

	Patients with Wide QRS Complex (n = 21)	Patients without Wide QRS Complex (n =198)	*p* Value
Troponin T, median, pg/mL	14,1 (7,6–37,9)	5,8 (3–12)	<0.001
Pro-BNP, median, pg/mL	346 (58–1363)	87 (31–261)	0.033

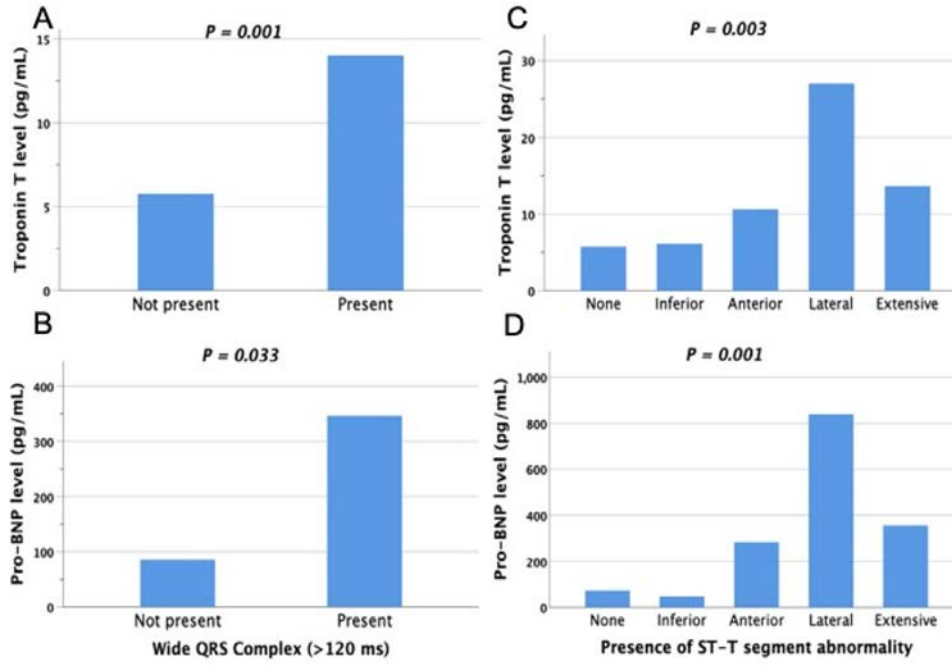

FIGURE 1. A. Bar graph demonstrating statistically significant higher level of median high sensitive troponin T in patients with a wide QRS complex (>120 ms) compared to patients without it. B. Bar graph showing statistically significant higher level of median pro-BNP in patients with a wide QRS complex (>120 ms) compared to patients without it. C. Comparison of median high sensitive troponin T levels among patients with various ST-T segment abnormality. D. Comparison of median pro-BNP levels among patients with various ST-T segment abnormality.

TABLE 6. Comparison of Levels of Myocardial Injury Biomarkers among Patients with Different Repolarization Abnormality

	None (n = 189)	Inferior (n = 5)	Anterior (n = 3)	Lateral (n = 18)	Extensive (n = 7)	*p* Value
Troponin T, median, pg/mL	5,8 (3–11.9)	6,14 (4,4–7,1)	10,6 (7,5–25,8)	27 (9–49)	13,7 (3,7–49)	0.003
Pro-BNP, median, pg/mL	73 (30–216)	47 (19–185)	283 (16–550)	839 (237–1720)	356 (72–1585)	0.001

in COVID-19 represent an important and uncharted area for investigation.

Our study has several strengths. First, we enrolled in only hospitalized patients who had PCR-proven COVID-19 pneumonia. Second, every patient underwent troponin T and pro-BNP level measurements. On the other hand, there were several limitations in the present study. First, because the patients were clinically observed only during hospital stay, so we cannot extrapolate the findings to outpatient settings or longer durations. Second, due to retrospective nature of this study, some parameters were not available for all patients, and in-hospital medications might not have been fully recorded. Third, the information from follow-up ECGs to detect dynamic changes would have increased the power of the study.

Moreover, because we could not obtain prior ECGs for all patients, we were not able to make a comparison between baseline and prior ECGs for detecting dynamic changes. In addition, routine cardiac imaging of every hospitalized COVID-19 patient would have added invaluable information about the correlation with ECG findings. However, this was impossible because of the highly contagious nature of the disease. Nevertheless, our data are consistent with growing data that myocardial involvement has a significant role in the prognosis of COVID-19.[17,18]

Our data demonstrate that ECG may be a useful tool in COVID-19, not only for measuring the QT interval, but also for predicting the prognosis of the hospitalized patients. The presence of a wide QRS complex (>120 ms) and lateral ST-T segment abnormality in the baseline ECG are associated with worse clinical outcomes and elevated levels of myocardial injury biomarkers. Such findings should alert the physicians and warrant closer monitoring of patients.

ACKNOWLEDGEMENTS

We would like thank all doctors, nurses, and other health providers at Istanbul Faculty of Medicine who were involved in taking care of COVID-19 patients. AO and TT accept full responsibility for the accuracy and the integrity of the data as the guarantor of this work and they contributed equally.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.1016/j.amjms.2020.12.012.

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Conflict of Interest: None declared.

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