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The relation between changes in electrocardiography and disease severity in SARS-CoV2 infected patients

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Original Article

Abstract

BACKGROUND: Coronavirus disease 2019 (COVID-19) may lead to myocardial damage and arrhythmia. Patients with ECG changes have shown an increased risk of mortality.

OBJECTIVE: We aimed to study the changes in the electrocardiogram, which may be of great significance for risk stratification of COVID-19-positive patients.

METHODS: A retrospective study was conducted to compare electrocardiogram changes and disease severity markers in COVID-19-positive patients admitted to a referral hospital between February 20 and March 20, 2020.

RESULTS: Our study consisted of 201 cases, including 123 males and 78 females. Ages ranged between 16 and 97 years old. Fifty-two (25.9%) cases had a history of ICU admission. Multivariate logistic regression analysis showed that a low O2 saturation level (OR = 0.920, 95% CI 0.868–0.976, p=0.005), several lab tests, ECG changes (OR = 46.84, 95% CI 3.876– 566.287, p = .002) and Age (OR = 1.03, 95% CI 1.000–1.065, p = .048) were the independent risk factors for predicting mortality rate.

In addition, we utilized multivariate logistic regression analysis, demonstrating that LBBB (OR = 4.601, 95% CI: 1.357–15.600, p=0.014) is the only ECG risk factor associated with morbidity in elderly patients with ECG changes.

CONCLUSIONS: ECG changes are strong indicators of high mortality rates in elderly COVID-19 patients. ECG interpretations should therefore be used for risk stratification and predicting the need for ICU admission.

Keywords: Covid-19,pneumonia, complete heart block

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Introduction

In December 2019, coronavirus disease (COVID-19) emerged in China and spread worldwide, leading to a global pandemic.[1] The SARS-CoV2 virus uses the Angiotensin-Converting Enzyme 2 (ACE2 receptor) to gain access to the upper and lower respiratory cells. ACE2 protein receptors are also expressed in many vital organs, including the heart.[2]

Infection with SARS-CoV-2 may result in varying degrees of cardiac damage.[3] Myocarditis has been reported as a worse prognosis for infected patients.

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Signs and symptoms of infection may range from asymptomatic to mild or severe. In some severe cases, patients may exhibit respiratory distress necessitating intensive clinical care.[4] There are five distinct causes of cardiac mortality and morbidity related to COVID-19: a) myocardial involvement, b) rhythm disturbances, c) thromboembolic events, d) cardiac complications resulting from medical interventions, and e) worsening of preexisting cardiac conditions and diseases.

The current medical practices and standard of care consider Electrocardiography (ECG) as a vital tool for evaluating cardiac health in SARS-CoV2-infected patients.[5] ECG is a low-cost procedure widely utilized in the global healthcare system and poses a minimal risk of infection transmission to clinical personnel.[6, 7]

Consequently, we chose to examine ECG and related disease severity markers in COVID-19-positive patients. This data analysis can help us better understand the potential relationship between ECG changes and other clinical and laboratory data contributing to a patient's increased mortality. We believe the research presented here will provide a more immediate diagnosis of cardiac health in COVID-19-positive patients, thereby allowing for a higher degree of risk stratification, mitigation, and increased patient survival rates.

Materials and Methods

Our retrospective study was used to compare the changes in the ECG and other markers of the disease severity in COVID-19-positive patients. This study was approved by the Medical Ethics Committee of Mashhad University of Medical Sciences (Approval Number: IR.MUMS.REC.1399.116).

Data: We analyzed data obtained from the electronic database of the state's health department regarding COVID-19-positive patients admitted to Emam Reza Hospital, an affiliated hospital of Mashhad University of Medical Science, between February 20, 2020, and March 19, 2020.

Examination method: Our study utilized documented clinical notes from a practicing cardiologist and readings from a twelve-channel conventional ECG performed on each patient upon hospital admission.

Per the 2009 ACC, AHA, and HRS ECG standardization and analysis guidelines, all ECG results were divided into either normal or abnormal categories (8).

Laboratory examination: After patients were admitted to the hospital, their serum biomarkers, including white blood cell (WBC) differential, urea, creatinine, Na, K, Erythrocyte Sedimentation Rate (ESR), and high-sensitivity C-reactive protein (hs-CRP) levels were measured. These tests were administered upon admission.

Statistical analysis: Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) v. 22.0. Normality distribution was examined using the Kolmogorov-Smirnov test. For statistical comparison of the two groups, measured data with a normal distribution were analyzed with an independent sample t-test, whereas those with a non-normal distribution were analyzed with the Mann-Whitney U test. Categorical data were analyzed using Chi-square or Fisher`s exact tests. Multivariate logistic regression was employed to determine the most effective variables pertaining to patient mortality. A p-value <0.05 was considered statistically significant.

Results

This study included a total of 201 cases, including 123 male and 78 female patients ranging in age from 16 to 97 years. According to the classification of the World Health Organization (WHO), elderly patients may have a higher mortality rate; thus, we divided the patients into two groups; ≥ 60 (elderly) and <60 years of age (nonelderly). The demographic data and changes in ECG at the time of admission are presented in Table 1.

Table 2 displays the vital signs and associated laboratory results obtained from patients at the time of hospital admission, as well as patient mortality outcomes according to age ≥ 60 (elderly) and <60 years (nonelderly).

Demographic Values	Frequency (%) N=201
Gender	
Male	123 (61%)
Female	78 (39%)
Age	
≥ 60	96 (48%)
< 60	105 (52%)
Risk Factors	
HTN	82 (40.8%)
DM	51(25.4%)
HLP	17(8.5%)
ESRD	10(5%)
Liver disease	1(0.5%)
CVD	48(23.9%)
PD	21(10.5%)
History of ICU admission	52(25.9%)
Death	33(16.4%)
ACEI or ARB	38 (18.9%)
ECG changes	
Abnormal Rhythm	7(3.5%)
RBBB	5(2.5%)
LBBB	13(6.5%)
ST elevation	3(1.5%)
ST depression	5(2.5%)
Inverted T wave	16(8%)
Long QT interval	8(4%)
Poor R progression	10(10%)
Low voltage	34(16.9%)
LAE	42(20.9%)
RAE	9(4.5%)
LVH	11(5.5%)
Tall R wave in V1	6(3%)
Axis	15(7.5%)
Q wave	7(3.5%)

Hypertension (HTN), diabetes mellitus (DM), hyperlipidemias (HLP), Angiotensin receptor antagonist (ARB), and Angiotensin-converting enzyme inhibitor (ACEI). End-stage renal disease (ESRD), Cardiovascular disease (CVD), and Pulmonary diseases (PD). RBBB: Right bundle branch block, LBBB: Left bundle branch block, LAE: left atrial enlargement, RAE: Right atrial enlargement, LVH: Left ventricular hypertrophy.

In this study, 42% of cases had normal ECG results, while 58% had abnormal ECG results. Patients with ECG changes had a higher heart rate (p=0.009), WBC (p=0.01) and neutrophil (p=0.01) count, creatinine (p=0.003), urea (p<0.001) and CRP (p=0.04) level, but a lower lymphocyte count (p=0.001) and O2 saturation level (p=0.007) (Table 3).

We used Multivariate logistic regression analysis on 201 patients to evaluate the effect of demographic and laboratory variables on in-hospital mortality (as a dependent variable). Thus, in the univariate analysis, we included all significant variables associated with in-hospital mortality in the regression model (using backward LR). Low O2 saturation levels, lymphocyte and PLC, counts, ESR levels, ECG changes, age, and urea levels were determined to be independent risk factors for patient mortality in the final step (Table 4). In another multivariate Logistic Regression analysis of different laboratory and clinical findings of patients based on age division (greater than or equal to 65 years), O2 saturation, Neutrophil, Lymphocyte, and platelet counts, and ECG changes were the independent risk factors for mortality, but only in patients older than 65.

Table 2. Comparison of laboratory results between deceased and fully recovered patients according to age subgroups

Parameters	Deceased (n=33) (mean ± SD)	Recovered (n=168) (mean ± SD)	p-value *
RR N/min	27.51 ± 11.15	23.27 ± 6.08	P=0.03
≥ 60 yrs	26.85 ± 12.13	23.12 ± 5.54	P=0.227
<60 yrs	28.66 ± 9.58	23.33 ± 6.5	P=0.05
HR beat/min	100.45 ± 23.94	94.37 ± 18.86	P=0.06
≥ 60 yrs	103.38 ± 26.01	91.01 ± 17.64	P<0.001
<60 yrs	95.33 ± 19.82	96.95 ± 19.4	P=0.80
T Centigrade	37.22 ± 1.03	37.31 ± 0.97	P=0.89
≥ 6o yrs	37.34 ± 1.25	37.28 ± 0.95	P=0.63
<60 yrs	37.01 ± 0.39	37.32 ± 1	P=0.49
O2 sat Percent	80.36 ± 9.85	87.22 ± 8.46	P<0.001
≥ 6o yrs	79.09 ± 11.89	86.637.99	P<0.001
<60 yrs	82.58 ± 4.07	87.76 ± 8.86	P<0.001
WBC per microliter	10575 ± 4568.05.81	9122.42 ± 4684.44	P=0.06
≥ 60 yrs	10595.23 ± 4766.91	9921.91 ± 47.53.92	P=0.54
<60 yrs	10536.36 ± 4386.17	8486.02 ± 4530.08	P=0.07
Neu per microliter	82.83 ± 8.67	76.03 ± 13.63	P=0.007
≥ 60 yrs	81.81 ± 9.00	77.29 ± 12.01	P=0.14
<60 yrs	84.77 ± 8.04	75.03 ± 14.69	P=0.01
Lymph per microliter	10.57 ± 7.19	17.38 ± 11.27	P<0.001
≥ 60 yrs	11.70 ± 8.13	16.14 ± 10.65	P=0.05
<60 yrs	8.41 ± 4.49	18.30 ± 12.43	P<0.001
PLC per microliter	184.40 ± 99.65	215.26 ± 86.31	P=0.07
≥ 60 yrs	187.76 ± 94.65	222.95 ± 76.96	P=0.06
<60 yrs	178.00 ± 113.12	208.53 ± 92.71	P=0.38
Hg grams per deciliter	11.95 ± 3.23	12.30 ± 2.31	P=0.57
≥ 60 yrs	12.41 ± 3.06	12.01 ± 2.03	P=0.32
<60 yrs	11.08 ± 3.52	12.53 ± 2.49	P=0.09
Cr milligrams per deciliter	1.63 ± 1.15	1.43 ± 1.60	P=0.03
≥ 60 yrs	1.63 ± 1.23	1.26 ± 0.76	P=0.24
<60 yrs	1.63 ± 1.03	1.57 ± 2.03	P=0.14
Urea milligrams per deciliter	80 ± 62.77	45.11 ± 27.09	P<0.001
≥ 60 yrs	81.09 ± 59.79	49.61 ± 25.32	P=0.01
<60 yrs	78.08 ± 58.50	41.35 ± 27.92	P<0.001
ESR mm/hr	56.93 ± 31.10	48.53 ± 31.53	P=0.12
≥ 60 yrs	60.35 ± 53.00	47.72 ± 31.14	P=0.12
<60 yrs	51.25 ± 30.29	40.10 ± 31.83	P=0.64
Na mEq/L	137.12 ± 5.20	136.18 ± 3.78	P=0.31
≥ 60 yrs	137.76 ± 5.17	136.38 ± 3.89	P=0.22
<60 yrs	136.00 ± 5.29	136.07 ± 3.74	P=0.91
K mEq/L	4.32 ± 0.71	4.15 ± 0.52	P=0.26
≥ 60 yrs	4.22 ± 0.70	4.17 ± 0.57	P=0.92
<60 yrs	4.50 ± 0.73	4.13 ± 0.48	P=0.06
CRP mg/L	128.85 ± 89.41	88.50 ± 64.46	P=009
≥ 60 yrs	136.67 ± 85.81	84.33 ± 60.50	P=0.01
<60 yrs	115.83 ± 97.53	91.35 ± 67.42	P=0.32

RR: Respiratory rate, HR: Heart rate, T: Temperature, O2 sat: O2 saturation, WBC: White blood cell, Neu: Neutrophil, Lymph: Lymphocyte, PLC: Platelet, Hg: Hemoglobin, Cr: Creatinine, ESR: erythrocyte sedimentation rate, CRP: high-sensitivity C-reactive protein.

*Mann-Whitney test was used.

Table 3. Comparative laboratory results of patients with and without ECG changes

Parameters (mean ± SD)	With ECG changes	Without ECG	p-value *
RR (N/min)	23.92 ± 7.98	23.97 ± 6.28	0.447
HR (beat/min)	98.29 ± 21.80	91.25 ± 15.93	0.009
T (Centigrade)	37.27 ± 1.03	37.31 ± 0.92	0.526
O2 sat (Percent)	85.05 ± 9.33	87.60 ± 8.46	007
WBC (per microliter)	9949.12 ± 4557.71	8544.42 ± 4734.44	0.013
Neu (per microliter)	78.77 ± 13.44	74.78 ± 12.45	0.011
Lymph (per microliter)	14.69 ± 12.03	18.38 ± 10.05	0.001
PLC (per microliter)	211.10 ± 95.17	208.39 ± 80.26	0.779
Hg (grams per deciliter)	12.20 ± 2.68	12.30 ± 2.18	0.671
Cr (milligrams per deciliter)	1.64 ± 1.73	1.22 ± 1.18	0.003
Urea (milligrams per deciliter)	58.12 ± 42.63	40.76 ± 26.28	<0.001
ESR (mm/hr)	51.06 ± 32.21	48.26 ± 30.53	0.586
Na (mEq/L)	136.35 ± 4.41	136.37 ± 3.55	0.782
K (mEq/L)	4.18 ± 0.65	4.16 ± 0.41	0.782
CRP (mg/L)	105.25 ± 79.09	80.71 ± 53.82	0.046

RR: Respiratory rate, HR: Heart rate, T: Temperature, O2 sat: O2 saturation, WBC: White blood cell, Neu: Neutrophil, Lymph: Lymphocyte, PLC: Platelet, Hg: Hemoglobin, Cr: Creatinine, ESR: erythrocyte sedimentation rate, CRP: high-sensitivity C-reactive protein.

*Mann-Whitney test was used.

Table 4. Multiple Logistic Regression of hospital mortality (using the Backward LR model) with various laboratory clinical findings

Parameters unit	Odds ratio	95% CI		p-value
		Lower	Upper	
O2 sat %	0.920	0.868	0.976	0.005
Lymph MCL	0.793	0.660	0.953	0.014
PLC MCL	0.993	0.987	0.999	0.034
ESR mm/hr	1.01	1.000	1.035	0.047
ECG changes	46.84	3.876	566.287	002
Age	1.03	1	1.06	0.04
Urea mg/dL	1.03	1.006	1.059	0.017

O2 sat: O2 saturation, Neu: Neutrophil, Lymph: Lymphocyte, PLC: Platelet, ESR: erythrocyte sedimentation rate, Cr: Creatinine, CI: Confidence interval.

The only mortality risk factor in nonelderly patients was lymphocyte count (Table 4a).

One of 33 patients who died of COVID-19 had a normal ECG (3%), whereas the remaining 32 patients (97%) had abnormal ECG (P<0.001). The frequency of non-sinus rhythm (P=0.056), arrhythmia (P=0.044), left bundle branch block (LBBB, P<0.001), inverted T wave in precordial leads (P=0.002), poor R progression (P=0.003), Low voltage QRS (P=0.006), tall R wave in V1 (P=0.025), and axis deviation (P=0.039) were significantly higher in patients who died of COVID-19 than patients who survived. Right bundle branch block (RBBB, P=0.593), ST elevation (P=0.420), ST depression (P=0.191), QT interval (P=0.102), left atrial enlargement (LAE, P=0.617), right atrial enlargement (RAE, P=0.636), left ventricular hypertrophy (LVH, P=0.877) and Q wave (P=0.653) did not differ between the two groups (Table 5).

More patients with ECG changes were admitted to the ICU than those with a normal ECG (34.5% vs. 14.3%, p=0.001).

On 116 patients with ECG changes, a multiple logistic regression analysis was performed to determine which ECG changes could be used to predict patient mortality. However, only LBBB (OR = 4.601, 95% CI: 1.357-15.6, p=0.014) was an independent ECG factor for predicting mortality in elderly patients.

Table 4a. Multiple Logistic Regression of in-hospital mortality by age group (using the Backward LR model) with laboratory and clinical findings

Age (yrs) N (%)	Parameters unit	Odds ratio	95% CI		p-value
			Lower	Upper	
	O2 sat %	0.79	0.691	0.912	0.001
≥ 60	Neu MCL	0.69	0.508	0.937	0.0١
96 (48%)	Lymph MCL	0.60	0.42	0.87	0.007
	PLC MCL	0.98	0.967	0.99٣	0.002
	ECG changes	227	7.193	7176.362	002
< 60	Lymph MCL	0.862	0.748	0.994	04
105 (52%)					

O2 sat: O2 saturation, Neu: Neutrophil, Lymph: Lymphocyte, PLC: Platelet, CI: Confidence interval.

Table 5. In-hospital mortality and different ECG changes in 201 COVID-19 patients.

Parameters	In-hospital mortality		Odds ratio	p-value
	Yes N=33	No N=168		
Non-SR N (%)	3 (42.9)	4(57.1)	4.08 (0.87, 19.14)	P=0.056*
Arrhythmia N (%)	6(33.3)	12(66.7)	2.87 (0.99, 8.3)	P=0.044*
RBBB N (%)	0(0)	5(100)	0.00	P=0.593ǂ
LBBB N (%)	7(53.8)	6(46.2)	7.22 (2.25, 23.19)	P<0.001*
ST elevation N (%)	1 (33.3)	2(66.7)	2.58 (0.23, 29.29)	P=0.420ǂ
ST depression N (%)	2 (40)	3 (60)	3.53 (0.57, 21.98)	P=0.191ǂ
Inverted T N (%)	7(43.8)	9(56.3)	4.73 (1.62, 13.8)	P=0.002*
QT interval	3 (73.1)	5(26.9)	3.24 (0.74, 14.28)	P=0.102*
Poor R progression N (%)	5(50)	5(50)	5.79 (1.57, 21.29)	P=0.003*
Low voltage	11(32.4)	23(67.6)	3.13 (1.34, 7.3)	P=0.006*
LA enlargement N (%)	8(19)	34(81)	1.25 (0.52, 3.02)	P=0.617*
RA enlargement N (%)	2(22.2)	7(77.8)	1.47 (0.29, 7.44)	P=0.636*
LVH N (%)	2(18.2)	9(81.8)	1.13 (0.23, 5.5)	P=0.877*
Tall R wave N (%)	3(50)	3(50)	5.47 (1.05, 28,34)	P=0.025*
Axis deviation N (%)	6(40)	9(60)	3.9 (1.28, 11.85)	P=0.039*
Q wave N (%)	2 (28)	5(72)	2.09 (0.39, 11.26)	P=0.653*

Non-SR: Non-sinus rhythm, RBBB: Right bundle branch block, LBBB: Left bundle branch block, LAE: left atrial enlargement, RAE: Right atrial enlargement, LVH: Left ventricular hypertrophy.
*Chi-Square;ǂFisher's Exact tests.

Limitations

Our research had several limitations. The current study was a retrospective, single-center study with a small patient population.

Discussion

COVID-19 can affect multiple organs, including the lungs, heart, liver, and kidneys.[1] COVID-19 can induce cardiac-related adverse events via multiple biological pathways, including the release of cytokines, stress-induced cardiomyopathy or arrhythmia resulting in myocarditis, and right-sided heart failure caused by pulmonary emboli or lung injury. These cardiac-related adverse conditions manifest as abnormal ECG changes.

Most cases of dyspnea are due to cardiac or pulmonary diseases, and ECG is an effective diagnostic tool for evaluating these patients. ECG is simple and quick to perform, has low operating costs, and poses a minimal risk of viral exposure to medical personnel.

Cardiac damage may worsen prognosis, whether in the presence or absence of other preexisting cardiovascular diseases.[9] COVID-19 may be the cause of all cardiovascular complications, or it may aggravate or amplify preexisting cardiac conditions. ECG may indicate a higher risk of mortality or morbidity

in this patient population.[10-12]

Most patients infected with COVID-19 may remain asymptomatic or demonstrate mild symptoms. However, COVID-19 may induce acute respiratory syndrome in nearly 15-20% of cases. In our study, 25.9% of patients required medical care in the intensive care unit (4). ECG changes were associated with a higher admission rate to the ICU than the other group (34.5% versus 14.3%, P-value=0.001).

Emam Reza hospital is considered a referral medical center for critically ill patients diagnosed with COVID-19. Although there is no statistical significance, our study's morbidity rate of 16.4% (33 cases) was comparable to a previous study by Elias et al. that found a mortality rate of 18% in COVID-19 patients.[13]

Patients with ECG changes had a higher mortality rate than those without (27.6% versus 1.2%, P-value<0.001). Angeli et al. showed that the length of in-hospital stay could be adversely affected by the development of changes in the ECG.[14]

The incidence of arrhythmia (P=0.04), left bundle branch block (P<0.001), right ventricular strain (P<0.001), poor R progression (P<0.001), Low voltage QRS (P<0.001), tall R wave in V1 (P=0.02) and axis deviation (P=0.03) were significantly higher in patients who died of COVID-19 than patients who survived. However, only LBBB was an independent ECG factor for predicting mortality among all ECG changes (OR=4.6%, P value=0.014).

According to another study, atrial fibrillation/flutter, right ventricular strain, and ST segment abnormalities were highly predictive of 48-hour outcomes.[13] Moey et al. hypothesized that ORS widening could indicate myocardial damage in those admitted to the ICU with elevated troponin-I.[15]

Multivariate analysis of different demographic data, laboratory findings and mortality, lower O2 saturation percentage (OR=0.92), neutrophil, platelet, lymphocyte count (OR=0.879, 0.793, 0.993) and higher ESR, age, and urea (OR=1.01, 1.03, 1.03) could be predictors for death. Changes in the ECG were the most significant predictor of death compared to other data (OR=46.84). In another study, ECG changes, respiratory rate, and O2 saturation<95% were the most significant predictors of patient mortality.[13]

ECG is regarded as an essential initial test for evaluating patients with dyspnea. A correct interpretation of the ECG, especially in the emergency department, could be the basis for immediate triage and risk stratification of COVID-19 patients.[16]

Conclusion

In COVID-19-infected patients with ECG changes, the incidence of mortality and ICU admissions was greater than in patients without ECG changes. Due to the widespread availability of ECG within emergency departments, COVID-19-positive patients should undergo ECG for triage and risk stratification. In addition, certain ECG changes with adverse cardiac outcomes, such as LBBB, are identified as predictors of patient mortality.

Ethical statements

Approval for accessing the patient health records was obtained from the local research ethics committee (IR.MUMS.REC.1399.116), and informed consent was obtained from all patients.

Conflict of Interest

The authors declare no conflict of interest.

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Authors' Contribution

All co-authors contributed and participated in the main design and revision of the

manuscript.

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