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ORIGINAL ARTICLE

Evaluation of Changes in Electrocardiography and Echocardiography during the Course of Infection in COVID-19 Disease

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ABSTRACT

Background: There are high impact of COVID-19 disease on cardiovascular system in either acute or recovery phase. The cardiac complications of COVID-19 are easily reflected by electrocardiography and echocardiography.
Aim: To predict significant cardiovascular affection throughout course of COVID-19 infection using electrocardiography and echocardiography. .
Methods: This retrospective cohort study was conducted at Cardiology Department, Faculty of Medicine and isolation department at Zagazig University Hospitals during the period of 2021 to 2022 on 100 cases with suspected or confirmed COVID-19. All patients were subjected to electrocardiographic and Echocardiographic assessment.
Results: QTc and number of patients with abnormal ECG were significantly higher in severe group compared to non-severe group. PR interval was significantly higher in severe group compared to non-severe group. LV mass, EF, E, and E/A ratio were significantly lower in severe group compared to non-severe group. LVEDD, A, number of patients with diastolic dysfunction and number of patients with pericardial effusion were significantly higher in severe group compared to non-severe group ($P<0.05$). Valvular lesion was significantly different between both groups.
Conclusions: Patients with severe COVID-19 disease exhibit significantly worse cardiac pathology across structural, systolic, and diastolic functions compared to non-severe disease. Patients with severe disease showed markers of myocardial injury, elevated cardiac biomarkers, ECG changes, impaired left ventricular function, and higher rates of arrhythmias, valvular lesions, pericardial effusion, and diastolic dysfunction.
Keywords: Electrocardiographic; Echocardiographic;COVID-19

INTRODUCTION

Cases of viral respiratory illnesses first surfaced in Wuhan, China in December 2019, and within a few weeks, the infections spread throughout the world. By March 2020, the World Health Organization had dubbed the infection coronavirus disease (COVID-19) and proclaimed a pandemic **[1].** Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) infection manifests clinically as respiratory symptoms such as fever, cough, dyspnea, and exhaustion. The infection can also cause pneumonia, acute respiratory distress syndrome (ARDS), and shock **[2].** "Right ventricular (RV) damage is prevalent in patients with COVID-19 and myocardial injury, while left ventricular damage is relatively rare and lacks specificity," according to a study on severe respiratory infections caused by COVID-19. Increased RV afterload and decreased RV contractility brought on by a number of conditions, including autoimmune injury, pulmonary thrombosis, acute respiratory distress syndrome, direct viral injury, hypoxia, and inflammatory response, could be the processes causing RV damage. In COVID-19 patients, RV dysfunction typically denotes a poor clinical outcome **[3].** Frequent medications used in the treatment and prevention of COVID-19 include azithromycin and hydroxychloroquine. It is well known that both medications increase the risk of torsades des pointes and extend the QT interval. Likewise, QT and PR

prolongation may result from lopinavir/ritonavir **[4].**

Five forms of cardiovascular problems are associated with COVID-19: arrhythmia, new-onset or worsening of pre-existing heart failure, thromboembolic disease, cardiac abnormalities generated by medical treatment, and cardiac damage (mostly due to ischemia or myocarditis) **[1].** Because of its widespread accessibility, low cost, and potential for remote monitoring, the electrocardiogram (ECG) is one of the most effective instruments in this context for evaluating the degree of cardiac involvement in COVID-19 patients and assessing the impact of drugs. In light of this, we suggested reviewing the function of the ECG in determining cardiac involvement in COVID-19 and highlighting pertinent clinical implications **[1].** In the early stages of COVID-19 infection, trans-thoracic echocardiography is another diagnostic technique that can be used to examine cardiac structural damage and evaluate heart function. This can help to lower the risk of heart injury while also avoiding cardiac problems and giving early therapy **[5].**

METHODS

This retrospective cohort study was conducted at Cardiology Department, Faculty of Medicine and isolation department at Zagazig University Hospitals during the period of 2021 to 2022 on 100 cases with suspected or confirmed COVID-19. Written informed consent was obtained from all participants, the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University (IRB number 9437). The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Those with COVID-19 who were older than 18 and either had a positive nasopharyngeal swab confirmed by RT-PCR or were highly suspected of having the virus based on their history, clinical symptoms, and imaging results were included in the study.

Patient with recent non Covid-19 infection, pregnant females, known patient with coronary artery disease or other cardiac disease, patients with cardiac muscle disorders, significant valvular diseases, known patient with liver and kidney impairment, patients suffering from electrolytes imbalance (hypokalemia, hypomagnesemia and hypocalcemia), malignancies or inflammatory diseases and patients diagnosed with atrial fibrillation before COVID-19 exposure, primary pulmonary artery hypertension (by exclusion groups from 2-5), asthma and chronic obstructive air way were excluded from the study. Specific clinical indicators, such as breathing more than 30 times per minute, oxygen saturation less than 94%, and lung infiltrates more than 50% as shown on radiographs, are used to diagnose severe COVID-19. A detailed history, a clinical examination, and laboratory tests including a complete blood count (CBC), creatinine and urea, serum ferritin, C-reactive protein (CRP), D-dimer, serum troponin, and serum electrolytes, sodium, and potassium, were performed on all patients.

***ECG at admission and after recovery of symptoms*:** Prior to beginning COVID-19 medication, twelve lead electrocardiograms (ECGs) of the patients were obtained while they were at rest and in a supine posture. The ECG was recorded at a rate of 25 mm/s, with a calibration of 1 mV/cm and a filter setting of 0.05–150 Hz. Every measurement was manually completed on the screen. Every patient had sinus rhythm. The time period between the start of the QRS complex and the conclusion of the T wave was designated as the QT interval. All leads had their QT intervals measured, and the longest QT interval was noted. The difference between the greatest and minimum QT interval in several leads was used to calculate QT dispersion, or QTd. The heart rate was calculated using the measured R-R interval.Using Bazett's formula, the corrected QT dispersion (QTdc) and correct QT interval (QTc) were determined: QT$\sqrt{}$(R-R interval) = QTc **[6].** The following parameters were measured: Cornell voltage (mm), ST-T anomalies present, PR interval (from the start of the P wave to the end of the R wave), and QRS (from the start of the Q wave to the end of the S wave) **[2].**

***Transthoracic echocardiography*:**

Transthoracic echocardiography was performed on admission and after recovery based on improvement of symptoms and investigation and /or negative nasopharygeal swab**.** The studies were done on GE Vivid7 digital ultrasonography system (General Electric Company, Milwaukee, WI, USA), with a phased-array transducer operating at 2.5–5.0 MHz and Hitachi Aloka ultrasound device machine S21 single crystal transducer (Hitachi company, japan).

Measurements:

- LV mass, LV ejection fraction (LVEF), and left ventricle end diastolic diameter (LVEDD) are all measured. A systolic ejection fraction of 50% or less was considered impaired LVEF **[7].**
- Presence of wall motion abnormality.

- Pulsed wave Doppler(PWD) E/A , deceleration time E/e.
- M-mode echocardiography offers a single line of information at a greater frame rate than two-dimensional echocardiography, and is widely used to assess left ventricular function. Fractional shortening, or diastolic dimension minus systolic dimension divided by diastolic dimension (normal 28% to 40%), is one of the M-mode measurements of function **[8].**
- Right ventricular function assessment by visual and the assessment of tricuspid annular plane systolic excursion (TAPSE) and right ventricular fractional area change (RV-FAC)**[7].**
- Assessing the diastolic function through the assessment of transmitral flow parameters, such as the E/A ratio, the E deceleration time (DT), and the early (E) and late (A) diastolic filling velocities, from an apical four chamber view using standard pulsed wave Doppler, the diastolic function is first categorized as either restricted, pseudonormal, normal, or impaired relaxation **[9].**
- Check for any valvular lesion or pericardial effusion, can be developed during the infection course.

All patient were subjected to calculating the left ventricular ejection fraction (LVEF%) using the Simpson method, and evaluating the mitral inflow and tissue Doppler annulus velocities and LA volume to determine the LV diastolic function.

***Follow up*:** by electrocardiography and echocardiography until discharge.

STATISTICAL ANALYSIS

Statistical analysis was done by SPSS v28 (IBM©, Chicago, IL, USA). Unpaired student t-test, Chi-square test, Fisher's exact test, Kaplan Meier curve and multiple logistic regression were used.

RESULTS

Age, BMI, breathing difficulties, and vital signs were all significantly different between the severe and non-severe groups, with a P-value of less than 0.001. The severe group's blood saturation of oxygen was significantly lower than that of the non-severe group (P<0.001). **[**Error! Reference source not found.**1]**

Platelet count was significantly lower in severe group compared to non-severe group. WBCs count, lymphocyte count, serum ferritin, CRP, the severe group's levels of LDH and d-dimer were considerably greater than those of the non-severe group. Cardiac biomarkers (Troponin-I and CK-MB) and serum creatinine levels in the severe group were significantly higher than in the non-sever group. **[Table 2]**.

Regarding the ECG assessment, QTc and number of patients with abnormal ECG were notably greater in the severe group as opposed to the non-severe group. In comparison to the non-severe group, the severe group's PR interval was significantly longer. Regarding the abnormal ECG findings, anteroseptal T wave inversion was detected in 14 (21.9%) patients in severe group and 2 (5.6%) patients in non-severe group with statistically significant difference **[Table 3]**

The severe group had considerably decreased LV mass, EF, E, and E/A ratio in comparison to the non-severe group. The number of patients with pericardial effusion, diastolic dysfunction, LVEDD, and A were all considerably greater in the severe group as compared to the non-severe group (P<0.05). There was a substantial difference in the valve lesion between the two groups **[**

Table 4]

When compared to the non-severe group, the severe group's hospital stay was noticeably longer. Compared to the non-severe group, the severe group had a considerably greater incidence of DVT and pulmonary embolism. When comparing the severe group to the non-severe group, mortality was considerably greater (P=0.002). **[**Error! Reference source not found.**5]**

On univariate logistic regression analysis, age, BMI, shortness of breath, SBP, DBP, HR, oxygen saturation, lymphocyte, serum ferritin, CRP, d-dimer, troponin, CK-MB, EF, E/A ratio, P wave, ECG (normal/abnormal), anteroseptal T wave inversion, STEMI/NSTEMI, diastolic function, valvular lesion, DVT and PE were significant predictors for the incidence of MACCE as shown in **Table 6.**

On Multivariate logistic regression analysis, BMI, shortness of breath, lymphocyte, serum ferritin, CK-MB, EF, E/A ratio, ECG (normal/abnormal), anteroseptal T wave inversion, diastolic function,

DVT and PE were significant predictors for the incidence of complications as shown in **(table 7)**

Table 1: Baseline characteristics of the studied groups

		Total (n=100)	Severe (n=64)	Non-severe (n=36)	P value
Age (years)	**Mean ± SD**	60.24 ± 11.26	67.81 ± 4.98	46.78 ± 5.16	**<0.001***
	Range	40 – 75	60 – 75	40 - 55	
		Total (n=100)	**Severe (n=64)**	**Non-severe (n=36)**	**P value**
Sex	**Male**	69 (69%)	47 (73.4%)	22 (61.1%)	0.292
	Female	31 (31%)	17 (26.6%)	14 (38.9%)	
BMI (Kg/m^2)	**Mean ± SD**	24.75 ± 2.72	25.56 ± 2.58	23.32 ± 2.37	**<0.001***
	Range	21 - 33.41	22.1 - 32.2	21 - 33.41	
Residence	**Rural**	56 (56%)	37 (57.8%)	19 (52.8%)	0.782
	Urban	44 (44%)	27 (42.2%)	17 (47.2%)	
Fever		66 (66%)	43 (67.2%)	23 (63.9%)	0.909
Cough		71 (71%)	45 (70.3%)	26 (72.2%)	0.840
Shortness of breath		68 (68%)	53 (82.8%)	15 (41.7%)	**<0.001***
SBP (mmHg)	**Mean ± SD**	140.1 ± 13.0	145.8 ± 10.05	130 ± 11.71	**<0.001***
	Range	110 - 160	130 - 160	110 - 150	
DBP (mmHg)	**Mean ± SD**	75.0 ± 10.44	78.59 ± 10.21	68.61 ± 7.62	**<0.001***
	Range	60 – 100	60 - 100	60 - 80	
HR (beats/min)	**Mean ± SD**	77.13 ± 8.21	80.09 ± 8.52	71.86 ± 4.14	**<0.001***
	Range	65 – 95	65 - 95	65 - 79	
RR (breath/min)	**Mean ± SD**	21.79 ± 1.73	22.45 ± 1.14	20.61 ± 2	**<0.001***
	Range	18 – 24	21 - 24	18 - 24	
Oxygen saturation (%)	**Mean ± SD**	95.31 ± 1.48	84.59 ± 1.12	96.58 ± 1.18	**<0.001***
	Range	75 – 98	75 - 87	95 - 98	
Temperature (° C)	**Mean ± SD**	37.13 ± 0.39	37.21 ± 0.41	36.99 ± 0.33	**0.009***
	Range	36.5 - 37.9	36.5 - 37.9	36.5 - 37.4	

BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate, RR: respiratory rate, *: statistically significant as P value <0.05

Table 2: Laboratory investigations of the studied groups

		Total (n=100)	Severe (n=64)	Non-severe (n=36)	P value
Hb (g/dL)	**Mean ± SD**	11.03 ± 1.37	11.13 ± 1.39	10.85 ± 1.34	0.337
	Range	8.9 - 13.8	8.9 - 13.8	9 - 13.7	
PLT (*10^3 cell /µL)	**Mean ± SD**	235.7 ± 42.7	216.6 ± 27.41	269.64 ± 44.7	**<0.001***
	Range	173 - 348	173 – 260	205 - 348	
WBCs (*10^3 cell /µL)	**Mean ± SD**	12.68 ± 3.31	13.68 ± 3.64	10.89 ± 1.53	**<0.001***
	Range	8.2 - 19.8	8.3 - 19.8	8.2 - 13.2	
Lymphocyte (µL)	**Mean ± SD**	1.26 ± 0.43	1.56 ± 0.15	0.73 ± 0.19	**<0.001***
	Range	0.36 - 1.77	1.24 - 1.77	0.36 - 1.1	
Serum ferritin (µg/L)	**Mean ± SD**	870.2±299.1	1073.4± 153.6	506.56 ± 57.43	**<0.001***
	Range	401 - 1362	814 - 1362	401 – 597	

CRP (mg/dL)	**Mean ± SD**	187.8 ± 50.9	220.6 ± 26.96	129.57 ± 25.02	**<0.001***
	Range	80 - 268.3	180 - 268.3	80 - 163.7	
d-dimer (mmol/L)	**Mean ± SD**	245.74 ± 81	893.6± 108.04	255.83 ± 91.37	**<0.001***
	Range	107 - 397	700 - 1093	113 – 397	
LDH (U/L)	**Mean ± SD**	494.6±157.1	604.9 ± 54.37	298.5 ± 57.78	**<0.001***
	Range	212 - 696	506 - 696	212 – 398	
Troponin-I (ng/L)	**Mean ± SD**	2.08 ± 2.05	3.19 ± 1.79	0.11 ± 0.24	<0.001*
	Range	0.04 - 6.06	0.11 - 6.06	0.04 - 1.2	
		Total (n=100)	**Severe (n=64)**	**Non-severe (n=36)**	**P value**
CK-MB (U/L)	**Mean ± SD**	28.61 ± 7.32	32.91 ± 4.8	20.41 ± 4.07	**<0.001***
	Range	14 – 40	25 - 40	14 – 27	
S. creatinine (mg/dL)	**Mean ± SD**	0.94 ± 0.16	0.96 ± 0.16	0.89 ± 0.16	**0.045***
	Range	0.7 - 1.2	0.7 - 1.2	0.7 - 1.2	
Urea (mg/dL)	**Mean ± SD**	44.9± 13.33	45.2 ± 13.99	44.5 ± 12.44	0.803
	Range	20 – 65	20 - 65	20 – 62	
Serum calcium (mg/dL)	**Mean ± SD**	8.72 ± 0.71	8.72 ± 0.67	8.72 ± 0.79	0.980
	Range	7.5 - 9.8	7.5 - 9.8	7.5 - 9.8	
Serum sodium (mEq/L)	**Mean ± SD**	138.45 ± 3.98	138.81 ± 4.05	137.81 ± 3.87	0.229
	Range	132 - 145	132 - 145	132 – 144	
Serum potassium (mEq/L)	**Mean ± SD**	3.95 ± 0.36	3.98 ± 0.36	3.91 ± 0.36	0.418
	Range	3.4 - 4.5	3.4 - 4.5	3.4 - 4.5	
ALT (U/L)	**Mean ± SD**	21.04 ± 4.5	20.64 ± 4.74	21.75 ± 4.07	0.241
	Range	15 – 30	15 - 30	16 – 29	
AST (U/L)	**Mean ± SD**	32.58 ± 7.53	32.11 ± 7.33	33.42 ± 8	0.410
	Range	20 – 45	20 - 45	21 – 45	
Serum bilirubin (µmol/L)	**Mean ± SD**	1.11 ± 0.08	1.11 ± 0.08	1.11 ± 0.09	0.808
	Mean ± SD	1.11 ± 0.08	1.11 ± 0.08	1.11 ± 0.09	

CBC: complete blood count, Hb: haemoglobin, PLT: platelet count, CRP: C-reactive protein, LDH: lactate dehydrogenase, CK-MB: creatine kinase-myocardial band, ALT: alanine aminotransferase, AST: aspartate aminotransferase, *: statistically significant as P value <0.05.

Table 3: ECG assessment and abnormal finding of the studied groups

		Total (n=100)	**Severe (n=64)**	**Non-severe (n=36)**	**P value**
P wave	**Normal**	5 (5%)	4 (6.3%)	1 (2.8%)	0.651
	Abnormal	95 (95%)	60 (93.8%)	35 (97.2%)	
PR interval (ms)	**Mean ± SD**	152.9±32.91	162.96±	135±18.89	**<0.001***
	Range	110-280	110-280	120-200	
QRS (s)	**Mean ± SD**	0.12 ± 0.01	0.12 ± 0.01	0.12 ± 0.01	0.179
	Range	0.11 - 0.13	0.11 - 0.13	0.11 - 0.13	
QTc (ms)	**Mean ± SD**	461.9 ± 46.04	480.5 ± 47.3	428.8 ± 16.2	**<0.001***
	Range	404 - 560	406 - 560	404 - 460	
QT (ms)	**Mean ± SD**	383.63 ± 44.24	384.6 ± 44.8	381.9 ± 44.41	0.771
	Range	310 - 457	310 - 457	321 - 455	
Q wave		3 (3%)	1 (1.6%)	2 (5.6%)	0.293
T wave inversion		16 (16%)	14 (21.9%)	2 (5.6%)	**0.045***
STEMI		27(27%)	22(34%)	5(13.88%)	0.483
NSTEMI		7(7%)	7(11%)	0(0%)	0.694
ECG	**Normal**	46 (46%)	15 (23.4%)	31 (86.1%)	**<0.001***
	Abnormal	54 (54%)	49 (76.6%)	5 (13.9%)	

ECG: electrocardiogram, STEMI: ST elevation myocardial infarction, NSTEMI: non- ST elevation myocardial infarction *: statistically significant as P value <0.05.

Table 4: Echocardiography of the studied groups

		Total (n=100)	Severe (n=64)	Non-severe (n=36)	P value
LV mass (g/m^2)	**Mean ± SD**	92.81 ± 3.92	92.06 ± 4.24	94.14 ± 2.93	**0.011***
	Range	85 – 99	85 - 99	90 - 99	
LVEDD (mm)	**Mean ± SD**	44.6 ± 2.13	45.78 ± 1.62	42.5 ± 1.06	**<0.001***
	Range	41 – 48	43 - 48	41 - 44	
EF %	**Mean ± SD**	57.1 ± 5.32	54.45 ± 3.9	61.81 ± 4.22	**<0.001***
	Range	48 – 68	48 - 62	55 - 68	
E (cm/s)	**Mean ± SD**	70.79 ± 11.49	64.14 ± 7.69	82.61 ± 6.8	**<0.001***
	Range	51 – 95	51 - 76	71 - 95	
A (cm/s)	**Mean ± SD**	63.91 ± 7.78	67.09 ± 7.19	58.25 ± 5.33	**<0.001***
	Range	50 – 79	54 - 79	50 - 66	
E/A ratio	**Mean ± SD**	1.13 ± 0.27	0.97 ± 0.14	1.43 ± 0.18	**<0.001***
	Range	0.66 - 1.9	0.66 - 1.25	1.11 - 1.9	
TAPSE (mm)	**Mean ± SD**	17.62 ± 1.17	17.5 ± 1.13	17.83 ± 1.25	0.176
	Range	16 – 19	16 - 19	16 - 19	
FAC %	**Mean ± SD**	44.72 ± 5.29	45.22 ± 5.07	43.83 ± 5.69	0.212
	Range	36 – 54	36 - 54	36 - 54	
Diastolic function	**Dysfunction**	11 (11%)	11 (17.19%)	0 (0%)	**0.007***
	Normal	89 (89%)	53 (82.81%)	36 (100%)	
Pericardial effusion		17 (17%)	15 (23.44%)	2 (5.56%)	**0.026***
Valvular lesion	**MR**	14 (14%)	10 (15.6%)	4 (11.1%)	**0.013***
	TR	9 (9%)	8 (12.5%)	1 (2.8%)	
	AR	12 (12%)	11 (17.2%)	1 (2.8%)	

LV: left ventricle, LVEDD: left ventricular end diastolic diameter, EF: ejection fraction, E/A: early to atrial filling velocity ratio, TAPSE: tricuspid annular plane systolic excursion, FAC: fractional area change, MR: mitral regurgitation, TR: tricuspid regurgitation, AR: aortic regurgitation, *: statistically significant as P value <0.05.

Table 5: Hospital stay of the studied groups

		Total (n=100)	Severe (n=64)	Non-severe (n=36)	P value
Hospital stay (days)	**Mean ± SD**	16.51 ± 3.49	21.17 ± 3.81	12.1 ± 2.4	**<0.001***
	Range	5 - 25	10 - 25	5 - 15	
DVT		14 (14%)	13 (20.3%)	1 (2.8%)	0.016*
Pulmonary embolism		13 (13%)	12 (18.8%)	1 (2.8%)	**0.029***
Myocardial infarction		9 (9%)	7 (10.9%)	2 (5.6%)	0.482
Mortality	**Died**	14 (14%)	14 (21.9%)	0 (0%)	**0.002***
	Alive	86 (86%)	50 (78.1%)	36 (100%)	

DVT: deep vein thrombosis, *: statistically significant as P value <0.05

Table 6: Univariate logistic regression analysis for prediction of MACCE

	Coefficient	SE	P	Odds ratio	95% CI
Age (years)	0.071	0.024	**0.003***	1.074	1.024 to 1.126
BMI (Kg/m^2)	0.305	0.089	**0.002***	1.357	1.137 to 1.618
Shortness of breath	-1.789	0.655	**0.006***	0.167	0.046 to 0.604
SBP (mmHg)	0.042	0.018	**0.023***	1.043	1.005 to 1.083
DBP (mmHg)	0.052	0.022	**0.018***	1.054	1.009 to 1.101
HR (beats/min)	0.057	0.027	**0.033***	1.059	1.004 to 1.117
RR (breath/min)	0.227	0.138	0.101	1.255	0.956 to 1.648
Oxygen saturation (%)	-0.378	0.163	**0.020***	0.684	0.497 to 0.942
Temperature (°c)	0.386	0.560	0.491	1.471	0.490 to 4.410
Hb (g/dL)	0.083	0.160	0.605	1.086	0.793 to 1.489
PLT (*10^3 cell /µL)	-0.0066	0.005	0.236	0.993	0.982 to 1.004
WBCs (*10^3 cell /µL)	0.0100	0.066	0.880	1.010	0.886 to 1.150
Lymphocyte (µL)	2.213	0.704	**0.002***	9.144	2.297 to 36.39
Serum ferritin (µg/L)	0.003	0.001	**<0.001***	1.003	1.001 to 1.005
CRP (mg/dL)	0.013	0.005	**0.006***	1.013	1.003 to 1.024
d-dimer	0.004	0.002	**0.034***	1.004	1.000 to 1.007
Troponin (ng/L)	0.280	0.109	**0.010***	1.324	1.068 to 1.640
CK-MB	0.113	0.036	**0.002***	1.119	1.043 to 1.201
S. creatinine (mg/dL)	2.797	1.438	0.052	16.41	0.97 to 275.01
EF %	0.152	0.050	**0.002***	0.859	0.778 to 0.947
LV mass	-0.093	0.056	0.101	0.910	0.814 to 1.018
LVDD	0.204	0.107	0.057	1.227	0.993 to 1.516
E/A ratio	-4.443	1.220	**<0.001***	0.011	0.001 to 0.128
TAPSE	0.179	0.191	0.347	1.196	0.822 to 1.740
P wave	-3.471	0.807	**<0.001***	0.031	0.006 to 0.151
PR interval (ms)	0.008	0.004	0.066	1.008	0.999 to 1.017
QRS (s)	-25.157	28.54	0.378	1.387	0.945 to 1.142
QTc (ms)	0.008	0.004	0.084	1.008	0.998 to 1.017
QT (ms)	-0.001	0.004	0.689	0.998	0.988 to 1.007
ECG	-1.90	0.496	**<0.001***	0.149	0.056 to 0.394
Anteroseptal T wave inversion	-3.471	0.807	**<0.001***	0.031	0.006 to 0.151
STEMI/NSTEMI	-1.904	0.496	**<0.001***	0.149	0.056 to 0.394
Diastolic function	-3.606	1.080	**0.001***	0.027	0.003 to 0.225
Valvular lesion	0.579	0.203	**0.004***	1.785	1.197 to 2.662
DVT	-4.040	1.074	**<0.001***	0.017	0.002 to 0.144
PE	-3.900	1.075	**<0.001***	0.0202	0.002 to 0.166

BMI: body mass index, **SBP**: systolic blood pressure, **DBP**: diastolic blood pressure, **HR**: heart rate, **RR**: respiratory rate, **Hb:** haemoglobin, **PLT**: platelet count, **CRP**: C-reactive protein, **CK-MB**: creatine kinase-myocardial band, **EF**: ejection fraction**, LV**: left ventricle, **E/A**: early to atrial filling velocity ratio, **TAPSE**: tricuspid annular plane systolic excursion, **STEMI**: ST elevation myocardial infarction, **NSTEMI:** non- ST elevation myocardial infarction, **DVT:** deep vein thrombosis, **PE:** pulmonary effusion, **SE:** standard error, **CI:** confidence interval, *: statistically significant as P value <0.05

Table 7: Multivariate logistic regression analysis for prediction of MACCE

	Coefficient	SE	P	Odds ratio	95% CI
Age (years)	0.020	0.045	0.652	1.021	0.934 to 1.114
BMI (Kg/m^2)	0.265	0.103	**0.010***	1.303	1.065 to 1.593
Shortness of breath	-1.327	0.674	**0.049***	0.265	0.070 to 0.994
SBP (mmHg)	0.025	0.026	0.352	1.025	0.973 to 1.079
DBP (mmHg)	0.024	0.030	0.414	1.025	0.966 to 1.086
HR (beats/min)	0.059	0.037	0.111	1.061	0.986 to 1.140
RR (breath/min)	-0.098	0.206	0.634	0.907	0.605 to 1.357
Oxygen saturation (%)	-0.249	0.249	0.316	0.779	0.478 to 1.269
Temperature (°c)	-0.390	0.710	0.583	0.677	0.168 to 2.722
Hb (g/dL)	0.172	0.206	0.403	1.188	0.793 to 1.778
PLT (*10^3 cell /µL)	0.010	0.009	0.263	1.010	0.992 to 1.027
WBCs (*10^3 cell /µL)	-0.076	0.071	0.284	0.9262	0.805 to 1.065
Lymphocyte (µL)	2.411	0.727	**0.001***	11.149	2.679 to 46.39
Serum ferritin (µg/L)	0.003	0.001	**<0.001***	1.0034	1.001 to 1.005
CRP (mg/dL)	-0.013	0.011	0.238	0.987	0.966 to 1.008
d-dimer	0.004	0.002	0.101	1.004	0.999 to 1.008
Troponin (ng/L)	-0.010	0.178	0.957	0.990	0.699 to 1.402
CK-MB	0.133	0.063	**0.036***	1.142	1.008 to 1.293
S. creatinine (mg/dL)	2.4209	1.570	0.123	11.256	0.51 to 244.64
EF %	-0.186	0.080	**0.020***	0.831	0.710 to 0.971
LV mass	0.029	0.083	0.727	1.030	0.874 to 1.211
LVDD	-0.066	0.171	0.698	0.936	0.669 to 1.308
E/A ratio	-4.957	1.852	**0.008***	0.007	0.002 to 0.265
TAPSE	0.238	0.272	0.382	1.269	0.744 to 2.162
P wave	-21.495	9627.337	0.998	0.000	0.000
PR interval (ms)	-0.003	0.007	0.670	0.997	0.983 to 1.010
QRS (s)	-61.099	41.770	0.144	0.000	0.000
QTc (ms)	-0.004	0.007	0.563	0.996	0.982 to 1.009
QT (ms)	-0.003	0.007	0.618	0.997	0.983 to 1.010
ECG	-2.114	0.726	**0.004***	0.121	0.029 to 0.501
Anteroseptal T wave inversion	-3.228	0.826	**<0.001***	0.0396	0.0078 to 0.2000
STEMI/NSTEMI	0.667	1.097	0.542	1.950	0.226 to 16.76
Diastolic function	-3.243	1.089	**0.003***	0.039	0.004 to 0.330
Valvular lesion	0.294	0.246	0.232	1.342	0.827 to 2.177
DVT	-4.040	1.074	**<0.001***	0.0176	0.002 to 0.144
PE	-2.648	0.886	**0.003***	0.070	0.012 to 0.401

BMI: body mass index, **SBP**: systolic blood pressure, **DBP**: diastolic blood pressure, **HR**: heart rate, **RR**: respiratory rate, **Hb:** haemoglobin, **PLT**: platelet count, **CRP:** C-reactive protein**, CK-MB**: creatine kinase-myocardial band, **EF**: ejection fraction**, LV**: left ventricle, **E/A:** early to atrial filling velocity ratio, **TAPSE**: tricuspid annular plane systolic excursion, **STEMI**: ST elevation myocardial infarction, **NSTEMI**: non- ST elevation myocardial infarction**, DVT:** deep vein thrombosis, **PE**: pulmonary effusion, **SE**: standard error, **CI:** confidence interval, *: statistically significant as P value <0.05

DISCUSSION

In our study, regarding the ECG assessment, QTc and number of patients with abnormal ECG were notably greater in the severe group as opposed to the non-severe group. PR interval was notably greater in the severe group as opposed to the non-severe group. Regarding the abnormal ECG findings, anteroseptal T wave inversion was detected in 14 (21.9%) patients in severe group and 2 (5.6%) patients in non-severe group with statistically significant difference.

Numerous investigations have documented a range of arrhythmias and abnormalities in the ECG that can arise in severe COVID-19 cases [10, 11]. Critically sick individuals are more likely to experience arrhythmias and ECG abnormalities [12, 13]. These events can happen in 33–93% of these cases. Atrial fibrillation/flutter was observed in 10.1% of patients during hospitalization and in 14.3% of patients upon admission, according to a study done in New York hospitals [14].

More than 13% of individuals with COVID-19 infection may experience QT interval prolongation. Chloroquine, hydroxychloroquine, and azithromycin are among the drugs that may cause this [15, 16]. The most frequent anomaly in patients needing to be admitted to the intensive care unit is T wave and ST segment alterations [17]. Our study demonstrates that patients with severe COVID-19 tended to have significantly worse left ventricular pathology across structural, systolic, and diastolic parameters compared to non-severe disease. Specifically, patients with severe disease had increased LV mass, higher LVEDD, reduced LV EF%, lower mitral E velocities, elevated A velocities, and reduced E/A ratios indicative of diastolic dysfunction - which was also more prevalent in this group. Additionally, more patients with severe disease exhibited higher rates of valvular lesions and pericardial effusion. In contrast, right ventricular metrics including TAPSE and FAC% were similar between both groups, implying preserved RV systolic function despite higher overall disease severity.

This is in line with studies that involved one hundred post-discharge patients. According to **Kujur et al. [18],** the majority of LV dysfunction patients had moderate to severe disease severity. This is also in line with other research that demonstrated myocardial injury in hospitalized COVID-19 patients and discovered a strong correlation between the degree of disease severity and the compromised left ventricular function [19]. According to a study assessing LV function using 2D-STE in 100 hospitalized COVID-19 patients, severe cases had worse LV function as determined by LVGLS **[20].**

The right ventricle may sustain direct damage in severe COVID-19 infection cases, such as acute respiratory distress syndrome with potential pulmonary thrombosis. This can lead to decreased RV contractility and failure as a result of increased pulmonary vascular resistance and RV afterload **[21].** 479 patients, or 39% of the total, exhibited left ventricular anomalies, according to another study that involved 1216 patients [22]. Twenty.4% of patients had LV systolic dysfunction and 49 instances (34.5%) had RV systolic dysfunction [23]. Conventional and 2-dimensional speckle tracking was frequently used to evaluate right ventricular (RV) dysfunction, which is a strong independent predictor of death [24]. Additionally, a research found that identifying COVID-19 individuals who are more likely to die after hospitalization can be aided by echocardiographic evidence of RV systolic dysfunction [25].

Similarly, 2D-Echocardiography and layer-specific longitudinal strain were performed in a study including 218 patients, and the results demonstrated an association between the severity of the illness and the reduction in GLS values [26].

Patients with more severe illnesses tend to have larger RVs, and RV dilatation is a more common cardiac defect than LV or RV functional decline. In COVID-19 patients, changes in TAPSE, S', and RVFAC are common and predictive of death; however, relative retention of longitudinal shortening in RV dysfunction suggests that RVFAC is a more suitable marker of overall RV function [27].

In our study, the severe group's hospital stay was noticeably longer than that of the non-severe group (21.17 ± 3.81days vs 12.1 ± 2.4days). **Al Oweidat et al. [28]** found that the mean and standard deviation for the length of hospital stay were higher (10.09 ± 9.08)**.**

In our study, incidence of DVT and when compared to the non-severe group, the severe group experienced considerably more pulmonary emboli. Comparably, PE has been identified in numerous trials as a presenting problem and/or COVID-19 consequence, especially in patients who are very sick **[29, 30].** In our study, the mortality rate in the severe group was significantly higher (P=0.002) than in the non-severe group (21.9% vs. 0%). The mortality rate of the first group of critically ill COVID-19 patients in Washington, USA, was 67%, which was greater than our findings [31]. Additionally, 48% of patients with SARS-CoV-2 infection who were hospitalized to the intensive

care unit died [32]. An Italian research of 1591 severely ill patients found that 26% of them died and 58% were still in the intensive care unit. These findings are comparable to ours **[33].** Moreover, in Jordan, reported that the mortality rate in their cohort was 23% **[28]**.

On univariate logistic regression analysis, age, BMI, shortness of breath, SBP, DBP, HR, oxygen saturation, lymphocyte, serum ferritin, CRP, d-dimer, troponin, CK-MB, EF, E/A ratio, P wave, ECG (normal/abnormal), anteroseptal T wave inversion, STEMI/NSTEMI, diastolic function, valvular lesion, DVT and PE were significant predictors for the incidence of MACCE.

On Multivariate logistic regression analysis, BMI, shortness of breath, lymphocyte, serum ferritin, CK-MB, EF, E/A ratio, ECG (normal/abnormal), anteroseptal T wave inversion, diastolic function, DVT and PE were significant predictors for the incidence of complications. This is in line with a research by Zhou et al. [34], which found a correlation between death and the advancing age of COVID-19 patients .

Serious diseases afflict the elderly and those in poorer health, potentially as a result of compromised immune systems [35]. Furthermore, dyspnea was found in 55.4% of COVID-19 patients with ≥ 2 comorbidities as opposed to 34.1%) in patients with just one ailment [36]. Our research supports the findings of those who discovered that, in COVID-19 patients, serum ferritin levels were considerably higher in non-survivors than in survivors during the disease's clinical stages and increased as the patients' condition worsened [34] . Additionally, lymphopenia and elevated C-Reactive Protein were found to be typical laboratory results linked to a poor prognosis [37] .

As low O2 saturation is linked to a higher degree of lung injury and delayed presentation, multiple investigations demonstrated, similarly to our analysis, that low O2 saturation at presentation is associated with higher COVID-19 mortality [38]. Shown that the need for mechanical ventilation and a higher risk of in-hospital death (odds ratio (OR)1.95) are linked to arrhythmias and abnormalities in the electrocardiogram [39] .Shown that QT interval prolonging is linked to a higher number of severe conditions that need to be admitted to the intensive care unit, as well as heart damage and death [40].

The 12-lead ECGs at admission of 1124 consecutive patients hospitalized with Covid-19 and respiratory distress were retrospectively analyzed. Age, the severity of the care environment, heart rate, ST-elevation, QTc prolongation, Q-waves, right bundle branch block, and atrial fibrillation were among the mortality predictors collected and reported, according to the analysis [41]. Aside from dyslipidemia, these factors also independently predicted 30-day mortality: lower EF, older age, SBP, O2 saturation, PAT, and diabetes. It was demonstrated that in COVID-19 patients without a history of structural cardiac disease, RV dilation is a predictor of 30-day and in-hospital mortality [23].

Higher D-dimer concentrations were also linked to severity and mortality in 5872 COVID-19 patients, according to a meta-analysis [42]. In our investigation, the severe group's platelet count was considerably lower than that of the non-severe group. The severe group exhibited considerably greater levels of WBCs, lymphocytes, serum ferritin, CRP, d-dimer, and LDH in comparison to the non-severe group. Serum creatinine and cardiac biomarkers (CK-MB and troponin-I) were considerably higher in the severe group than in the non-sever group. This finding is somewhat consistent with the observation [43] that patients diagnosed with severe COVID-19 also showed signs of lymphopenia and high D-dimer. As indicators of the severity and progression of the disease, rising neutrophil counts, CRP, d-dimer, and LDH levels might be employed, as can falling lymphocyte numbers [35]. In our investigation, the severe group's serum creatinine and cardiac biomarkers (CK-MB and Troponin-I) were considerably greater than those of the non-severe group. Reported that CPK and inflammatory biomarkers were elevated in individuals with cardiac damage, which is consistent with our results [23, 44]. According to published research, the frequency of myocardial damage presenting with high hsTnI ranged from 7.2–19.7% in COVID-19 patients overall and from 23% to 27% in critically ill patients [12, 45].

CONCLUSIONS

Based on these findings, the study concludes that patients with severe COVID-19 disease exhibit significantly worse cardiac pathology across structural, systolic, and diastolic function compared to non-severe disease. Patients with severe disease showed markers of myocardial injury, elevated cardiac biomarkers, ECG changes, impaired left ventricular function, and higher rates of arrhythmias, valvular lesions, pericardial effusion, and diastolic dysfunction. Higher disease severity was associated with prolonged hospitalization, increased thromboembolic events, and greater mortality.

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