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Cardiac involvement according to echocardiographic findings in severe coronavirus disease 2019

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Original Article

Abstract

BACKGROUND: Coronavirus disease 2019 (COVID-19) has led to considerable morbidity and mortality worldwide and myocardial injury has been one of the most common findings in the affected patients. However, published evidence of cardiac evaluation by imaging techniques including echocardiography is rare. We aimed to evaluate myocardial involvement by echocardiography in patients with severe COVID-19.

METHODS: We studied 64 patients with severe COVID-19 who were admitted in the intensive care unit (ICU) in Khorshid Hospital, Isfahan, Iran, from February 20, 2020 until May 20, 2020. Demographic characteristics, laboratory tests, and electrocardiography (ECG) data were collected and transthoracic echocardiography (TTE) using a focused time-efficient echocardiography protocol was performed.

RESULTS: Mean age of the participating patients was 66.40 ± 14.14 years (range: 34.0-92.0 years), and 35 patients (54.7%) were men. Reduced left ventricular (LV) systolic function was seen in 20 (32%) patients. Only 4 patients had LV ejection fraction (LVEF) less than 40%. Cardiac troponin I (cTn-I) was elevated (over 15 pg/ml) in 39 (60.9%) patients and was significantly associated with higher mortality in these patients (P = 0.05). In addition, dynamic ST and T wave changes and new bundle branch blocks had a significant association with adverse clinical outcome (P = 0.05 and P = 0.02, respectively).

CONCLUSION: New LV systolic dysfunction (LVSD) in patients with severe COVID-19 was mild to moderate and not uncommon and had no significant adverse effect on the prognosis of these patients, although elevation of cardiac biomarkers could predict mortality and had an adverse effect on clinical outcome.

Keywords: Echocardiography; Coronavirus Disease 2019; Cardiac Involvement

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Introduction

Coronavirus disease 2019 (COVID-19) caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) began in Wuhan, China, and has led to considerable morbidity and mortality.[1,2] More than five million people were infected worldwide until this date, and 129341 of them were in Iran.[3] Although most people infected with this virus experienced mild or uncomplicated illness, approximately in 14% of them, the severe disease was developed requiring hospitalization and oxygen support and 5% of them required admission to an intensive care unit (ICU).[4,5]

Severe pneumonia in adolescents or adults is

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defined as the presence of fever or suspected respiratory infection, plus one of these criteria: respiratory rate > 30 breaths/minute, severe respiratory distress, or blood oxygen saturation (SpO2) ≤ 93% on room air.[6]

In severe cases, COVID-19 can be complicated by acute respiratory distress syndrome (ARDS), sepsis and septic shock, and multi-organ failure, including acute kidney injury and cardiac injury.[4]

There has been evidence of correlation between the comorbidities and COVID-19 fatality rate. The mortality rate in patients with cardiovascular disease (CVD) and hypertension (HTN) was reported 10.5% and 6%, respectively.[7,8]

Elevation of cardiac biomarkers as a feature of cardiac involvement in COVID-19 is a prominent feature and is related with a worse outcome.[9]

Some potential mechanisms of cardiovascular injury have been identified and consist of direct myocardial injury from hemodynamic derangement or hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction or thrombosis due to hypercoagulability, or systemic inflammation (cytokine storm), which may also destabilize coronary artery plaques.[10] Besides, coronavirus enters into the endothelial cells and cardiac myocytes through angiotensin-converting enzyme 2 (ACE2), and due to its entrance, two categories of cardiac symptoms happen:

1. Microvascular and macrovascular dysfunctions that lead to acute coronary syndrome (ACS)
2. Cytokine storm and inflammation manifested as myocarditis and heart failure (HF)[11]

Acute myocardial injury was reported as a complication of Middle East respiratory syndrome coronavirus (MERS-CoV).[10] Furthermore, among the causes of death in a Wuhan cohort study, myocardial injury and HF contributed to 40% of deaths, either entirely or in combination with respiratory failure.[11]

In a retrospective study in China, 187 patients with COVID 19 were evaluated, and 27.8% of them had myocardial injury indicated through high level of troponin I (Tn-I).[12]

As far as we know, there are rare data about echocardiographic characteristics of patients with severe COVID-19 with or without myocardial injury, and their correlation with mortality is unknown. This study aims to evaluate the association between survival and echocardiographic and electrocardiographic (ECG) characteristics of patients with severe COVID-19 who were admitted to the ICU with or without myocardial injury in Khorshid Hospital, Isfahan, Iran.

Materials and Methods

Study design and participants: This was a single center, prospective cohort study that was performed at Khorshid Hospital which was the main COVID-19 center in Isfahan at time of collecting data. Our study was approved by Ethics Committee of Isfahan University of Medical Sciences (IR.MUI.MED.REC.1399.028).

COVID-19 was diagnosed through positive reverse transcription polymerase chain reaction (RT-PCR) nasopharynx swab test. All patients with severe consecutive COVID-19 who were admitted in the ICU from February 20, 2020 until May 20, 2020 were entered in this study. Patients with unknown history of left ventricular (LV) dysfunction, patients with history of any structural heart disease for which there was no documented ECG or echocardiography before COVID-19 disease, and patients with poor imaging quality in transthoracic echocardiography (TTE) were excluded from our study.

Data collection: In the first day of ICU admission, demographic characteristics were collected and these laboratory tests were checked: complete blood count (CBC)/diff, c-reactive protein (CRP), blood urea nitrogen (BUN), Cr, Na, K, Ca, Ph, albumin (Alb), aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), and high-sensitivity Tn (hs-Tn).

Level of hs-Tn was checked at least two times and the level greater than 15 pg/ml was reported abnormal in our laboratory. Patients were divided into three categories:

1. Normal: hs-Tn less than 15 pg/ml
2. Mildly elevated: hs-Tn between 15 and 150 pg/ml
3. Significantly elevated: hs-Tn greater than 150 pg/ml

ECG was taken daily during ICU admission. ECG in first day of admission was considered as an initial ECG for each patient and any new changes in daily ECG in comparison to initial ECG was recorded as new ECG changes. ECG findings were reported as presence of sinus rhythm, sinus tachycardia, tachyarrhythmia, bradycardia, ST-T changes, bundle branch block (right and left), other changes [such as atrioventricular (AV) blocks, hemifascicular blocks, poor R wave progression, and etc.], and dynamic ECG changes during ICU admission.

TTE was done in the first day of admission according to patient condition and by focused time-efficient echocardiography protocol.[13] Echocardiography data were acquired by using a

portable echocardiography machine (Siemens, Acuson P500, Germany, with a P4-2 cardiac probe). TTE was done by an experienced cardiologist, echocardiography fellowship; image acquisition was performed with settings optimal for LV and right ventricular (RV) assessment and interpreted off-line and blind.

Echocardiographic findings including cardiac chamber sizes, LV ejection fraction (LVEF) representing LV systolic function, tricuspid annular plane systolic excursion (representing RV systolic function), presence of pericardial effusion, and pulmonary artery pressure (PAP) were recorded.

Echocardiographic abnormalities were defined as reduced LVEF, presence of global or regional wall motion abnormality (RWMA), LV hypertrophy (LVH), abnormal RV systolic function, abnormal PAP, and pericardial effusion (PE) (≥ 5 mm).

LV systolic function measured with Simpson's method and LVEF were categorized into four groups: normal (> 53%), mild dysfunction (40%-53%), moderate dysfunction (30%-39%), severe dysfunction (less than 30%), and previously abnormal ejection fraction (EF).[14]

According to 2015 European Society of Cardiology/European Respiratory Society (ESC/ERS) guidelines, signs of pulmonary HTN (PH) are defined as peak tricuspid regurgitation velocity (TRV) > 2.8 m/s with echocardiographic signs including the changes of ventricles as RV/LV basal diameter ratio > 1.0, or pulmonary changes as pulmonary acceleration time < 105 msec and/or mid systolic notching (MSN), or signs of the increase of right atrial pressure (RAP) as inferior vena cava (IVC) diameter > 21 mm with decreased inspiratory collapse (< 50% with a sniff or < 20% with quiet inspiration). At least two of the above echocardiographic signs are required to determine higher probability of PH.[15] In addition, possible myocarditis was defined according to myocarditis criteria.[16]

Outcome: Mortality was defined as death "in hospital" or "in one-month follow-up". Poor clinical outcome included "ICU admission more than 14 days", "mechanical ventilation", and "in-hospital death".

Statistical analysis: The statistical analysis was completed with SPSS software (version 22.0, IBM Corporation, Armonk, NY, USA). Categorical variables were described as number and percentages. Continuous variables were expressed as mean and standard deviation (SD). Kolmogorov-Smirnov test was used for testing normality assumption. Continuous variables were compared with independent samples t-test, while categorical variables were analyzed by chi-square test or Fisher's exact test (if needed). P-value less than 0.05 was considered as statistical significance.

Results

Demographic and clinical characteristic: The demographic and baseline clinical characteristics of patients with COVID-19 are summarized in table 1. There were 85 ICU-admitted patients with confirmed diagnosis of severe COVID-19 in our study population. However, according to study design, we excluded 20 patients (9 patients with unknown past history of structural heart diseases and 11 patients with poor imaging quality).

The mean age was 66.40 ± 14.14 years (range: 34.0-92.0 years), and 35 patients (54.7%) were men.

Of 64 patients, 34 (53.1%) had HTN, 24 (37.5%) had diabetes, and 11 (17.2%) had coronary heart disease (CHD), which were the most common coexisting conditions. Among them, 35 patients (54.7%) needed intubation and invasive mechanical ventilation and other 29 patients (45.3%) needed nasal O2 therapy or non-invasive ventilation (NIV) during ICU duration. After all, 22 (34.4%) patients died, including 19 (29.7%) in hospital and 3 (4.7%) after discharge. Patients were discharged after 19.2 ± 9.1 (mean hospital stay) days.

TTE findings: Echocardiographic data are listed in table 2. Reduced LV function was seen in 20 (31%) patients and only 4 patients had LVEF less than 40% which was not associated with poor clinical outcome and mortality [possible myocarditis: 16 (23.4%), myocardial infarction (MI): 2 (3.1%), and Takotsubo syndrome (TTS): 2 (3.1%)].

Two patients had acute MI, one of them had anterior wall MI and another inferior MI. Patients with MI showed severe reduced LVEF and segmental wall motion abnormalities. Tn-I in these patients was elevated more than ten times.

Although 52 (81.3%) patients had normal RV systolic function and 12 (18.7%) patients had decreased RV systolic function, there was no significant relation between RV systolic dysfunction (RVSD) and mortality or poor clinical outcome. However, among 19 (29.7%) patients with PH, mortality was not significantly increased.

The pericardial effusion was mild in majority of patients except in one patient that myopericarditis had happened during the disease course and resolved after prednisolone therapy.

Table 1. Clinical characteristic of patients with coronavirus disease 2019 (COVID-19) according to mortality and clinical outcomes

Variables	All patients (n = 64)	Clinical outcome		P	Mortality		P
		Good* (n = 32)	Poor* (n = 32)		No (n = 42)	Yes (n = 22)	
Age (year)	66.00 ± 14.10	65.80 ± 14.70	66.20 ± 13.70	0.90	64.70 ± 14.10	68.50 ± 14.10	0.31
Vital signs in admission							
Temperature on admission (°C)	37.54 ± 0.94	37.40 ± 0.82	37.60 ± 1.04	0.52	37.50 ± 0.89	37.60 ± 1.04	0.65
Blood oxygen saturation (%)	76.40 ± 12.20	77.30 ± 10.50	75.50 ± 13.70	0.54	78.40 ± 10.20	72.60 ± 14.70	0.06
Heart rates (bpm)	97.80 ± 16.80	97.90 ± 13.60	97.80 ± 19.60	0.98	98.00 ± 14.90	97.50 ± 20.30	0.90
Respiratory rates (bpm)	27.50 ± 6.40	28.20 ± 7.10	26.80 ± 5.70	0.38	27.90 ± 7.00	26.80 ± 5.30	0.54
SBP (mmHg)	135.67 ± 21.00	133.70 ± 19.10	137.60 ± 23.50	0.46	134.10 ± 19.10	138.50 ± 23.90	0.44
DBP (mmHg)	81.20 ± 15.70	79.40 ± 13.70	83.10 ± 17.50	0.35	80.50 ± 14.30	82.50 ± 18.30	0.61
Sex (male)	35 (54.00)	19 (59.40)	16 (50.00)	0.45	25 (59.50)	10 (45.50)	0.28
Coexisting condition							
HTN	34 (53.10)	18 (56.30)	16 (50.00)	0.61	23 (54.80)	11 (50.00)	0.71
Diabetes	24 (37.50)	12 (37.50)	12 (37.50)	0.69	15 (35.70)	9 (40.90)	0.68
CHD	11 (17.10)	6 (18.80)	5 (15.60)	0.74	7 (16.70)	4 (18.20)	0.87
History of arrhythmia	2 (0.03)	1 (3.10)	1 (3.10)	0.75	2 (4.80)	0 (0)	0.30

Continuous and categorical variables are reported as mean ± standard deviation (SD) and number (percent), respectively; chi-square and independent t t-test were used for categorical and continuous variables

* Poor clinical outcome included "intensive care unit (ICU) admission more than 14 days", "mechanical ventilation", and "in-hospital death" and the rest were good clinical outcome

BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; HTN: Hypertension; CHD: Coronary heart disease

Table 2. Echocardiographic, electrocardiographic (ECG), and laboratory findings of patients with coronavirus disease 2019 (COVID-19) according to mortality and clinical outcomes

Variables	All patients (n = 64)	Clinical outcome		P	Mortality		P
		Good** (n = 32)	Poor** (n = 32)		No (n = 42)	Yes (n = 22)	
Echocardiography data							
LVSD	20 (31.2)	8 (25.0)	12 (37.5)	0.27	12 (28.6)	8 (36.4)	0.52
RVSD	12 (18.7)	5 (15.6)	7 (21.9)	0.52	8 (19.0)	4 (18.2)	0.93
PAH	19 (29.0)	7 (21.9)	12 (37.5)	0.16	11 (26.2)	8 (36.4)	0.40
Pericardial effusion	5 (7.8)	1 (3.1)	4 (12.5)	0.14	3 (7.1)	2 (9.1)	0.78
Regional wall motion abnormality	12 (18.7)	4 (12.5)	8 (25.0)	0.19	6 (14.3)	6 (27.3)	0.21
LVH	10 (15.6)	7 (70.0)	3 (30.0)	0.16	7 (16.7)	3 (13.6)	0.75
ECG findings							
New sinus tachycardia	39 (60.9)	18 (56.3)	21 (65.6)	0.44	25 (59.5)	14 (63.6)	0.74
New tachyarrhythmia	8 (12.5)	4 (12.5)	4 (12.5)	0.44	6 (14.3)	4 (50.0)	0.44
New ST-T change	15 (23.4)	9 (28.1)	6 (18.8)	0.37	10 (23.8)	5 (22.7)	0.92
New transient ECG change	12 (18.7)	3 (9.4)	9 (28.1)	0.05*	7 (16.7)	5 (22.7)	0.56
New bundle branch block	8 (12.5)	1 (3.1)	7 (21.9)	0.02*	2 (4.8)	6 (27.3)	0.01*
Laboratory findings							
Hb (g/dl) (130-175 g/l)	12.40 ± 2.00	12.38 ± 1.80	12.40 ± 2.20	0.89	12.26 ± 1.70	12.70 ± 2.40	0.40
Leukocytes (/µl)	8982.80 ± 6414.20	8506.20 ± 5491.20	9456.00 ± 7279.40	0.55	8671.40 ± 4975.00	9577.20 ± 8633.70	0.59
Platelets ($\times 10^{-3}$/µl)	212437.50 ± 81434.90	237312.50 ± 7072.60	187562.50 ± 84716.20	0.01*	225452.40 ± 75912.70	187590.90 ± 87511.90	0.07
CRP (mg/l) (0-10 mg/l)	39.80 ± 26.60	41.10 ± 25.40	38.50 ± 28.10	0.70	40.80 ± 27.80	37.80 ± 24.50	0.66
LDH (U/l) (120-250 U/l)	884.10 ± 400.90	810.30 ± 331.40	980.70 ± 473.50	0.25	845.20 ± 379.20	961.90 ± 451.90	0.46
CTn-I (pg/ml) (0-15 pg/ml)	456.10 ± 1252.50	281.60 ± 802.30	646.30 ± 1607.0	0.32	288.90 ± 753.90	669.40 ± 1856.40	0.29
CTn-I more than 15 pg/ml	44 (68.7)	21 (65.5)	23 (71.9)	0.58	25 (59.5)	19 (86.4)	0.05*
CTn-I more than ten times (150 pg/ml)	12 (18.8)	4 (33.1)	8 (66.7)	0.19	6 (14.3)	6 (27.3)	0.21
Sodium (mEq/l)	134.90 ± 4.50	135.80 ± 4.70	134.10 ± 4.20	0.12	136.00 ± 4.50	132.90 ± 3.80	0.09
Potassium (mEq/l)	3.90 ± 0.50	4.00 ± 0.60	3.90 ± 0.50	0.26	4.00 ± 0.50	3.80 ± 0.40	0.21
ALT (U/l)	59.95 ± 67.60	49.20 ± 26.20	77.00 ± 89.50	0.04*	48.90 ± 48.10	81.00 ± 92.20	0.07
AST (U/l)	41.80 ± 50.80	36.60 ± 39.20	46.90 ± 60.40	0.41	40.60 ± 49.30	44.10 ± 54.80	0.79
ALP (U/l)	169.03 ± 83.50	154.00 ± 40.90	183.00 ± 109.90	0.18	160.10 ± 48.90	186.10 ± 125.50	0.23
PH	7.35 ± 0.09	7.37 ± 0.10	7.34 ± 0.07	0.23	7.36 ± 0.09	7.33 ± 0.07	0.03*
Calcium (mg/dl)	8.29 ± 0.74	8.47 ± 0.66	8.11 ± 0.78	0.05	8.38 ± 0.74	8.12 ± 0.73	0.20
Magnesium (mEq/l)	1.92 ± 0.23	1.87 ± 0.18	1.97 ± 0.27	0.12	1.94 ± 0.23	1.89 ± 0.24	0.45
Albumin (g/dl)	3.81 ± 0.48	3.90 ± 0.38	3.60 ± 0.54	0.02*	3.85 ± 0.34	3.73 ± 0.45	0.33
Arterial PCO_2 (mmHg)	45.71 ± 14.00	43.40 ± 11.40	48.00 ± 16.10	0.50	46.85 ± 16.02	43.53 ± 9.00	0.37
BUN (mg/dl)	31.50 ± 23.30	30.70 ± 25.40	32.30 ± 21.40	0.79	32.20 ± 25.70	30.20 ± 18.40	0.74
Creatinine (mg/dl)	1.70 ± 1.90	1.80 ± 2.10	1.66 ± 1.70	0.77	1.80 ± 2.20	1.59 ± 1.30	0.68

Continuous and categorical variables are reported as mean ± standard deviation (SD) and number (percent), respectively; chi-square test and independent t-test were used for categorical and continuous variables.

* Showing significant difference; ** Poor clinical outcome included "intensive care unit (ICU) admission more than 14 days", "mechanical ventilation", and "in-hospital death" and the rest were good clinical outcome

LVSD: Left ventricular systolic dysfunction; RVSD: Right ventricular systolic dysfunction; PAH: Pulmonary arterial hypertension; LVH: Left ventricular hypertrophy; ECG: Electrocardiography; CRP: C-reactive protein; Hb: Hemoglobin; LDH: Lactate dehydrogenase; CTn-I: Cardiac troponin I; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ALP: Alkaline phosphatase; PCO2: Partial pressure of carbon dioxide; PH: Power of hydrogen; BUN: Blood urea nitrogen

Electrocardiographic findings: Serial ECG was done during ICU admission. Sinus tachycardia was the most finding seen in 39 (60.9%) patients; however, new dynamic change and bundle branch blocks had significant association with poor clinical outcome (P = 0.05, P = 0.02, respectively).

Laboratory results: Laboratory findings are shown in table 2. Cardiac troponin I (cTn-I) was elevated (over 15 pg/ml) in 39 (60.9%) patients. This was significantly associated with higher mortality in these patients (P = 0.05).

Discussion

In our study, we evaluated cardiac systolic function by TTE in patients with severe COVID-19 in Khorshid Hospital that was one of the main COVID-19 centers in Isfahan.

This study showed that new mild to moderate decreased LV systolic function in patients with severe COVID-19 had no significant effect on mortality, while Tn-I elevation was significantly associated with increased in-hospital and one-month follow-up mortality in patients with severe COVID- 19. Indeed, LV systolic dysfunction (LVSD) was mild to moderate and this degree of dysfunction was not associated with increased mortality; however, Tn-I augmentation was not parallel with LVSD in this study and this increase was associated with increased mortality.

In addition, new ECG changes, thrombocytopenia, and increased ALT were also significantly associated with higher mortality and poor clinical outcome in severe patients who needed ICU admission.

Similar to our study, Shi et al.[6] and Shi et al.[17] showed that cardiac injury was a common condition among hospitalized patients with COVID-19 in China, and it was associated with a higher risk of in-hospital mortality. However, the frequency of cardiac injury in our patients was more than Shi et al.[6] study (68.0% versus 19.7%) which can be due to the selection of severe COVID-19 in our study. However, Deng et al. study showed that elevation of Tn-I was more likely related to systemic disorders not cardiac injury (as myocarditis) and could be the warning sign for death in patients with COVID-19.[18] It seems that mild to moderate LVSD is probably associated with COVID-19 and does not directly affect mortality or clinical outcome, although it is associated with increased Tn-I level.

The precise pathophysiology of direct (infiltration of virus into myocardial cells and myocardial inflammation) or indirect myocardial injury (respiratory failure and hypoxemia, multi-organ dysfunction, hyper-inflammation accompanied by cytokine storm leading to reduction in coronary blood flow, destabilization of coronary plaque, and microthrombogenesis) in previously healthy patients with severe COVID-19 is not completely understood.[4-19] We showed possible myocarditis as direct mechanism in 23% of patients and indirect mechanism (MI and stress cardiomyopathy) in 6% of patients.

Limitations: Our study had some limitations. We could not perform cardiac magnetic resonance (CMR) and myocardial biopsy as gold standard methods for myocarditis diagnosis. In addition, echocardiography, ECG parameters, and all the laboratory tests were measured in the first week of ICU admission and some of the patients' complications occurred in the second week. Besides, some of patients had long-term course of disease and some new complications occurred in these settings and changed the time and course of disease, and finally our sample size was small for regression analysis.

Conclusion

New LVSD in patients with severe COVID-19 using a focused time-efficient echocardiography protocol was not uncommon; however, decreased LVEF was not less than 40% and did not have a significant adverse effect on the prognosis of these patients, although elevation of cardiac biomarkers could predict mortality and had an adverse effect on clinical outcome.

Acknowledgments

None.

Conflict of Interests

Authors have no conflict of interests.

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