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ORIGINAL PAPER

Echocardiographic and electrocardiographic findings in COVID-19 patients: a cross-sectional study

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Abstract

There are still many gaps in our knowledge regarding the direct cardiovascular injuries due to COVID-19 infection. In this study, we tried to find out the effect of SARS-CoV-2 infection on cardiac function in patients without any history of structural heart disease by electrocardiographic and echocardiographic evaluations. This was a cross-sectional study on patients with COVID-19 infection admitted to Imam Reza hospital, Mashhad, Iran between 14 April and 21 September 2020. COVID-19 infection was verified by a positive reverse-transcriptase polymerase chain reaction (PCR) assay for SARS-CoV-2 using nasopharyngeal/oropharyngeal samples. We enrolled all patients over 18 years old with definite diagnosis of COVID-19 infection. All patients underwent a comprehensive transthoracic echocardiography at the first week of admission. Clinical and imaging data were collected prospectively. In total, 142 patients were enrolled in this study. The mean age of participants was 60.69 ± 15.70 years (range: 30–90 years). Most patients were male (82, 57.7%). Multivariate analysis showed that O2 saturation at admission was independently a predictor of re-hospitalization ($P < 0.001$). RV size ($P < 0.001$), dyslipidemia ($P < 0.001$), ejection fraction (EF) ($P < 0.001$), age ($P = 0.020$), systolic blood pressure ($P = 0.001$), O2 saturation ($P = 0.018$) and diabetes ($P = 0.025$) independently predicted 30-days mortality. Echocardiography can be used for risk assessment in patients with COVID-19, especially in those with previous history of diabetes and dyslipidemia. The infection could result in ventricular dysfunction, even in those without previous history of structural heart disease.

Keywords COVID-19 · Echocardiography · Electrocardiography · Right ventricular dysfunction

Abbreviations

COVID-19	Coronavirus disease 2019
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
PCR	Polymerase chain reaction
CT	Computerized tomography
ECG	Electrocardiography
RV	Right ventricle
PASP	Pulmonary artery systolic pressure
EF	Ejection fraction
TPI	Troponin I
TTE	Transthoracic echocardiography
GGO	Ground glass opacification
PHT	Pulmonary hypertension
ESR	Erythrocyte Sedimentation Rate
CPK	Creatine phosphokinase
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
OR	Odds ratio
PAT	Pulmonary acceleration time
FWLS	Free wall longitudinal strain
CRP	C reactive protein
hsTnI	High sensitive troponin I
PE	Pulmonary embolism
ARDS	Acute respiratory distress syndrome
BNP	Brain natriuretic peptide

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Background

Knowledge about short- and long-term complications of COVID-19 infection is growing worldwide [1]. COVID-19 is now a serious pandemic infection with different clinical features [2]. It is shown that severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) can involve respiratory tract and patients with previous cardiovascular disorders are at a higher risk [3]. The underlying mechanisms are yet to be clear [4]. It has been suggested that cardiac injury in COVID-19 infection is possibly related to its clinical outcomes. It was reported that patients with cardiac injury are associated with higher rate of morbidity, more abnormal laboratory and radiological findings [5]. Pathological findings (mononuclear inflammatory infiltration in heart tissue) in patients with COVID-19 infection suggest a direct damage to cardiac tissues [6]. Moreover, it is indicated that previous cardiovascular disorders may also be more predisposing to cardiac injury due to COVID-19 infection [5]. Keeping in mind the cardiovascular presentations, underlying cardiovascular disorders and its adverse events can help the clinician to make better decisions in the management of COVID-19 infection [2].

Cardiac adverse events related to COVID-19 infection are prevalent (nearly 25%) and can increase the risk of mortality [7]. Most recent studies on COVID-19 cardiac complications have reported clinical or laboratory findings with no imaging confirmation.

Transthoracic echocardiography is a reliable technique to detect ventricular dysfunction or valve disorders and risk assessment, as an additive guide to select better treatment strategies for hemodynamic stabilization [8]. However, due to a risk of viral contamination, echocardiographic evaluation in patients with COVID-19 infection has been limited [9, 10]. To improve our knowledge about the pathophysiology of direct cardiovascular injuries due to COVID-19 infection [2], we tried to find the effect of SARS-CoV-2 infection on cardiac function in patients without any history of structural heart disease by electrocardiographic and echocardiographic evaluations.

Methods

This was a cross-sectional study, performed on admitted patients with COVID-19 infection, in Imam Reza hospital, Mashhad, Iran between 14 April and 21 September 2020.

Eligibility

COVID-19 infection was verified by a positive reverse-transcriptase polymerase chain reaction (PCR) assay for SARS-CoV-2 using nasopharyngeal/oropharyngeal samples. We included all patients over 18 years old and a definite diagnosis of COVID-19 infection. We excluded patients with previous structural heart disease by history taking or evidences of old myocardial infarction in ECG. Figure 1 shows the flowchart of study.

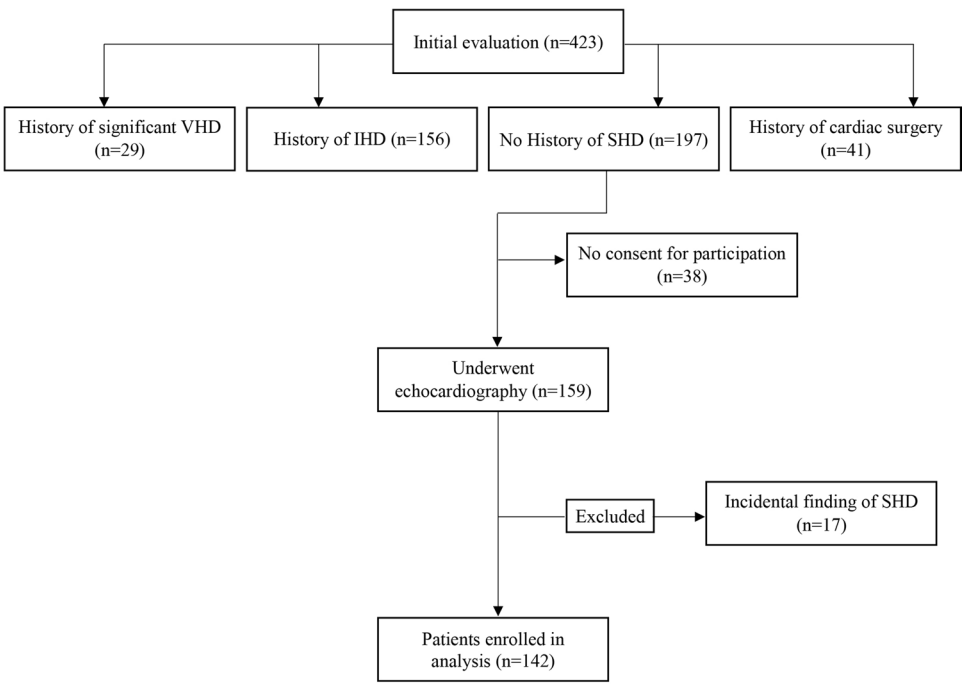

Fig. 1 Flowchart of study. *VHD* valvular heart disease, *IHD* ischemic heart disease, *SHD* structural heart disease

Evaluations

At admission, demographic data, comorbid conditions, medications, physical examination, lung CT scan and laboratory findings were all recorded. Cardiac injury was determined as troponin I (TPI) above the 99th percentile at presentation. Clinical and imaging data were collected prospectively and a 12-lead electrocardiography was performed. All patients were followed about three months for re-admission, cardiac events and in-hospital mortality or 30-day mortality.

Echocardiography

All patients underwent a comprehensive transthoracic echocardiography (TTE) within two weeks after symptoms presentation, in a standard manner with the same equipment (M7, Mindray Bio-Medical Electronics, Shenzhen, China) and by one cardiologist. All echocardiographic studies were performed bedside at COVID-19 intensive care unit (ICU) or non-ICU. Personal protection included airborne precautions, N-95 respirator masks, fluid-resistant gowns, and 2 sets of gloves, head covers, eye shields, and shoe covers. Most of measurements were performed offline to reduce the exposure time and contamination with two-to-three-seconds clips derived from standard echo views. ventricular size, systolic and diastolic function, pulmonary artery systolic pressure (PASP), presence of pericardial effusion, valvular stenosis or regurgitation were assessed. Abnormal TTE was defined as LV and RV enlargement, RV dysfunction (by visual assessment), ejection fraction (EF) $\leq 54\%$, pericardial effusion, PASP ≥ 35 mmHg, moderate and severe diastolic dysfunction and valvular regurgitation or stenosis more than mild.

Exclusion criteria

In order to better assessment of direct effects of COVID-19 on the echocardiographic parameters, patients with documented history of previous structural heart diseases (like valvular stenosis, primary valvular regurgitation, previous MI and heart surgery, etc.) were excluded.

Ethics

The ethics committee of Mashhad University of Medical Sciences approved the study (Ethical No IR.MUMS.REC.1399.016). We obtained an informed consent from all patients or their legal guardians. The study was conducted in accordance with the declaration of Helsinki. Informed written consent was given prior to the inclusion of subjects or their families, in the study.

Results

In Total, 142 patients with diagnosis of Covid-19 infection, were enrolled who were admitted in Imam Reza Hospital, Mashhad, Iran. The mean age of participants was 60.69 ± 15.70 years (range: 30–90). Most patients were male (82, 57.7%). Known comorbidities were found in 45.1%, with hypertension (23.9%) and diabetes mellitus (23.9%) as the most common ones. The more frequent symptoms were dyspnea on admission (90.8%), cough (54.9%) and fever (49.3%). Baseline demographic characteristics, comorbidities, clinical and laboratory data were presented in Table 1.

Left axis deviation (LAD) was the most common finding (15.5%) in ECG. Seventy-eight patients had sinus tachycardia (55%), one atrial fibrillation (0.7%) and two AVNRT (1.4%). Ten patients had right axis deviation (RAD) and others normal axis.

Bilateral peripheral ground glass opacification (GGO) was the most common specific CT scan manifestation (38%) followed by diffused bilateral GGO (35.2%), unilateral GGO (14.1%), and bilateral pleural effusion (9.9%). Other non-specific presentations were consolidation (63.4%), and lymphadenopathy (31%).

Abnormal TTE was seen in 71 cases (50%). Preserved left ventricle EF (LVEF: 50–54%) was noted in 21 cases and reduced EF (EF < 50%) in 8 (5.6%). Seventy-nine patients had mild diastolic dysfunction (56.3%), two of them moderate (1.4%) and others had normal diastolic function. Mild RV dilation was seen in 14 cases (9.9%) and moderate in two cases (1.4%). Mild pulmonary hypertension (PHT) was found in 11 subjects. Representative example of mild RV enlargement and mild PHT in a COVID-19 patient without prior cardiovascular disorder is shown in Fig. 2. Pericardial effusion was detected in 32 cases (22 minimal PE and 10 mild PE). The echocardiographic indices are shown in Table 2.

In-hospital mortality was seen in 22 patients (15.5%), 30 days-mortality in 24 patients (16.9%) and re-admission in 3 patients (2.1%). RV dysfunction was reported in 49 patients (34.5%) (39 mild RV dysfunction, 8 moderate, 2 severe). Table 3 demonstrates the patients' characteristics stratified by RV function. RV size was correlated with O2 saturation ($r = -0.302$, $P < 0.001$), TPI ($r = 0.337$, $P = 0.019$), ESR ($r = 0.207$, $P = 0.035$) and Platelet count ($r = 0.355$, $P < 0.001$). CPK ($P = 0.033$) and calcium ($P = 0.005$) levels were significantly higher in patients with RV dysfunction.

Table 1 Baseline demographic characteristics, comorbidities, clinical and laboratory data

Parameter	
Age (years) (mean ± SD)	60.69 ± 15.70
Sex (N, %)	
Male	82, 57.7
Female	60, 42.3
Comorbidities (N, %)	
Diabetes	34, 23.9
Hypertension	34, 23.9
Dyslipidemia	16, 11.3
History of Stroke	4, 2.8
Chronic kidney disease	6, 4.2
Lung disease	9, 6.3
History of malignancy	3, 2.1
Current smoker	8, 5.6
Addiction	17, 11.9
Vital sign (Mean ± S.D)	
Heart rate (beats/minute)	102.43 ± 19.23
Systolic blood pressure (mmHg)	135.46 ± 20.66
Diastolic blood pressure (mmHg)	84.62 ± 20.66
O_2 saturation (%)	88.31 ± 7.83
Temperature (°C)	37.38 ± 0.79
Respiratory rate (breaths/minute)	23.92 ± 5.42
Symptoms (N, %)	
Cough	78, 54.9
Dyspnea	129, 90.8
Fever	70, 49.3
Weakness	57, 40.1
Chest pain	2, 1.4
Gastrointestinal	25, 17.6
Confusion	8, 5.6
Laboratory data (Mean ± S.D)	
Blood sugar (mg/dL)	142.12 ± 22.88
Urea (mg/dL)	43.94 ± 17.03
Creatinine (mg/dL)	1.34 ± 0.7
Sodium (meq/L)	136.28 ± 3.49
Potassium (meq/L)	4.09 ± 0.60
Total bilirubin (mg/dL)	0.61 ± 0.43
Direct bilirubin (mg/dL)	0.25 ± 0.15
Calcium (mg/dL)	8.54 ± 0.89
Magnesium (mg/dL)	2.06 ± 0.42
Aspartate transaminase (IU/L)	44.90 ± 18.23
Alanine aminotransferase (IU/L)	49.06 ± 17.66
Alkaline phosphatase (IU/L)	212.32 ± 80.91
Lactate dehydrogenase (IU/L)	606.65 ± 230.51
Creatine phosphokinase (IU/L)	350.97 ± 143.38
Cardiac troponin I (ng/ml)	56.22 ± 25.24
PCO2 (mmHg)	42.10 ± 13.79
HCO3 (meq/L)	27.28 ± 6.57
White blood cell (10^6/L)	8860.56 ± 5732.48
PMN (%)	77.88 ± 9.31
Lymph (%)	16.65 ± 7.93
Hemoglobin (g/dL)	13.18 ± 2.11
MCV (fL)	84.54 ± 7.01
RDW (%)	14.08 ± 1.74
Platelet (10^9/L)	222.18 ± 126.55
CRP (mg/L)	88.63 ± 74.66
ESR (mm/h)	42.9 ± 30.26

PCO2 partial pressure of carbon dioxide, *HCO3* bicarbonate, *PMN* polymorphonuclear leukocytes, *Lymph* lymphocyte, *MCV* mean corpuscular volume, *RDW* red cell distribution width, *CRP* C-reactive protein, *ESR* erythrocyte sedimentation rate

Our findings demonstrated that SBP (OR: 0.885, P = 0.006), DBP (OR: 1.24, P = 0.003), male gender (OR: 0.40, P = 0.004), higher age (OR: 1.37, P = 0.002), smoking (OR: 0.011, P = 0.035), addiction (OR: 0.031, P = 0.011), hypertension (OR: 2.41, P = 0.003), asthma (OR: 0.10, P = 0.032), bilateral peripheral GGO (OR: 23.6, P = 0.019) and coronary calcification (OR: 5.74, P = 0.005) were predictors of in-hospital mortality. Moreover, age (OR: 1.29, P = 0.002), hypertension (OR: 2.99, P = 0.004) and bilateral peripheral GGO (OR: 26.81, P = 0.023) were predictors of 30-day mortality.

In multivariate regression analysis, lower O2 saturation at the time of admission was independently predictor of re-admission (P < 0.001). Furthermore, PASP (P = 0.026), dyslipidemia (P = 0.002) and RV dilation (P = 0.037) were significantly predictors of in-hospital mortality (after adjusting for possible cofounders). RV enlargement (P < 0.001), dyslipidemia (P < 0.001), lower LVEF (P < 0.001), older age (P = 0.020), SBP (P = 0.001), O2 saturation (P = 0.018), shorter pulmonary acceleration time (PAT) (P = 0.005) and diabetes (P = 0.025) independently predicted 30-days mortality. Patients with cardiac injury were mostly females (P = 0.001), had hypertension (P = 0.001), more comorbidities (P = 0.026), bilateral peripheral GGO (P = 0.001), RV dysfunction (P = 0.029), a higher level of CPK (P = 0.018) and coronary calcification (P = 0.006), compared to those without. Likewise, in patients with cardiac injury, pericardial effusion was significantly more reported (16 versus one, P = 0.021). LV dysfunction (EF lesser than 55%) was seen in 20.4% of cases; probably most of them due to COVID-19 related cardiac injury.

Discussion

Investigations regarding cardiovascular complications in COVID-19 in developing countries are limited. To the best of our knowledge, our study is the first to assess cardiac

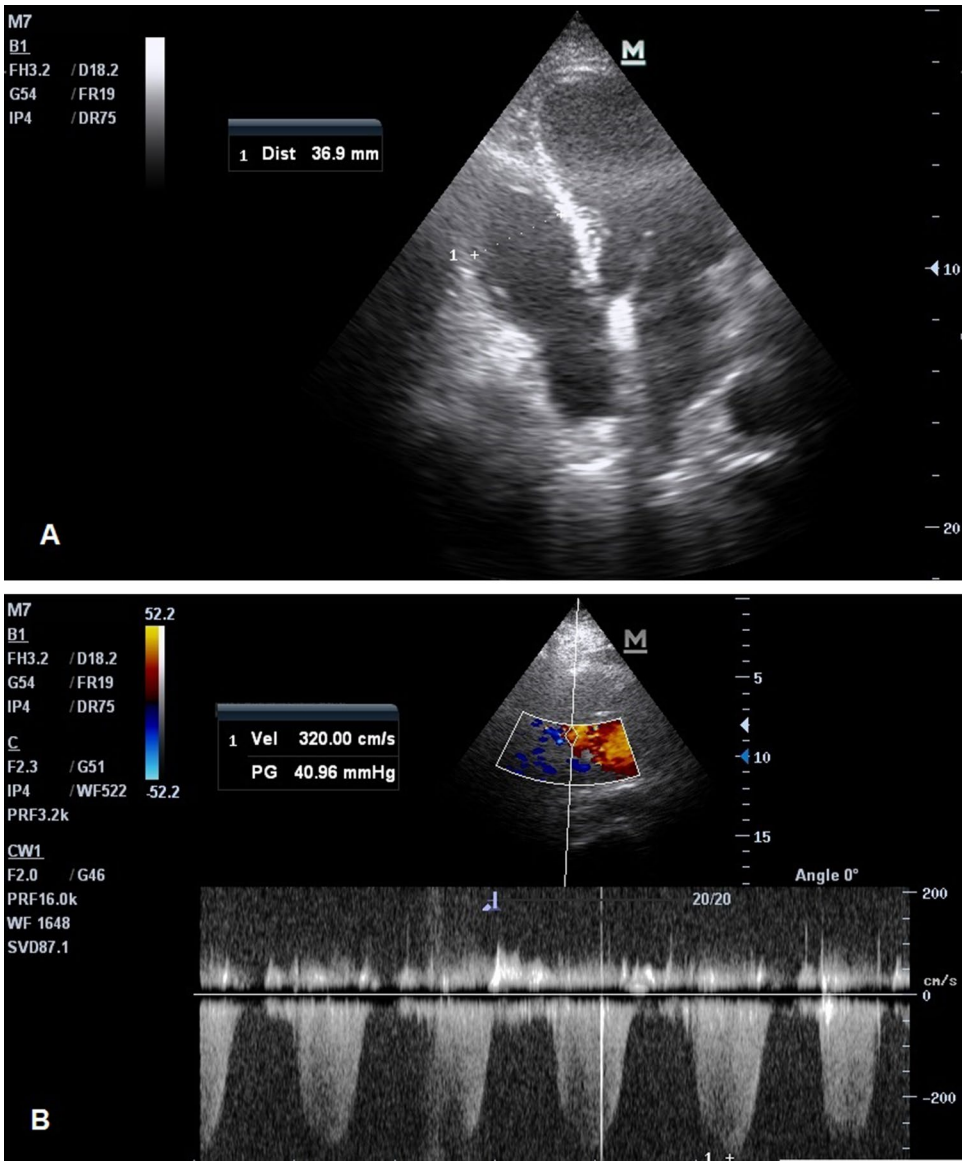

Fig. 2 Example of two-dimensional transthoracic echocardiography of a patient without previous cardiovascular disorder. **A** Mild right ventricular enlargement in RV focused view; **B** off axis four-chamber view of the heart suggesting moderate TR with TR gradient about 41 mmHg. Estimated pulmonary arterial pressure in this patient was 44 mmHg (considering normal IVC size and collapsibility)

function in COVID-19 patients using both echocardiography and ECG findings in those without any significant structural heart diseases. Our aim was to evaluate the direct impact of SARS-CoV-2 infection on cardiovascular indices in hospitalized patients.

As above-mentioned, patients with cardiac injury had a higher level of CPK, comorbidities and RV dysfunction. In a similar study, patients with cardiac injury had higher levels of inflammatory biomarkers, underwent mechanical ventilation more probably and had higher mortality rate and RV dysfunction. They showed that two indices of strain echocardiography, i.e. LV global longitudinal strain and RV free wall longitudinal strain (FWLS) were powerful predictors of higher mortality in patients with COVID-19 infection [11].

RV dysfunction in COVID-19 patients is common and associated with a pro-thrombotic and inflammatory state which is determined by D-dimer and CRP elevation. In our study, RV systolic dysfunction was reported in 49 cases (34.5%) and LV systolic dysfunction in 20.4%. We showed a significant correlation between RV size and TPI level. In another retrospective study, LV systolic function was hyperdynamic or normal in most cases (89%), RV was dilated in 41%, and impaired in 27%. It was demonstrated that RV systolic dysfunction (defined by fractional area change) was significantly associated with elevated D-dimer and CRP but not related to hsTnI [9].

Similar to our study, in another investigation, all indices of RV hemodynamics and function were worse compared to the LV parameters. They introduced RV dilation and dysfunction as major abnormal echocardiographic patterns among deteriorating patients [10]. Many conditions can increase pulmonary vascular resistance or pulmonary pressure in

Table 2 Echocardiographic findings of patients

Echocardiographic parameters	Mean ± S.D
LVEDD	42.08 ± 4.70
LVEF	54.96 ± 4.23
E	74.47 ± 18.82
A	84.67 ± 22.01
E′	9.16 ± 2.39
E/ e′	7.95 ± 2.34
LVOT VTI	18.87 ± 3.98
TV annulus	29.10 ± 4.12
RV diameter	27.27 ± 3.97
RV S′	12.33 ± 2.36
TAPSE	19.04 ± 3.39
RVOT VTI	16.65 ± 3.24
PAT	97.63 ± 22.32
PASP	24.43 ± 6.52

LVEDD left ventricular end diastolic diameter, *LVEF* left ventricular ejection fraction, *LVOT* left ventricular outflow tract, *VTI* velocity time integral, *RV* right ventricle, *TAPSE* tricuspid annular plane systolic excursion, *RVOT* right ventricular outflow tract, *PAT* pulmonary acceleration time, *PASP* pulmonary artery systolic pressure

hospitalized patients and precipitate acute RV failure. These conditions include pulmonary embolism (PE), hypoxic pulmonary vasoconstriction, decrease in lung volume, excessive positive end-expiratory pressure, pneumonia, hyper-carbia, the use of α-agonists or a combination of all these factors [10]. In COVID-19 infection, the probable mechanisms capable for this condition include: (1) direct myocardial injury by virus; (2) increased RV afterload in ARDS; (3) hypoxic pulmonary vasoconstriction; (4) pulmonary micro-thrombosis, and (5) endothelial and microvascular injury [12]. Besides, RV dilation and dysfunction may also affect LV function by ventricular interdependence and paradoxical septal motion which causes reduction in LV volume and cardiac output [11]. We showed that RV dilation is a predictor of in-hospital and 30-days mortality in COVID-19 patients without any previous structural heart diseases. Similarly, Argulian et al. showed that RV dilation was predictive for in-hospital mortality in patients with COVID-19 [13]. Another observation was reported by Szekely et al. [10], who demonstrated that increased RV end diastolic area was significantly associated with mortality. We demonstrated that dyslipidemia, lower EF, older age, SBP, O2 saturation, PAT and diabetes were also independently predictors of 30-days mortality. In line with our study, the only baseline echocardiographic parameters significantly associated with mortality were low LVEF, elevated E/e' ratio, increased RV end diastolic area, and higher Tei index [10]. Therefore, identifying RV impairment could guide physicians to limit positive end-expiratory pressure and avoid hyper-capnic state, which could adversely affect RV performance by inducing pulmonary arteriolar vasoconstriction and increased RV afterload [9].

During acute RV pressure overload and resultant RV dysfunction and reduced cardiac output, systemic blood pressure will decrease, which may result in lowering coronary perfusion and additional reduction in RV contractility [10].

In Szekely et al. study, patients with shorter PAT were older, had more comorbidities, and worse lung disease, lower oxygen saturation, higher LV filling pressure, and higher biomarkers (D-dimer, BNP, Troponin-I, and CRP). They suggested that elevated pulmonary vascular resistance in COVID-19 infection is multifactorial and related to parenchymal lung disease, pulmonary vascular disease, and elevated left atrial pressure, all leading to cardiac injury [10]. Similar to our findings, there is a low prevalence of LV impairment in prior studies [9]. Previous case reports and publications showed that COVID-19 is a known cause of severe cardiac dysfunction such as fulminant myocarditis, even without common COVID-19 symptoms or pulmonary involvement [14–16]. Cardiac injury is prevalent in patients admitted with COVID-19 infection and related to high mortality rate [2] but LV systolic dysfunction is not very common.

This study performed on a larger sample size compared to previous investigations. We enrolled only patients with positive PCR test for COVID-19. Another strength of this study was excluding patients with previous underlying heart diseases, which helps to elucidate the pure effects of COVID-19 on cardiovascular system. Besides, all of evaluations were performed by a single cardiologist, which eliminates inter-observer discrepancies.

Limitation

Further studies on the reasons of RV dysfunction are needed.

Conclusion

RV systolic dysfunction was prevalent in patients with COVID-19 and can be correlated with higher mortality. Moreover, lower LVEF, shorter PAT and lower O2 saturation, older age, elevated SBP, dyslipidemia and diabetes were independently predictors of 30-days mortality. Using echocardiography for risk assessment in patients with COVID-19, especially with previous history of diabetes, hypertension and dyslipidemia, can be helpful and lead to better management.

Table 3 Patients' characteristics stratified by RV function

Parameter	RV dysfunction (n = 49)	Normal RV function (n = 93)	P value
Age (Mean ± S.D)	62.63 ± 16.29	59.26 ± 15.63	0.240
Gender (N, %)			
Male	23, 46.9	59, 63.4	0.044
Female	26, 53.1	34, 36.6	
Heart rate (Mean ± S.D)	100.63 ± 19.69	103.24 ± 19.21	0.464
Systolic blood pressure (Mean ± S.D)	135.40 ± 19.86	135.39 ± 21.45	0.999
Diastolic blood pressure (Mean ± S.D)	83.73 ± 12.77	85.48 ± 16.01	0.504
O_2 saturation (Mean ± S.D)	88.10 ± 5.88	88.42 ± 8.85	0.808
Laboratory data (Mean ± S.D)			
Urea	41.18 ± 16.77	44.46 ± 17.37	0.556
Creatinine	1.23 ± 0.7	1.40 ± 0.8	0.595
Calcium	8.87 ± 0.99	8.35 ± 0.78	0.005
Lactate dehydrogenase	598.51 ± 211.97	606.82 ± 241.96	0.835
Creatine phosphokinase	472.00 ± 181.02	243.50 ± 146.01	0.033
White blood cell	7302.04 ± 3021.20	9621.97 ± 6665.41	0.064
Hemoglobin	13.58 ± 2.16	12.92 ± 2.06	0.108
RDW	14.45 ± 1.96	13.84 ± 1.57	0.082
PMN	76.03 ± 8.69	78.76 ± 9.61	0.985
Lymph	18.16 ± 7.04	15.87 ± 8.37	0.090
Platelet			
CRP	80.44 ± 63.8	92.69 ± 79.49	0.370
ESR	47.25 ± 30.33	39.47 ± 30.10	0.217
ECG assessment			
ECG rhythm (%)			
Sinus tachycardia	48.9	49.4	0.740
Atrial fibrillation	0	1.3	
Normal sinus rhythm	51.1	49.4	
ECG axis (%)			
Normal axis	73.3	74.7	0.543
LAD	22.2	15.6	
RAD	4.4	10.1	
ECG abnormality (%)			
RBBB	8.2	6.6	0.42
LAHB	12.2	9.9	
LBBB	4.1	0	
LAHB + RBBB	4.1	0	
Symptoms (%)			
Chest pain	4.1	0	0.121
Nausea and vomiting	12.2	13.2	0.550
Diarrhea	8.2	2.2	0.112
Dyspnea	87.8	91.2	0.354
Cough	55.1	56	0.528
Fever	51	49.5	0.50
Weakness	26.5	45.1	0.024
Confusion	8.2	4.4	0.289
Comorbidities (%)			
Diabetes	28.6	22	0.253
Addiction	5.6	6.3	0.667
Smoker	2.1	3.5	0.580
Dyslipidemia	12.2	11	0.513
Hypertension	20.4	26.4	0.284

Table 3 (continued)

Parameter	RV dysfunction (n = 49)	Normal RV function (n = 93)	P value
History of stroke	0	4.4	0.174
Asthma	4.1	6.6	0.424
COPD	0	2.2	0.421
Malignancy	2.2	4.1	0.437
ESRD	4.1	3.3	0.429
CT scan findings (%)			
Bilateral peripheral GGO	46.9	34.1	0.095
Bilateral diffused GGO	32.7	34.7	0.358
Unilateral GGO	8.2	17.6	0.10
Bilateral pleural effusion	8.2	11	0.416
Consolidation	69.4	59.3	0.161
Lymphadenopathy	24.5	33	0.198
Echocardiographic parameters			
LVEDD (Mean ± S.D)	41.61 ± 4.64	42.27 ± 4.77	0.089
E/ e′ (Mean ± S.D)	8.05 ± 3.09	7.90 ± 1.85	0.755
LVEF (n, %)			
≥ 55	34, 69.4	79, 84.9	0.024
50–54	9, 18.4	12, 12.9	
40–49	6, 12.2	2, 2.2	
Pulmonary hypertension (n, %)	6, 12.8	5, 5.6	0.193
RV size (n, %)			
Normal	41, 83.7	85, 90.4	0.145
Mild enlargement	6, 12.2	8, 8.6	
Moderate enlargement	2, 1.4	0	

RDW red cell distribution width, *PMN* polymorphonuclear leukocytes, *Lymph* lymphocyte, *CRP* C-reactive protein, *ESR* erythrocyte sedimentation rate, *ECG* electrocardiogram, *LAD* left axis deviation, *RAD* right axis deviation, *RBBB* right bundle branch block, *LBBB* left bundle branch block, *LAHB* left anterior hemi block, *COPD* chronic obstructive pulmonary disease, *ESRD* end stage renal disease, *GGO* ground glass opacification, *LVEDD* left ventricular end diastolic diameter, *LVEF* left ventricular ejection fraction, *RV* right ventricle

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Authors contributions FK designed the study, collected data and performed echocardiographic assessments, wrote the first draft of the article and approved the final version; HP participated in data analysis and echocardiographic assessments, read the final version and approved it; AS participated in data collection, statistical analysis and writing first draft of the article. AE designed the study, wrote the first draft of the article and approved the final version.

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Data availability The datasets used or analyzed in the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors declare that they have no competing interests.

Ethical approval and consent to participate This study was approved by the ethical committee of the medical faculty of Mashhad University of Medical Sciences. The ethical code of the study was IR.MUMS.REC.1399.016. All patients filled a written informed consent form before entering the study.

Consent for publication All patients took consent to publish their data anonymously.

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