



## Original Article

## Patterns of cardiovascular diseases in COVID-19 patients admitted to tertiary cardiac care centre



Beerasha Puttegowda, MD, DM, Arun B. Shivashankarappa, MD, DM, DNB<sup>\*</sup>,  
 Suhradip Dutta, MD, (DM), Rama Chikkamuniswamy, MD, DM, Prabhavathi Bhat, MD, DM,  
 Shanmugam Krishnan, MD, DM, Manjunath C. Nanjappa, MD, DM, FRCP, FACC, FICC

Department of Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bangalore, Karnataka, India

## ARTICLE INFO

## Article history:

Received 17 May 2021

Received in revised form

16 October 2021

Accepted 21 October 2021

Available online 22 October 2021

## Keywords:

COVID-19

ACS

STEMI

NSTE-ACS

Myocarditis

## ABSTRACT

**Objectives:** To analyse the pattern of cardiovascular diseases (CVDs) in COVID-19 patients admitted to tertiary cardiac care centre.

**Methods:** We retrospectively analysed 511 adult patients admitted between July 1, 2020, and November 30, 2020, with COVID-19 infection and having either new onset or pre-existing CVDs. Clinical features, electrocardiogram (ECG), echocardiography, chest X-ray, biomarkers, haematological and biochemical parameters were analysed.

**Results:** The mean age of the patients was  $56.62 \pm 14.74$  years. Male: Female ratio was 2.78:1. Pre-existing CVDs were present in 258 patients (50.5%). The most common cardiovascular manifestation was acute coronary syndrome (ACS), seen in 259 patients (50.7%). ST-segment elevation myocardial infarction (STEMI) was more common than non-ST-segment elevation ACS (NSTEMI-ACS). Possible myocarditis was seen in 52 patients (10.1%). Rhythm and conduction abnormalities were noted in 144 patients (28.2%), the most common being QT prolongation, seen in 51 patients (10%). In-hospital mortality occurred in 97 patients (18.9%). Age, serum Ferritin level, D-dimer, NT-pro-BNP and total leukocyte count were significantly higher among patients with in-hospital mortality compared to survival group. Blood lymphocyte count and Haemoglobin level were significantly lower in mortality-group, compared to survival-group. Incidence of pre-existing CVDs, cardiogenic shock, heart failure, atrial fibrillation (AF), and renal failure were significantly higher in mortality-group compared to survival-group.

**Conclusion:** The most common CVD in COVID-19 patients in our study was ACS. STEMI was more common than NSTEMI-ACS. Advanced age, elevated serum ferritin, D-dimer, NT pro-BNP, leucocytosis, lymphopenia, lower haemoglobin, pre-existing CVDs heart failure, cardiogenic shock, AF and renal failure were associated with increased mortality in these patients.

© 2021 Cardiological Society of India. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

### 1. Introduction

The manifestations of coronavirus disease 2019 (COVID-19) infection ranges from mild illness to severe pneumonia, multi-organ failure, and death.<sup>1,2</sup> Cardiac complications are common, seen in 20–25% of COVID-19 infected patients and are associated with increased mortality.<sup>2,3</sup> The estimated mortality rate in COVID-19 is about 3.4%. But, in the presence of cardiovascular disease

(CVD), mortality rate may be increased to about 10.5%.<sup>4,5</sup> This study was conducted to analyse the pattern of CVDs in COVID-19 patients admitted to tertiary cardiac care centre.

### 2. Materials and methods

#### 2.1. Study setting

We retrospectively studied 511 adult patients admitted between July 1st, 2020 and November 30th, 2020 to our institute with COVID-19 infection and having either new onset or pre-existing CVD. Since it was a retrospective study, consent was not required. The study was conducted in accordance with the Declaration of

<sup>\*</sup> Corresponding author. Department of Cardiology, Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bannergatta road, Jayanagar-9th Block, Bangalore, Karnataka, India. Tel.: +91 9972664753.

E-mail address: [arunbsmedicine@gmail.com](mailto:arunbsmedicine@gmail.com) (A.B. Shivashankarappa).

Helsinki. Ethical approval was obtained from the Institutional Ethics committee.

### 2.2. Definitions

CVDs included were coronary artery disease, myocarditis, heart failure, valvular heart disease, cardiomyopathies, congenital heart diseases, pericardial diseases, arrhythmias, cerebrovascular events (CVEs), peripheral arterial diseases and venous thromboembolic diseases. Acute myocardial injury and myocardial infarction were defined according to the Fourth Universal Definition of Myocardial Infarction. Myocardial injury was defined as an increase in serum levels of cardiac troponin-T above the 99th percentile of upper reference limit. It could be ischemic (acute coronary syndrome) or non-ischemic (myocarditis). In our study, possible myocarditis was diagnosed based on clinical background, electrocardiography (ECG), echocardiography and elevated troponin T. ECG features suggestive of possible myocarditis were sinus tachycardia, global ST-T changes and arrhythmias. Echocardiographic features were global hypokinesia and reduced left ventricular (LV) ejection fraction. Although cardiac magnetic resonance imaging (C-MRI) is commonly used to diagnose myocarditis, it was associated with risk of infection to staff and potential contamination of lab and instruments. Hence, it was not done for diagnosing myocarditis in our study.

### 2.3. Inclusion criteria

Patients, aged  $\geq 18$  years, diagnosed with COVID-19 infection by a positive rapid antigen test or reverse-transcriptase polymerase chain reaction assay for severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) and admitted with CVDs.

### 2.4. Exclusion criteria

COVID-19 patients without CVDs.

### 2.5. Study design

Data was collected from patients' admission files using the self-designed proforma. Demographic data, comorbid conditions, medications, physical examination, and laboratory findings including troponin-T, N-terminal pro-Brain natriuretic peptide (NT-pro-BNP), D-dimer and Ferritin levels were analysed. ECG, chest X-ray and echocardiography of all patients were systematically studied.

### 2.6. Statistics

A descriptive and inferential statistical analysis were done. The quantitative variables were expressed as mean  $\pm$  standard deviation (S.D.) and compared between groups using unpaired *t*-test. The qualitative variables were expressed as number (percentage) and compared between groups using Chi-square/Fisher's exact test. A *p*-value  $< 0.05$  was considered statistically significant. SPSS version 22.0 software was used for statistical analysis.

## 3. Results

511 patients were studied. 376 (73.5%) were male. Table 1 shows the baseline characteristics, pre-existing CVDs and presenting symptoms. 209 patients (40.9%) were above 60 years. Comorbidities were present in 360 patients (70.5%), with diabetes mellitus being the most common. Pre-existing CVDs were present in 258

**Table 1**  
Baseline characteristics and presenting complaints.

	No. of patients (n = 511)	Percent
Age (years)	56.62 $\pm$ 14.74	-
Sex ratio	2.78:1	-
<b>Comorbidities</b>	<b>360</b>	<b>70.5</b>
Hypertension	185	36.2
Diabetes Mellitus	207	40.5
Renal Failure	90	17.6
Chronic obstructive pulmonary disease	21	4.1
Dyslipidemia	63	12.3
<b>Pre-existing CVD<sup>a</sup></b>	<b>258</b>	<b>50.5</b>
Ischemic heart disease	150	29.4
Valvular heart disease	40	7.8
Dilated cardiomyopathy	30	5.9
Congenital heart diseases	2	0.4
Heart failure	145	28.4
Cerebrovascular events	8	1.6
Others	36	7.0
<b>Presenting complaints</b>		
Chest Pain	296	57.9
Dyspnea	230	45.0
Giddiness	17	3.3
Syncope	3	0.6
Palpitations	21	4.1
Fatigue	38	7.4
Pedal edema	5	1.0
Cough	30	5.9
Fever	37	7.2

<sup>a</sup> CVD: Cardiovascular diseases.

patients (50.5%), the most common being ischemic heart disease (IHD). The most common symptom at admission was chest pain.

### 3.1. Acute coronary syndrome (ACS)

The most common cardiovascular manifestation was acute coronary syndrome (ACS). ST-segment elevation myocardial infarction (STEMI) was seen in 161 patients (31.5%). It was more common than non-ST-segment elevation ACS (NSTEMI-ACS), which was seen in 99 patients (19.4%). Other cardiovascular manifestations of COVID-19 are summarised in Table 2.

Among 161 patients with STEMI, 76 patients (47.2%) presented within the window period and were eligible for thrombolysis. 72 were lysed with intravenous Streptokinase, 2 with Reteplase, 1 each with Tenecteplase and Urokinase. 85 patients (52.8%) with STEMI presented late and were not eligible for thrombolysis. Hence, they were treated conservatively. Cardiogenic shock was seen in 20 patients (12.4%) and mortality in 16 patients (9.9%).

Patients with NSTEMI-ACS were managed medically. 13 patients (13.1%) had cardiogenic shock and 22 patients (22.2%) had mortality.

Among patients with ACS, 2 patients underwent coronary angiogram (CAG), 1 of them had double vessel disease and underwent percutaneous coronary intervention with stenting to both the vessels. The other had diffuse triple vessel disease and was managed with guideline-directed medical therapy.

### 3.2. Myocarditis and pericarditis

Non-ischemic myocardial injury in the form of possible myocarditis was seen in 52 patients (10.1%). They presented with atypical chest pain, heart failure and arrhythmias. Among them, 4 patients had associated pericarditis, resulting in myopericarditis. On echocardiography, mild, moderate and severe LV systolic dysfunction was seen in 27 patients, 15 patients and 10 patients respectively. All of them were treated medically. 10 patients (19.2%)

**Table 2**  
Cardiovascular manifestations.

	No. of patients (n = 511)	Percent
<b>Acute coronary syndrome</b>	<b>259</b>	<b>50.7</b>
<b>Myocarditis</b>	<b>52</b>	<b>10.1</b>
<b>Heart failure</b>	<b>227</b>	<b>44.4</b>
<b>Cardiogenic shock</b>	<b>55</b>	<b>10.8</b>
<b>Pulmonary hypertension</b>	<b>155</b>	<b>30.3</b>
<b>Rhythm and conduction disturbance</b>	<b>144</b>	<b>28.2</b>
Sinus tachycardia	46	9.0
Ventricular tachyarrhythmias	28	5.5
Ventricular tachycardia	16	3.1
Ventricular fibrillation	1	0.2
Ventricular premature complexes	11	2.2
Atrial fibrillation	24	4.7
Sinus bradycardia	14	2.7
A.V. nodal block <sup>a</sup>	27	5.2
First degree A.V. nodal block	2	0.4
2:1 A.V. block	2	0.4
New onset complete A.V. nodal block	14	2.7
QTc Prolongation	51	10.0
Right bundle branch block	5	1.0
Left bundle branch block	16	3.1
<b>Venous thromboembolism</b>	<b>11</b>	<b>21.5</b>
<b>Cerebrovascular events</b>	<b>2</b>	<b>0.4</b>

<sup>a</sup> A.V.: Atrio-ventricular.

had cardiogenic shock. Isolated pericardial effusion were noted in 2 patients. One had mild and the other had moderate pericardial effusion. They presented with atypical chest pain. On further investigations, there was no other obvious cause. They had normal biventricular function. Hence, they were considered probably due to COVID-19 infection.

### 3.3. Rhythm and conduction abnormalities

They were noted in 144 patients (28.2%). The most common was QTc prolongation, seen in 51 patients (10%). 30 of them had associated ACS, 15 had possible myocarditis and 3 had hypokalemia. 3 patients had QTc prolongation after treatment with hydroxychloroquine and the drug was stopped. Torsedes de pointes was seen in 2 and were treated with defibrillation. Rest of the rhythm and conduction abnormalities are given in Table 2.

**Table 3**  
Comparison of parameters between group with asymptomatic and symptomatic COVID-19 respiratory disease.

Parameters	Asymptomatic (n = 242)	Symptomatic (n = 269)	p-value
Age (Mean ± SD, years)	54.14 ± 14.69	58.84 ± 14.46	<0.001
Serum Ferritin (Mean ± SD, µg/L)	362.78 ± 339.71	505.52 ± 481.86	<0.001
Serum Procalcitonin (Mean ± SD, ng/ml)	6.67 ± 91.65	2.29 ± 26.33	0.453
D-Dimer level (Mean ± SD, µg/ml)	0.75 ± 2.78	1.13 ± 2.49	0.099
Serum Troponin T (Mean ± SD, ng/ml)	0.38 ± 0.65	0.40 ± 0.75	0.728
Total leukocyte count (Mean ± SD, cells/µl)	9803.24 ± 4777.07	12449.78 ± 5741.68	<0.001
Lymphocyte (Mean ± SD, % of total leukocyte count)	19.26 ± 10.29	15.21 ± 9.96	<0.001
Haemoglobin (Mean ± SD, g/dl)	12.99 ± 1.92	12.44 ± 2.00	0.002
Platelet (Mean ± SD, lakhs/µl)	2.61 ± 0.93	3.73 ± 1.81	0.274
NT-Pro BNP (Mean ± SD, pg/ml)	2353.25 ± 4121.53	5716.29 ± 8563.18	<0.001
Cardiogenic shock, n (%)	12 (4.1%), 230	43 (16%), 226	<0.001
Heart failure, n (%)	85 (35.1%)	142 (52.8%)	0.001
Renal failure, n (%)	23 (9.5%)	67 (24.9%)	<0.001
Diabetes Mellitus, n (%)	80 (33.1%)	127 (47.2%)	0.001
Hypertension, n (%)	70 (28.9%)	115 (42.8%)	0.001
Chronic obstructive pulmonary disease, n (%)	6 (2.5%)	15 (5.6%)	0.078
Pre-existing CVD, n (%)	120 (49.6%), 122	138 (51.3%), 131	0.72
Atrial Fibrillation, n (%)	6 (2.5%)	18 (6.7%)	0.025
Mortality, n (%)	22 (9.1%)	76 (28.3%)	<0.001

NT-pro BNP: N-terminal pro-Brain Natriuretic peptide, CVD: Cardiovascular diseases. SD: Standard deviation.

### 3.4. Venous thromboembolism (VTE)

VTE was seen in 11 patients. 4 patients had deep vein thrombosis (DVT) and 6 had pulmonary thromboembolism (PTE). 1 patient had DVT with PTE. 1 patient with DVT had multiple site thrombosis. 3 patients with massive PTE were thrombolysed with streptokinase. 2 of them died. Rest of the patients with PTE and all patients with DVT were treated with anticoagulation.

### 3.5. Pattern of respiratory system involvement

242 patients were asymptomatic. 269 patients had symptomatic COVID-19. Pulmonary involvement was seen in 245 patients (47.9%). Comparison between patients with asymptomatic and symptomatic respiratory disease is given in Table 3. Age, serum Ferritin, leukocyte count and NT-pro-BNP were higher in symptomatic group compared to asymptomatic group. Also, heart failure, cardiogenic shock, renal failure, pre-existing CVDs, atrial fibrillation and mortality rate were higher in symptomatic group, compared to asymptomatic.

### 3.6. Abnormalities in biomarkers, hematological and biochemical parameters

Serum Troponin T was elevated in 311 patients (60.9%) suggestive of myocardial injury. NT pro-BNP was elevated above the age specific cut off (Age<45 years: >450 ng/l, 45–75 years: >900 ng/l, >75 years: >1800 ng/l) in 270 patients (52.8%), suggestive of heart failure. D-dimer was raised in 175 patients (34.2%). Ferritin and Procalcitonin were elevated in 139 (27.2%) and 82 patients (16.1%) respectively. Leukocytosis (216 patients, 42.4%) and lymphopenia (247 patients, 48.3%) were the common haematological abnormalities. Thrombocytopenia and thrombocytosis were present in 46 (9.0%) and 42 patients (8.2%) respectively. Hyperkalemia was the most common dyselectrolytemia (21 patients, 4.1%) noted, followed by hypokalemia (13 patients, 2.5%). There was positive correlation between troponin T and leukocyte count. (Pearson's correlation coefficient = 0.113, p = 0.011).

**Table 4**  
Comparison of parameters with respect to Outcome.

Parameters	Patients who survived to discharge (n = 414)	Patients with in-hospital mortality (n = 97)	p-value
Age (Mean ± SD, years)	55.72 ± 14.50	60.41 ± 15.21	<b>0.005</b>
Serum Ferritin (Mean ± SD, µg/L)	386.13 ± 380.25	656.18 ± 530.39	< <b>0.001</b>
Serum Procalcitonin (Mean ± SD, ng/ml)	3.77 ± 70.01	6.86 ± 44.55	0.677
D-Dimer level (Mean ± SD, µg/ml)	0.71 ± 1.8	1.98 ± 4.64	< <b>0.001</b>
Serum Troponin T (Mean ± SD, ng/ml)	0.39 ± 0.69	0.38 ± 0.77	0.890
Total leukocyte count (Mean ± SD, cells/µl)	10572.04 ± 5238.92	13816.43 ± 5636.71	< <b>0.001</b>
Lymphocyte (Mean ± SD,% of total leukocyte count)	18.20 ± 10.45	11.51 ± 7.34	< <b>0.001</b>
Haemoglobin (Mean ± SD, g/dl)	12.82 ± 1.95	12.22 ± 2.07	<b>0.007</b>
Platelet count (Mean ± SD, lakhs/µl)	3.35 ± 12.76	2.56 ± 1.03	0.538
NT-pro-BNP, <sup>a</sup> (Mean ± SD, pg/ml)	3531.74 ± 6756.05	6597.43 ± 7617.67	< <b>0.001</b>
Cardiogenic shock, n (%)	10 (2.4%)	45 (46.4%)	< <b>0.001</b>
Heart failure, n (%)	172 (41.5%)	55 (56.7%)	<b>0.009</b>
Renal failure, n (%)	61 (14.7%)	29 (29.9%)	<b>0.001</b>
Diabetes Mellitus, n (%)	165 (39.8%)	42 (43.3%)	0.566
Hypertension, n (%)	144 (34.8%)	41 (42.3%)	0.197
COPD,**n (%)	15 (3.6%)	6 (6.2%)	0.258
Pre-existing CVD, <sup>b</sup> n (%)	196 (47.3%)	62 (63.9%)	<b>0.003</b>
Atrial fibrillation, n (%)	15 (3.6%)	9 (9.3%)	<b>0.027</b>

<sup>a</sup> NT-pro-BNP:N-terminal pro-Brain natriuretic peptide, \*\*COPD:Chronic obstructive pulmonary diseases.

<sup>b</sup> CVD:Cardiovascular diseases.

### 3.7. In-hospital outcome

Mortality occurred in 97 patients (18.9%). Comparison between in-hospital survival group and mortality group is shown in Table 4. Age, serum Ferritin level, D-dimer, NT-pro-BNP and total leukocyte count were significantly higher among patients with mortality, compared to those who survived to discharge. Blood lymphocyte count and hemoglobin level were significantly lower in mortality-group, compared to survival-group.

Incidence of pre-existing CVD, heart failure, cardiogenic shock, renal failure and atrial fibrillation (AF) were significantly higher in mortality-group compared to survival-group.

## 4. Discussion

Cardiac involvement is common, especially in hospitalized COVID-19 patients. Patients with cardiac risk factors and pre-existing CVDs tend to have more severe form of disease with worse clinical outcomes. In our study, pre-existing CVDs were present in 50.5%, which was higher than previous studies.<sup>6,7</sup> This association is possibly due to CVDs being more prevalent in patients with advancing age, who have a functionally impaired immune system, or elevated levels of Angiotension converting enzyme-2 (ACE-2), which also predisposes them to COVID-19.<sup>7</sup>

### 4.1. Acute myocardial injury

Acute myocardial injury was seen in 60.8% in our study. This is much higher than in other studies,<sup>5,8</sup> probably due to admission of selective cardiac cases at our institute. Guo et al demonstrated that elevated troponin T levels are associated with higher levels of biomarkers such as C-reactive protein (CRP), procalcitonin, greater leukocyte counts, indicating that myocardial injury correlates with the severity of inflammation.<sup>3</sup> Similarly, in our study, there was positive correlation between troponin T and leukocyte count. Various mechanisms of cardiac involvement in COVID-19 have been proposed. One potential mechanism is direct myocardial involvement mediated by ACE2. Other mechanisms include cytokine storm and hypoxia-induced excessive intracellular calcium, leading to cardiac myocyte apoptosis.<sup>7</sup>

### 4.1.1. ACS

Ischemic myocardial injury results from cytokine storm and catecholamine surge, predisposing to plaque rupture or erosion (Type 1 myocardial infarction). It may be also due to hypoxic injury, supply-demand mismatch, coronary artery spasm and thrombosis (Type 2 myocardial infarction).<sup>3,9</sup> STEMI in COVID patients has high mortality (33%) as shown in North American COVID-19 Myocardial Infarction Registry.<sup>10</sup> However, in our study, STEMI patients had a mortality rate of 9.9%. and NSTEMI-ACS had mortality rate of 22.2%.

### 4.1.2. Myocarditis and pericarditis

The association between coronavirus infection and myocarditis is not new, as it was also noticed during Middle East respiratory syndrome coronavirus (MERS-CoV).<sup>11</sup> In our study, possible myocarditis was diagnosed based on troponin T, ECG and echocardiographic findings and it was noted in 10.1%. Based on similar criteria, Deng et al found 12.5% cases of possible myocarditis in their retrospective study.<sup>12</sup> However, a cardiac-MRI based study revealed higher incidence (78%) of myocarditis.<sup>13</sup>

A case of COVID-19 complicated with myopericarditis causing pericardial effusion and cardiac tamponade requiring pericardiocentesis, has been reported in previous literature.<sup>14</sup> In our study, pericardial effusion was seen in 2 patients (0.4%).

### 4.2. Heart failure

New-onset heart failure and acute decompensation of chronic heart failure (ADHF) have been documented in patients with COVID-19. Heart failure is postulated to occur in COVID-19 due to the over-activation of immune system, and downregulation of ACE2, leading to increased levels of Angiotensin II.<sup>15</sup> In our study, 227 patients (44.4%) had heart failure. Among them, majority had ADHF (145 patients, 63.9%). Remaining were new onset heart failure. In previous studies, incidence of heart failure in admitted patients with COVID-19 ranged from 23 to 33%.<sup>16,17</sup> In a meta-analysis of 35 studies, patients with pre-existing heart failure had 8 times higher mortality.<sup>18</sup> In our study, mortality in heart failure patients was 24.2%, which was 1.6 times higher, compared to those without heart failure. (14.8%).

### 4.3. Cardiogenic shock

Studies from Germany and the USA have reported cardiogenic shock as a significant complication of COVID-19.<sup>19,20</sup> A systematic review showed that approximately 8% of patients developed heart failure or cardiogenic shock as a manifestation of COVID-19.<sup>21</sup> In our study, cardiogenic shock was seen in 55 patients (10.8%). Case series from Spain showed 75% mortality with cardiogenic shock due to COVID-19.<sup>22</sup> In our study, cardiogenic shock was associated with mortality rate of 81.8%.

### 4.4. Rhythm and conduction abnormalities

Arrhythmia in COVID-19 infection can be due to acute cardiac injury from different etiologies such as ischemia, direct myocardial damage, systemic inflammatory response syndrome or it could be due to the effects of drugs used to treat COVID-19. In a study by Dawei Wang, 16.7% had arrhythmia.<sup>6</sup> In our study, rhythm and conduction disturbance was seen in 28.2%. There is concern about hypokalemia in COVID-19 patients, because of the interaction of the virus with the renin-angiotensin-aldosterone system (RAAS).<sup>7</sup> Hypokalemia increases the vulnerability to various arrhythmia, and in our study, it was seen in 2.5% of patients.

QT<sub>c</sub> prolongation is common in these patients as noted in our study and also in a previous study.<sup>23</sup> Advanced age, renal failure, underlying CVDs, myocardial injury, and drugs like hydroxychloroquine or azithromycin, contribute to it.<sup>23</sup>

### 4.5. In-hospital outcome

In hospital mortality in our study was 18.9%, which was higher than the study by Tang et al (11.5%).<sup>24</sup> Tang et al reported elevated D-dimer level, approximately 3.5 times higher in mortality cases than survivors.<sup>24</sup> In our study, mortality cases had elevated D-dimer, 2.8 times higher, compared to survivors. A multinational observational study showed that COVID-19 complicated with heart failure was associated with an increased mortality,<sup>25</sup> which was also noted in our study. In a meta-analysis, one-fourth of the hospitalized patients with COVID-19 develop CVDs and arrhythmia, which increased the mortality by 20 times.<sup>18</sup> In our study also, CVDs and AF were associated with increased mortality.

### 4.6. Limitations

This study was conducted in tertiary cardiac care centre. Hence, only COVID-19 patients with CVDs were included. Our study might not reflect the true cardiovascular complications of COVID-19 due to referral bias. Hence, the results cannot be applied to general population with COVID-19 infection. It was a retrospective study and hence the follow up data is not available. CAG was not done in most of the patients with ACS. Acute myocarditis was diagnosed only based on ECG, echocardiography and troponin T. Cardiac MRI was not done. Hence, subclinical myocarditis might have been missed. Procoagulant workup was not done for VTE patients.

## 5. Conclusion

The most common CVD in COVID-19 patients was ACS. STEMI was more common than NSTEMI-ACS. Advanced age, elevated serum Ferritin level, D-dimer, NT-pro-BNP, leukocytosis, lymphopenia, lower hemoglobin level, presence of pre-existing CVDs, heart

failure, cardiogenic shock, AF and renal failure were important predictors of mortality in these patients.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ihj.2021.10.007>.

## References

- Bhatraju PK, Ghassemieh BJ, Nichols M, et al. Covid-19 in critically ill patients in the Seattle region: case series. *N Engl J Med*. 2020;382:2012–2022.
- Zhou F, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet*. 2020;395:1054–1062.
- Guo T, Fan Y, Chen M, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019. *JAMA Cardiol*. 2020;5(7):811–818.
- Yang X, Yu Y, Xu J, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. *Lancet Respir Med*. 2020;8:475–481.
- Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet*. 2020;395:497–506.
- Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. *J Am Med Assoc*. 2020 Mar 17;323(11):1061–1069.
- Clerkin KJ, Fried JA, Raikhelkar J, et al. COVID-19 and cardiovascular disease. *Circulation*. 2020 May 19;141(20):1648–1655.
- Guan WJ, Ni ZY, Hu Y, et al. Clinical characteristics of coronavirus disease 2019 in China. *N Engl J Med*. 2020;382:1708–1720.
- Schoenhagen P, Tuzcu EM, Ellis SG. Plaque vulnerability, plaque rupture, and acute coronary syndromes: (multi)-focal manifestation of a systemic disease process. *Circulation*. 2002 Aug 13;106(7):760–762.
- Garcia S, Dehghani P, Grines C, et al. Initial findings from the North American COVID-19 myocardial infarction registry. *J Am Coll Cardiol*. 2021;77(16):1994–2003.
- Alhobgani T. Acute myocarditis associated with novel Middle East Respiratory Syndrome coronavirus. *Ann Saudi Med*. 2016 Jan-Feb;36(1):78–80.
- Deng Q, Hu B, Zhang Y, et al. Suspected myocardial injury in patients with COVID-19: evidence from front-line clinical observation in Wuhan, China. *Int J Cardiol*. 2020;311:116–121.
- Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5:1265–1273.
- Hua A, O'Gallagher K, Sado D, Byrne J. Life-threatening cardiac tamponade complicating myo-pericarditis in COVID-19. *Eur Heart J*. 2020 Jun 07;41(22):2130.
- Hari Krishnan S, Mohanan PP, Chopra VK, et al. Cardiological society of India position statement on COVID-19 and heart failure. *Indian Heart J*. 2020;72:75–81.
- Xu XW, Wu XX, Jiang XG, et al. Clinical findings in a group of patients infected with the 2019 novel coronavirus (SARS-CoV-2) outside of Wuhan, China: retrospective case series. *BMJ*. 2020 Feb 19;368:m606.
- Zhou F, Yu T, Du R, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. *Lancet*. 2020 Mar 28;395:1054–1062, 10229.
- Momtazmanesh S, Shobeiri P, Hanaei S, et al. Cardiovascular disease in COVID-19: a systematic review and meta-analysis of 10,898 patients and proposal of a triage risk stratification tool. *Egypt Heart J*. 2020;72:41.
- Bemtgen X, Krüger K, Supady A, et al. First successful treatment of COVID-19 induced refractory cardiogenic plus vasoplegic shock by combination of pVAD and ECMO—a case report. *Am Soc Artif Intern Organs J*. 2020;66:607–09.
- Harari R, Bangalore S, Chang E, Shah B. COVID-19 complicated by acute myocardial infarction with extensive thrombus burden and cardiogenic shock. *Cathet Cardiovasc Interv*. 2021;97(5):E661–E666.
- Shafi AMA, Shaikh SA, Shirke MM, Iddawela S, Harky A. Cardiac manifestations in COVID-19 patients-A systematic review. *J Card Surg*. 2020 Aug;35(8):1988–2008.
- Sánchez-Recalde Á, Solano-López J, Miguelena-Hycka J, Martín- Pinacho JJ, Sanmartín M, Zamorano JL. COVID-19 and cardiogenic shock. Different cardiovascular presentations with high mortality. *Rev Esp Cardiol*. 2020;73(8):669–672.
- Changal K, Paternite D, Mack S, et al. Coronavirus disease 2019 (COVID-19) and QTc prolongation. *BMC Cardiovasc Disord*. 2021;21:158.
- Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *J Thromb Haemostasis*. 2020;18:844–847.
- Mehra MR, Desai SS, Kuy S, Henry TD, Patel AN. Cardiovascular disease drug therapy, and mortality in COVID-19. *N Engl J Med*. 2020;382(25):e102.