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A study of cardiovascular involvement in Covid – 19 patients

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Abstract
Aim: To study the Cardiovascular involvement in COVID-19 Patients.
Study Design: It is an observational study.
Study Group: Among 200 patients who were COVID-19 positive, 40 patients had different cardiovascular manifestations.
Methods: Study group consisted of 200 patients hospitalized COVID-19 patients over 5 months from July 2020 to November 2020, were evaluated for a symptomatic profile, blood investigations, chest radiograph data, classified according to COVID-19 severity, and all investigations related to cardiovascular involvement.
Number of Patients: 200 patients.
Period of the Study: 5 months - From July 2020 to November 2020
Inclusion Criteria: All those who were diagnosed with COVID-19 positive and with various cardiac diseases.
Exclusion Criteria: All those without COVID-19 positive and cardiac disease or with COVID-19 positive and without cardiac disease.
Investigations: Electrocardiogram, 2D ECHO, Troponin I, Complete Blood Count with Differential Count, CRP, Interleukin 6, D-Dimer, LDH, Ferritin.

Keywords: Covid – 19, myocardial infraction, niv support, electrocardiogram

Introduction

COVID-19 or CORONA VIRUS DISEASE-2019 is caused by SARS-COV-2, a Beta coronavirus. It rapidly spread, resulting in a huge number of cases throughout the world, declaring it a global pandemic by WHO on March 11, 2020. COVID-19 is predominantly a Respiratory disease with severity ranging from Mild to Fatal. The mild disease was reported in 81%, severe disease with dyspnea, hypoxia, >50% of lung involvement was reported in 14%, a critical disease with respiratory failure, shock, multi-organ dysfunction was reported in 5%, and the overall case fatality rate was 2.3%. The prevalence of cardiovascular system involvement in COVID-19 is around 8%. COVID-19 patients can present with myocarditis, Type 1 or Type 2 MI, NSTEMI, dilated cardiomyopathy, heart failure, severe pulmonary artery hypertension, pericardial effusion, pericarditis, acute and chronic pulmonary thromboembolism, and arrhythmias. With preexisting cardio, vascular disease patients can develop adverse cardiovascular outcomes Including-Heart Failure, Cardiogenic Shock, Arrhythmias, recurrent myocardial infarction, and Thromboembolic disease result from the occurrence of coronavirus disease 2019. In them, the final outcome is worse, increasing the case fatality rate. Patients with COVID-19 and having cardiovascular system involvement had prolonged hospital stay and required ICU care. More patients required Noninvasive ventilatory support or HFNC support, or ventilatory support, and the case fatality rate was more than COVID-19 patients with no cardiac involvement.

It is crucial to diagnose the cardiovascular disease early in COVID 19 and treat it early to mitigate the disease's complications and reduce morbidity and mortality.

Our study was conducted to determine various cardiac abnormalities associated with Coronavirus disease 2019, presentation of the patients, electrocardiographic changes, and the prognosis associated with the same.

Results

We studied 200 patients with Corona Virus Disease 2019; among them, we selected 40 cases with cardiovascular involvement and COVID 19 positive. These patients presented with complaints of fever, cough, cold, anosmia, chest pain, breathlessness, palpitations, loose stools either to the emergency department or to the COVID-19 clinic of Malla Reddy Narayana Multispecialty hospital, and were admitted in COVID-19 isolation ward or COVID-19 ICU, based on the severity of the disease. Among 40 patients, 29 patients were males, and 11 were females, with 6 patients aged less than 45 years, 20 patients aged between 45 and 65, and 14 patients greater than 65.

Patients diagnosed with myocardial infarction had chest pain, fever, cough, and cold for 2-3 days but presented with progressing dyspnea and chest pain.

1. 11 Patients - History of old myocardial infarction. They had undergone either stenting or CABG;
    - 4 Patients - Presented with dilated cardiomyopathy with severe left ventricular dysfunction and heart

failure
   - 2 Patients - Presented with recurrent Myocardial Infarction with moderate to severe left ventricular dysfunction and a significant rise in Troponin I
   - 1 Patient - severe pulmonary artery hypertension with dilated RA/RV
   - 2 patients - just presented with COVID-19 symptoms
2. 14 Patients - Presented with Acute Myocardial infarction;
   - 7 Patients - Had anterior wall Myocardial infarction
   - 3 Patients - With anterior wall MI had moderate to severe LV dysfunction and presented with cardiogenic shock
   - 5 Patients - Presented with inferior and posterior wall Myocardial infarction
   - 2 Patients - With inferior and posterior Myocardial infarction had a cardiogenic shock
   - 2 Patients - Presented with lateral wall Myocardial infarction
3. 3 Patients – Had NSTEMI with a significant increase in Troponin I levels
4. 2 Patients - Had old myocardial infarction had a permanent pacemaker
5. 1 Patient - Presented with LBBB and was on PPI
6. 2 Patients - Had bradycardia with a heart rate less than 60 beats per minute.
7. 1 Patient - Presented with ventricular bigeminy
8. 3 Patients - Had atrial fibrillation
9. 2 Patients - Who developed atrial fibrillation when on Non Invasive ventilation (NIV) support
10. 1 Patient - Presented with Atrial fibrillation where she was a known case of CRHD
11. 3 Patients – Had severe pulmonary artery hypertension with RA/RV dilated and a thin rim of pericardial effusion
12. 1 Patient – Had acute pulmonary thromboembolism
13. 1 Patient - Had chronic pulmonary thromboembolism.

Inflammatory markers like CRP, IL-6, D-Dimer, Ferritin, LDH were all significantly higher, as shown in the table1. 4 Patients were on NIV support and recovered, and 6 patients succumbed to death.

Table 1: Inflammatory Markers in Covid 19 Patients with Cardiovascular Disease

Inflammatory markers	Mean value of 40 patients	Normal reference range
CRP	46.75 mg/l	< 6 mg/l – NEGATIVE
IL-6	12.755 pg./ml	UPTO 4.4 pg./ml
D-DIMER	1575 ng/ml	80-650 ng/ml
FERRITIN	827 ng/ml	30-400 ng/ml (Males) 13-150 ng/ml (Females)
LDH	445 IU/L	98-192 IU/L

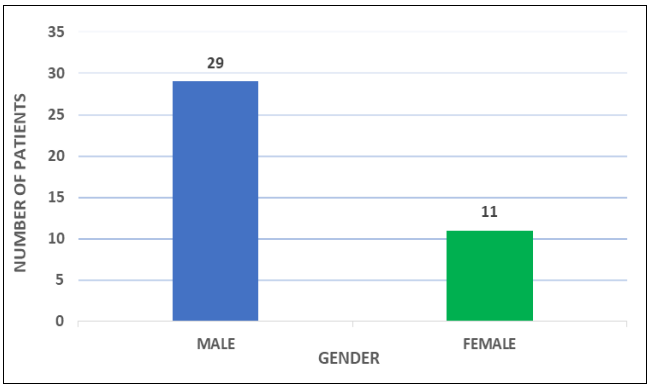

Fig 1: Gender Distribution in Patients with Covid 19 and Cardiovascular Disease

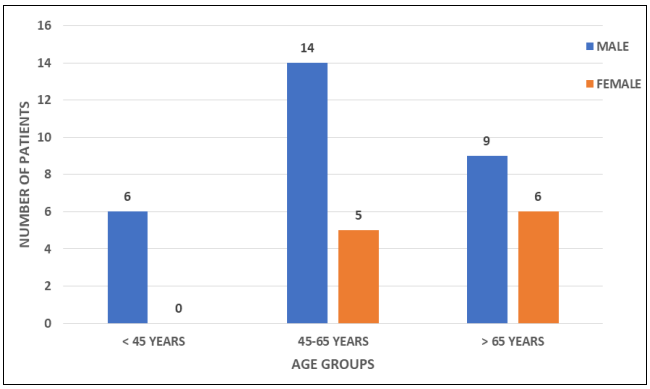

Fig: 2: Comparision Of Age Groups And Gender Distribution

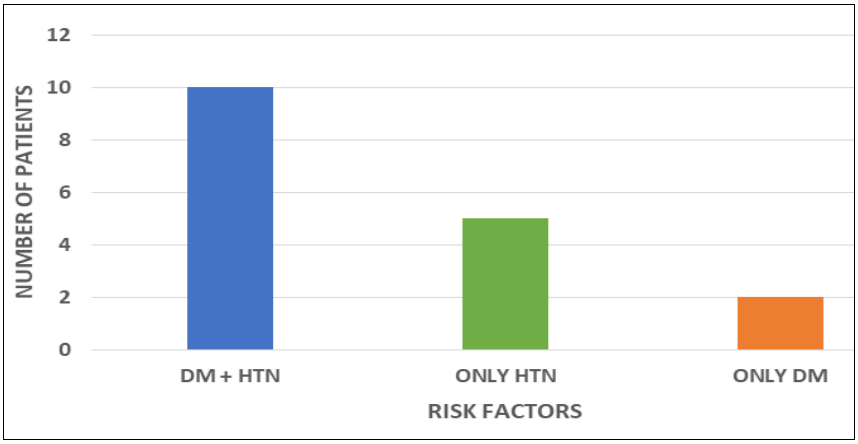

Fig 3: Risk Factors – Hypertension and Diabetes Mellitus

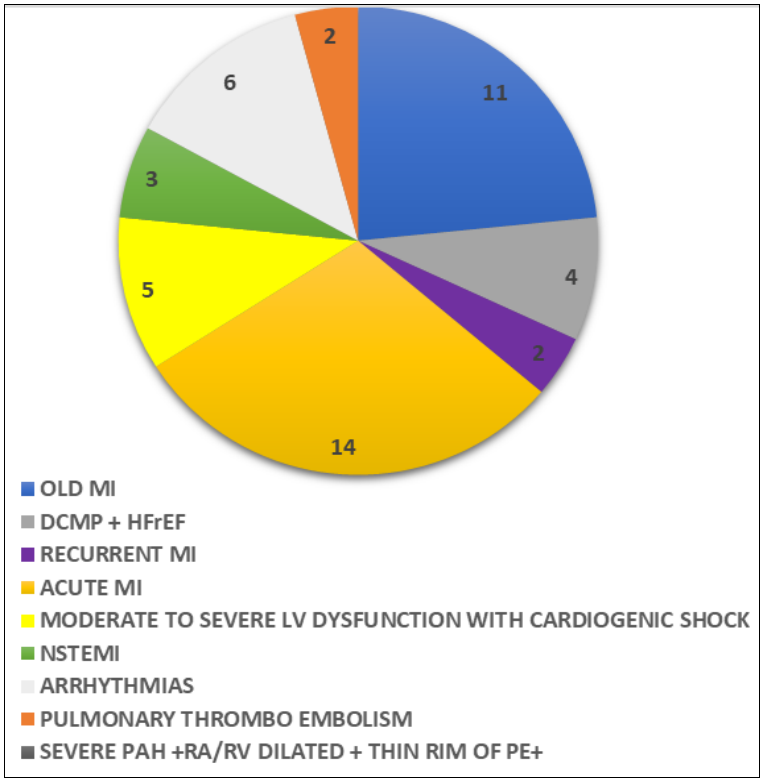

Fig 4: Cardivascular Manifestations in Covid 19 Patients

ECG Changes

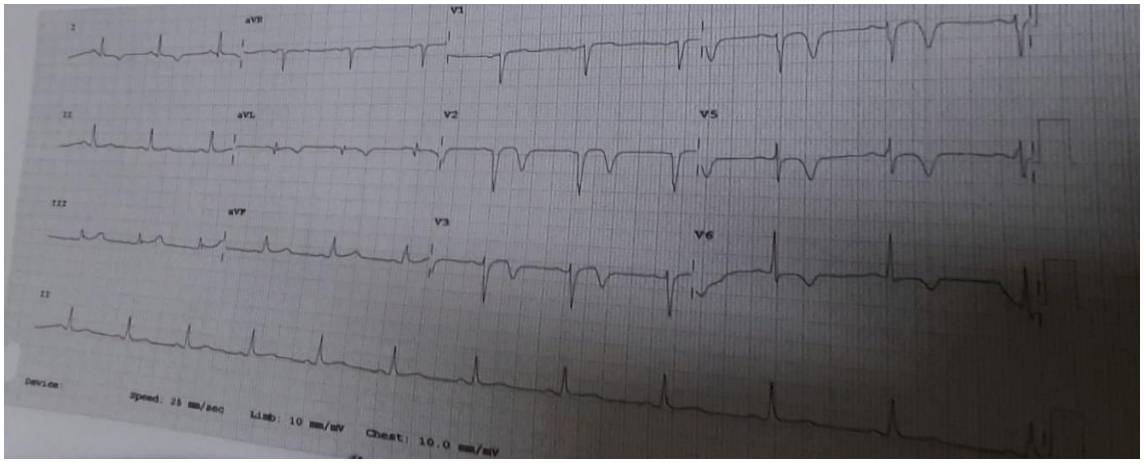

Fig 5: Anterior Myocardial Infarction

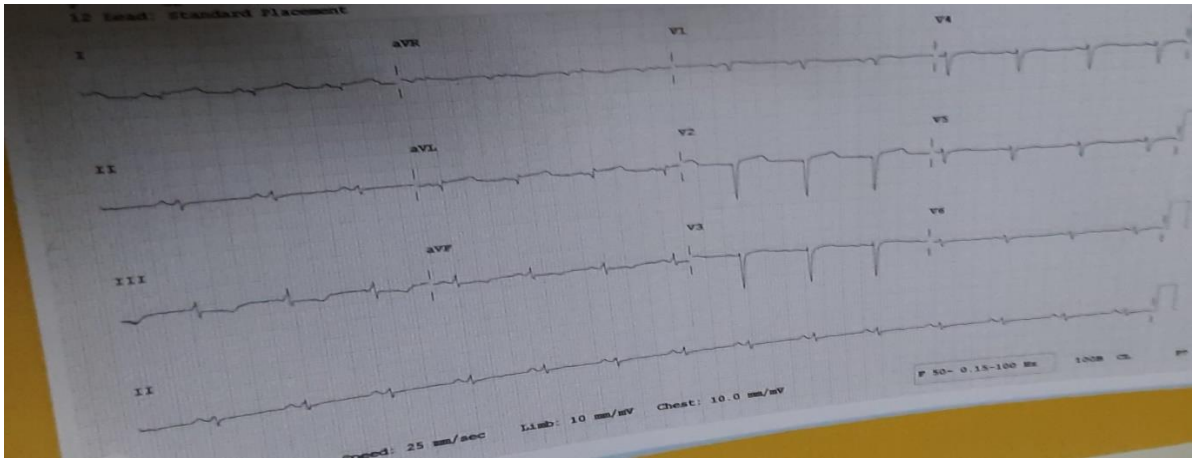

Fig 6: Anterior Wall Mi With Low Voltage Complexes

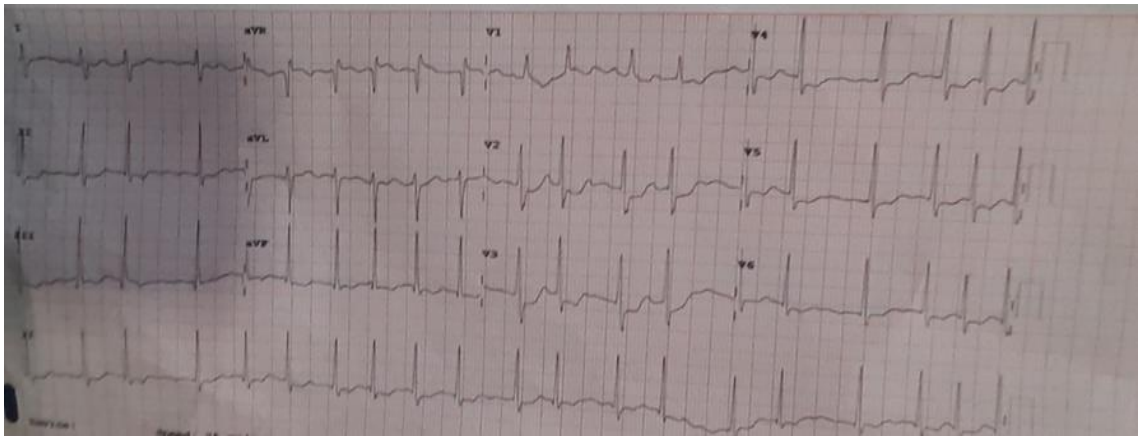

Fig 7: Atrial Fibrillation

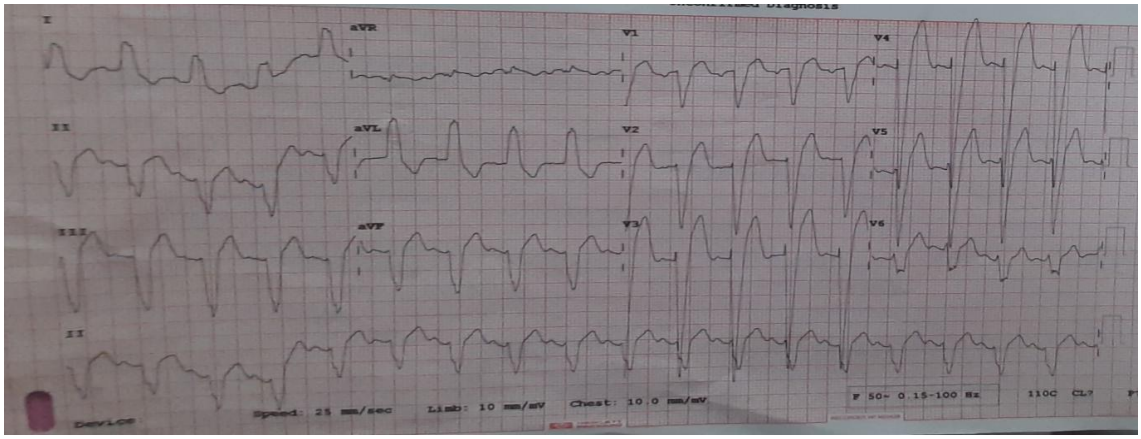

Fig 8: LBBB With Anterior Myocardial Infarction

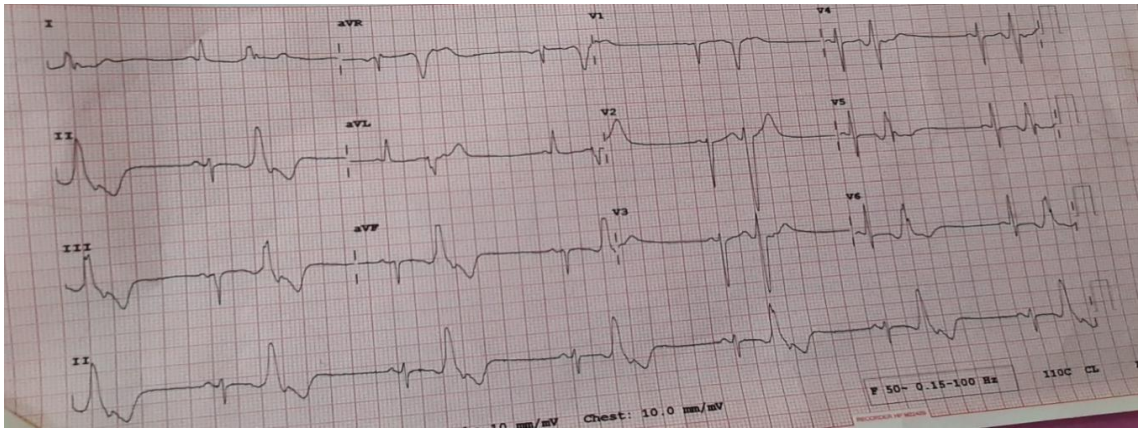

Fig 9: Ventricular Bigeminy

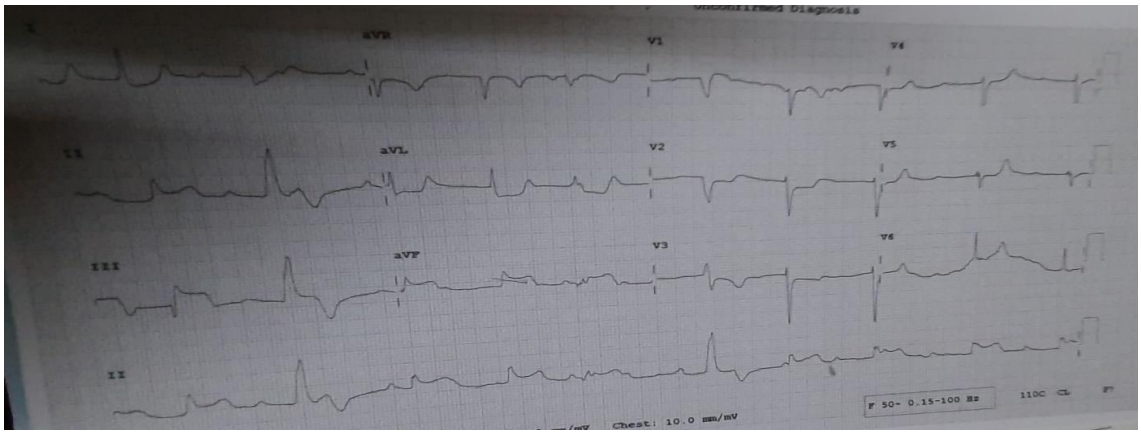

Fig 10: Old Anterior Wall MI with Acute Recurrent Inferior Wall MI

Discussion

Our observational study took up 200 COVID-19 positive cases admitted in either COVID-19 ward or COVID-19 ICU isolation. Among those 200 COVID 19 cases, 40 cases which had cardiovascular involvement diagnosed by history, ECG, 2D echo, Troponin I, CT pulmonary angiogram were taken for study. The mean age of presentation with Cardiovascular disease was 53 years, the youngest being 23 years, and the oldest 93 years, and the Male: Female ratio was 2.636. Elderly age group > 65 years were 14 patients (35%). Cardiac involvement in COVID-19 range from subclinical myocardial injury to well defined clinical entities.

The mechanism that coronavirus cause damage to the heart involves

1. Primary myocardial involvement – Coronavirus has a tropism for endothelium, and this can be linked by the virus binding to angiotensin-converting enzyme 2. Hence coronavirus damaging myocardium and causing myocarditis and ventricular dysfunction can be substantiated.
2. Secondary cardiac involvement – Hyper inflammatory response in the advanced stage of the disease elicits cytokine storm. These cytokines have been implicated in myocardial injury and adverse remodeling. IL 1 can cause atherothrombosis and hyperinflammatory milieu, which may provoke atherosclerotic plaque rupture, causing Type 1 myocardial infarction and pulmonary embolism. And COVID-19 itself is a state of a thromboembolic event. And as a consequence of hypoxia-induced myocardial damage leads to Type 2 myocardial infarction.

The patients presented with elevated Troponin I levels, ECG changes consistent with myocardial infarction or ischemia, and 2Decho showing wall motion abnormalities had a history of chest pain, fever for 3-5 days associated with progressive dyspnea and cough. Average Troponin I with patients having myocardial infarction or NSTEMI was >10. And 35.71% of patients with acute myocardial infarction had moderate to severe left ventricular dysfunction with cardiogenic shock. Patients who had a prior myocardial infarction and were managed by either stenting or CABG presented with recurrent myocardial infarction with significantly high values of Troponin I, and 36.36% of patients had ischemic dilated cardiomyopathy with acute decompensated heart failure. Patients who presented with COVID-19 pneumonia associated with cardiovascular abnormalities had relatively high inflammatory markers, as mentioned above in Table 1. This can explain myocardial injury because of associated inflammation and complications and the severity of COVID-19 in cardiovascular diseases. 2 Patients who had old myocardial infarction had a permanent pacemaker, and 1 patient presented with LBBB and was on PPI. 2 patients had bradycardia with a heart rate less than 60 beats per minute. 1 Patient presented with ventricular bigeminy, and 3 patients had atrial fibrillation with 2 patients who developed atrial fibrillation when on NIV support, and 1 patient presented with Atrial fibrillation where she was a known case of CRHD. Severe pulmonary artery hypertension and RA/RV dilated with a thin rim of pericardial effusion is one of the features that we found where 3 patients presented with this after an average of 10-12 days from the date of admission, which also suggested prolonged hospitalization and patient going for the requirement of NIV support or death. And 2 patients presented with pulmonary thromboembolism, and these patients had a high value of inflammatory markers and persistently increased in requirement of oxygen and persistent cough with hemoptysis. 4 patients who had severe cardiovascular disease with Coronary artery disease and severe LV dysfunction required NIV support or HFNC support with a prolonged hospital stay of more than 20 days and recovered. But 6 patients (15%) died. And 66.66% were older age group, 33.33% were females, and average days of hospitalization before death for 66.66% of patients were more than 30 days, and 2 patients who had acute Myocardial infarction and NSTEMI died within 2 days. 2 patients developed atrial fibrillation when they went into the stage for the requirement of NIV support, 1 patient had severe LV dysfunction, and 1 patient had severe PAH with RA/RV dilated.

Conclusion

Coronavirus disease 2019 by itself can cause myocardial injury as we saw patients presenting with a history of chest pain, fever for 2-3 days and on investigating showed myocardial involvement. Patients with a history of cardiac disease showed exacerbation of the disease and serious complications. As coronavirus disease, 2019 severity increases involving > 50% of lung results in severe pulmonary artery hypertension with RA/RV dilation, and the patient requires ICU care and invasive or noninvasive ventilatory support. Pulmonary embolism chances increase as patient complaints of persistent cough, hemoptysis, and elevated inflammatory markers, including D-Dimers. Arrhythmias are more commonly associated with the patient on either invasive or noninvasive ventilators, but COVID-19 causing myocarditis or exacerbation of prior cardiac disease can also result in arrhythmias. And cardiovascular involvement in COVID-19 results in prolonged hospitalization, ICU care, and worse outcomes, including an increase in the mortality rate.

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