CASE REPORT

Case Report: A case of Dilated Cardiomyopathy in COVID-19; A case report [version 1; peer review: awaiting peer review]

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Abstract

As of 2022, myocardial injury associated with COVID-19 has been one of the most discussed topics in literature. Though variety of cardiac manifestations have been reported and described in scientific literature, case of dilated cardiomyopathy (DCM) has not been well reported and described. We present a case of DCM post-COVID-19 without any co-morbidities who was admitted several times for cardiac symptoms post-COVID-19. As it was a new finding associated with COVID-19, it has been worth understanding the variations in which cardiac conditions manifest in COVID-19.

Keywords

Dilated cardiomyopathy; COVID-19

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Introduction
Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), manifests typically with respiratory symptoms but cardiac signs and symptoms have also been common in the process of disease and post-COVID-19. Although the cardiac cases in COVID-19 have been much less as compared to respiratory cases, a review found cardiac injury to be reported in 19.7% to 29.8% of cases.¹ The involvement of cardiac myocardium in COVID-19 has been due to various mechanisms and up to 20-30% in hospitalized patients as manifested by elevated troponin levels.²

Although lungs have been the primary target of SARS-CoV-2, cardiovascular involvement has been common in hospitalized patients ranging from mild cardiac injury to complications like acute myocardial infarction, myocarditis, arrhythmias, thrombotic complications and heart failure.³ Till date various cases of stress-induced (Takotsubo) cardiomyopathy have been described with a classic case described by a case report where a patient presented with fever for five days and progressive dyspnea on exertion.⁴

The cardiac-related conditions like myocarditis, heart failure, thrombotic complications, stress-induced cardiomyopathies associated with COVID-19 have been described in literature. But cases of dilated cardiomyopathy (DCM) in adult have not been described in literature, though a case of DCM has been reported in a child of one year old.⁵ We hereby present a case of dilated cardiomyopathy (DCM) in a 35 years male post-COVID-19 without any co-morbidities.

Case presentation
A 35-year-old Hindu male tested COVID-19 positive (RT-PCR positive) dated six months back presented to our center with cough and shortness of breath. The cough was often associated with hemoptysis and shortness of breath was mostly on lying flat and walking with New York Heart Association (NYHA) grade-II at first. It progressed to NYHA grade III gradually. It was associated with orthopnea and paroxysmal nocturnal dyspnea (PND). It was followed by recurrent hospital admission for the same complaints complemented with dizziness, loss of consciousness and chest pain. For two times he was admitted in high dependency unit and was even treated with nor-adrenaline and dopamine for severe hypotension (SBP/DBP: 80/60). There was not any significant medical, family and psycho-social history related to the case. There were also not relevant past interventions.

On general examination, he was conscious and oriented to time, place and person. There was bilateral pitting pedal edema but the jugular venous pressure (JVP) was not raised. As for vital parameters, his blood pressure was more of consistent with SBP/DBP of 90/60 mm of Hg and his saturation was maintained at 90% in room air.

On systemic examination, systolic murmur was heard at the apex beat area which was consistent with carotid pulse and was non-radiating in nature. Bilaterally, crepitations were heard in infra-scapular and infra-axillary regions.

He was diagnosed as having Covid-19 infection from the real time-polymerase chain reaction (RT-PCR) 6 months back. He was kept on isolation for 14 days and discharged after the RT-PCR test was negative. He started developing shortness of breath after a month of Covid-19 infection. This was followed by recurrent hospital admission and diagnosed was DCM post-Covid-19.

The baseline laboratory investigations are shown in Table 1.

The ultrasonography of abdomen and pelvis showed normal scan. The chest x-ray showed cardiomegaly. Similarly, pulmonary function test (PFT) was performed to exclude any respiratory pathology. PFT showed moderate restriction with insignificant post-bronchodilator responsiveness. The 12-lead electrocardiogram showed wide QRS complexes and right bundle branch block (RBBB) pattern. Then, echocardiography was performed to narrow down the diagnosis. The 2D echocardiography findings are given below:

- Left atrium (LA): 4.6 cm; Right atrium (RA): 5.9 cm; Left ventricle (LV): 6.7 cm; Right ventricle (RV): 5.9 cm [Dilated all chambers of heart]
- Global LV hypokinesia
- Severe LV systolic dysfunction [Ejection fraction (EF): 10-15%]
- Severe Mitral regurgitation (MR), mild Pulmonary regurgitation (PR)
Severe Tricuspid regurgitation (TR)

Severe Pulmonary artery hypertension (PAH) with elevated Pulmonary artery systolic pressure (ePASP) of 71 mmHg

RV systolic dysfunction [Tricuspid annular plane systolic excursion (TAPSE): 15 mm and RV systolic prime (RVS)'

LV diastolic dysfunction-grade III

The figure for 2D-echocardiography and 12-lead electrocardiogram are shown in Figures 1 and 2 respectively. The high-resolution computed tomography (HRCT) scan of chest showed cardiomegaly with enlarged pulmonary trunk and diffuse ground glass opacity in basal segment of lungs. He was diagnosed as dilated cardiomyopathy (DCM) secondary to covid-19 infection resulting in heart failure with reduced ejection fraction (HFrEF) of 10-15%. He was kept on following medications Table 2.

The patient was advised on fluid restriction of less than 1 liter/day and low salt diet. He was then discharged home with plan for follow up in cardiac out-patient department as needed [SOS (Si Opus Sit)].

The patient was stable on discharge. In previous follow-ups his status was assessed by clinical examination, laboratory investigations like N-terminal pro b-type natriuretic peptide (NT-pro-BNP), troponin I, electrocardiogram and 2D echocardiography. The patient was satisfied under medications and was taking regular medications as described above.

### Discussion

COVID-19 associated myocardial injury has well been described in various literatures throughout the world.\(^3\) The overall pooled prevalence of acute myocardial injury was found to be 29% in hospitalized COVI-19 patients by 19 studies.\(^5\) Various mechanisms have been proposed for myocardial injury. Some of the well described mechanisms are hyperinflammatory reaction, hyperactivation of renin angiotensin aldosterone system pathway and cytokine storm.\(^3\)

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**Table 1. Baseline laboratory investigations.**

<table>
<thead>
<tr>
<th>Laboratory tests</th>
<th>Result</th>
<th>Unit</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Leukocytes Count</td>
<td>10.6</td>
<td>10^3/μL</td>
<td>4-11</td>
</tr>
<tr>
<td>Neutrophil</td>
<td>49</td>
<td>%</td>
<td>40-80</td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>45</td>
<td>%</td>
<td>20-40</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>12.9</td>
<td>g/dl</td>
<td>13-17</td>
</tr>
<tr>
<td>Platelet Count</td>
<td>201</td>
<td>10^3/μL</td>
<td>150-450</td>
</tr>
<tr>
<td>Urea</td>
<td>39</td>
<td>mg/dl</td>
<td>17-43</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.9</td>
<td>mg/dl</td>
<td>0.7-1.3</td>
</tr>
<tr>
<td>Sodium</td>
<td>134</td>
<td>mEq/L</td>
<td>135-145</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.1</td>
<td>mEq/L</td>
<td>3.5-5.5</td>
</tr>
<tr>
<td>Bilirubin Total</td>
<td>0.8</td>
<td>mg/dl</td>
<td>0.1-1.2</td>
</tr>
<tr>
<td>Bilirubin Direct</td>
<td>0.3</td>
<td>mg/dl</td>
<td>0.0-0.2</td>
</tr>
<tr>
<td>Alkaline Phosphatase (ALP)</td>
<td>48</td>
<td>U/L</td>
<td>53-128</td>
</tr>
<tr>
<td>Alanine Transferase (ALT)</td>
<td>34</td>
<td>U/L</td>
<td>0-35</td>
</tr>
<tr>
<td>Aspartate Transferase (AST)</td>
<td>32.7</td>
<td>U/L</td>
<td>0-35</td>
</tr>
<tr>
<td>Random Blood Glucose</td>
<td>115</td>
<td>mg/dl</td>
<td>70-140</td>
</tr>
<tr>
<td>Prothrombin time (PT)</td>
<td>15.2</td>
<td>seconds</td>
<td>11-13.5</td>
</tr>
<tr>
<td>CPK NAC</td>
<td>68</td>
<td>U/L</td>
<td>20-200</td>
</tr>
<tr>
<td>CPK MB</td>
<td>17.7</td>
<td>U/L</td>
<td>&lt; 35</td>
</tr>
<tr>
<td>Troponin I</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CPK NAC: Creatine Phospho-Kinase N-Acetyl Cysteine; CPK MB: Creatine Phospho Kinase-MB.
Figure 1. 2D echocardiography. Legend: Arrows showing dilated ventricles and atrium.

Figure 2. 12 lead electrocardiogram (ECG). Legend: White arrow showing wide QRS complex and Black arrows showing right bundle branch block (RBBB) pattern in leads V1 and V6.

Table 2. Medications.

<table>
<thead>
<tr>
<th>Dosage form</th>
<th>Drug</th>
<th>Dose</th>
<th>Route</th>
<th>Times a day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tablet</td>
<td>Digoxin</td>
<td>0.25 mg</td>
<td>PO (Per oral)</td>
<td>OD (Once a day)</td>
</tr>
<tr>
<td>Tablet</td>
<td>Metoprolol</td>
<td>12.5 mg</td>
<td>PO</td>
<td>OD</td>
</tr>
<tr>
<td>Tablet</td>
<td>Ramipril</td>
<td>1.25 mg</td>
<td>PO</td>
<td>OD</td>
</tr>
<tr>
<td>Tablet</td>
<td>Frusemide + Spironolactone</td>
<td>20 mg + 50 mg (1 Tab)</td>
<td>PO</td>
<td>OD</td>
</tr>
<tr>
<td>Tablet</td>
<td>Thiamine</td>
<td>100 mg</td>
<td>PO</td>
<td>OD</td>
</tr>
<tr>
<td>Tablet</td>
<td>Torsemide</td>
<td>10 mg</td>
<td>PO</td>
<td>OD</td>
</tr>
</tbody>
</table>
Similarly, abnormal endothelial function and downregulation of angiotensin-converting enzyme-2 (ACE-2) receptor in cardiac myocytes by SARS-CoV-2 have also predominant role in myocardial injury.6

A systematic review found SARS-CoV-2 to cause irreversible changes in heart with right heart dilatation due to pulmonary overload as its main features. Similarly, autopsy microscopic findings suggested necrosis, micro thrombosis and lymphocytic infiltration in myocardium.7 A Systematic review and meta-analysis by Corica et al found prevalence of right ventricular dysfunction (RVD) in COVID-19 patients was almost 1 out of 5 patients.8 As described by Corica et al, the mortality with COVID-19 and RVD was in excess to that with COVID-19 only. In regards to cardiac biomarkers for myocardial injury in COVID-19, a systematic review and metaanalysis by Zhu et al found strong association of creatine kinase (CK), CK-MB, lactate dehydrogenase (LDH), troponin I, NT-pro BNP (brain natriuretic peptide) with severity of COVID-19.9

COVID-19 associated Takotsubo cardiomyopathy has been reported in literature. A classic case of Takotsubo cardiomyopathy in COVID19 was reported by Gomez et al where a 57 year old presented with 5 days of fever (maximum 39.7° Celsius) and progressive dyspnea on exertion and developed rapid deterioration of cardiorespiratory status during hospitalization.4 Similar case of stress-induced (TCM) cardiomyopathy was also reported by Tsao et al in a 59 year old woman who also fulfilled the Mayo clinic criteria for TCM cardiomyopathy.4,10 Our case did not fulfil the criteria as both the ventricles were involved in both systole and diastole.

Although cases of dilated cardiomyopathy (DCM) in adult have not been reported till date, a case DCM has been reported in one year old healthy boy without any co-morbidities.5 Among various causes of DCM, viral infection is one of the secondary causes.11 The viral genomes have been detected in myocardial samples of patients with DCM in spite of undetectable infiltrating inflammatory cells.12 Due to the activation of persistent immune mechanism after viral infection, it has been presumed to lead to DCM.13

In regards to our case, he progressed gradually with cardiac symptoms after COVID-19 infection before he was diagnosed as DCM post-COVID-19. Ultimately, he developed heart failure with reduced ejection fraction of 10-15%. He has been repeatedly visiting our center whenever the cardiac symptoms worsen since the diagnosis was made.

Conclusions
COVID-19 has been a burden due to its global impact on livelihood. Apart from respiratory symptoms and complications, myocardial injury and its consequences should also be equally sought out and thoroughly evaluated in COVID-19 patients. It is necessary to keep in mind that COVID-19 can present with wide variances of cardiac symptoms and complications as described above. Among them DCM also stands as a consequence in COVID-19 as evidenced in this case. Therefore, a thorough cardiac evaluation which include cardiac biomarkers, ultrasonogram of abdomen and pelvis, echocardiography and electrocardiogram should be performed in COVID-19 patients. It helps us to guide our management strategies and prevent further cardiac complications in COVID-19 patients.

The patient was treated on hospital basis with both pharmacological and dietary interventions. According to the patient, he felt relieved after the therapeutic interventions. He was performing daily activities without any hindrance. Similarly, with the intervention his sleep activity was sound and good. He was feeling more comfortable with the treatment and was convinced for the regular follow up.

Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient/parent/guardian/relative of the patient.

Data availability
All data underlying the results are available as part of the article and no additional source data are required.

Consent
Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient/parent/guardian/relative of the patient.
References


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