http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v9i2.28

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

An Unusual Presentation and Sequel of COVID-19

Authors
Dr Aayushi Gupta, Dr Bhagwan Mantri, Dr Pawan Mangla, Dr BA Shastry
Moolchand Hospital, New Delhi

Introduction

The outbreak of COVID-19, caused by the novel coronavirus SARS-CoV-2, has affected a huge number of patients worldwide. The clinical presentation spectrum is wide, from asymptomatic patients to critically ill cases. Most pulmonary infections are mild, but severe and critical cases have been described, especially in the elderly, developing with dyspnea, hypoxia, major lung involvement in imaging, respiratory failure, shock and multiple organ failure.[1] However, extra pulmonary manifestations of the disease are also increasingly being reported.[2-6]

Extrapulmonary features are mainly due to the cytokine storm, whereby pro- inflammatory cytokines and chemokines such as tumour necrosis factor- α, IL-1β and IL-6 are overproduced by the immune system, resulting in multiorgan damage.

Cases of heart involvement by the coronavirus 2019 disease (COVID-19), developing with acute myocarditis have also been described, mainly in severe cases.[7,8] Chest CT, however, is limited in terms of heart assessment.[9] Thus, these patients with clinically suspected COVID-19 myocarditis have been assessed by other imaging methods, such as echocardiography and cardiac magnetic resonance imaging (CMR).[10]

We describe a case of patient of myocarditis with heart failure due to COVID 19.

Case Report

28 year old female came to the hospital with complaints of multiple episodes of loose stool and vomiting since 1 day. Patient had 10-15 episodes of watery loose stools and 4-5 episodes of vomiting. She also gave history of fever since 1 day, maximum Up to 100'F associated with chills and rigours. No previous history of any co morbidities or any cardiac or respiratory illness. She was referred from an outside facility where she had constant hypotension despite fluids and inotropic support.

On arrival, in emergency department, she was afebrile, her pulse was 125/min, tachypnea present (RR-30), BP -90/60 mm Hg on vasopressor support, SPO2 – 94% on 2L O2.

She was conscious, oriented. Pallor was present. No cyanosis, clubbing or oedema was present. On Auscultation bilateral mild crepitations were present.

She was primarily managed in emergency department and then shifted to ICU.

On investigation

Her Haemoglobin was 7.3 mg/dl, TLC -6200, Platelet count - 1.1 lakh. Peripheral smear was

done which showed microcytic hypochromic anemia with neutrophilic leukocytosis and thrombocytopenia. AST, ALT and ALP were normal. KFT was normal.

Her ABG on Day 1 showed metabolic and respiratory alkalosis with hypoxia (PaO2- 59).

COVID 19 RTPCR done was negative.

D-dimer was > 5000. NT pro BNP was 9900. Cardiac enzymes were normal. Her CRP was positive (48), LDH was 350.

X-RAY Chest was done which showed bilateral haziness in right lower zone with angle blunting of right > left side. (Figure 1)

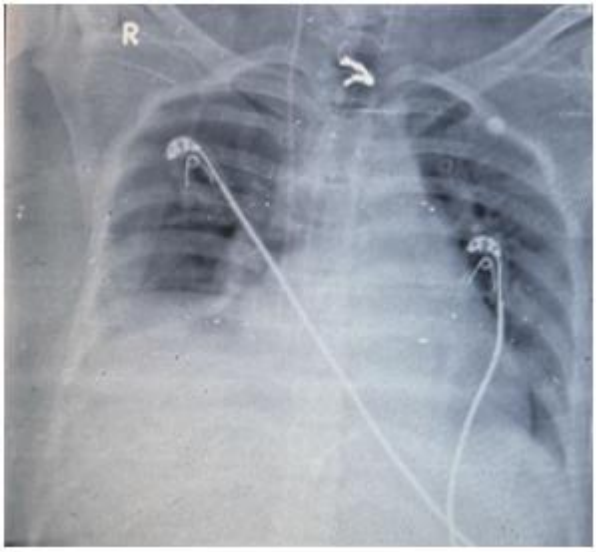
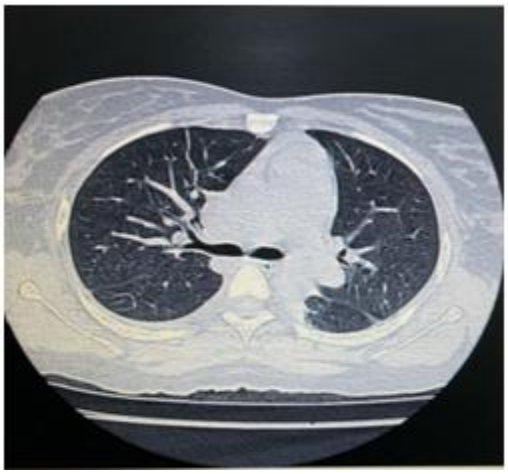


Fig 1

NCCT chest was done following this, which showed Air space opacity in the bilateral lower lobes with bilateral minimal pleural effusion with? Pulmonary Edema.



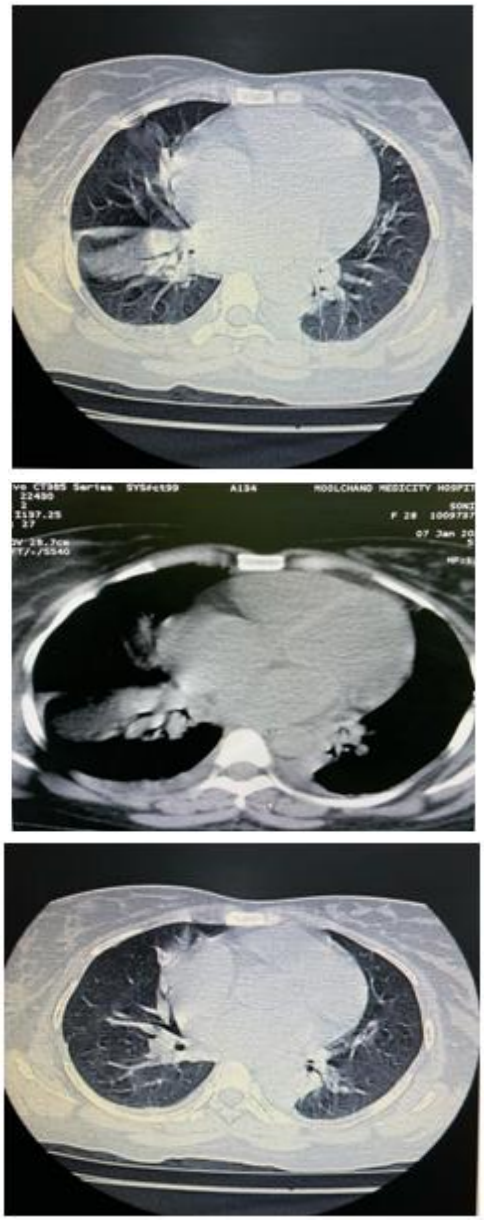


Figure 2

ECG done showed prolonged QT interval. (Figure 3)

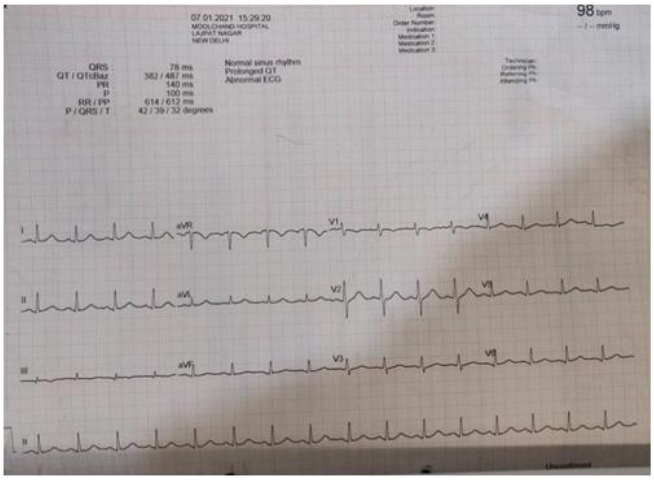


Figure 3

Bedside 2D Echo was done in ICU which showed Global LV Hypokinesia, with LVEF of 35-40%. RV dysfunction with dilated all cardiac chambers. Grade 2 LVDD. PASP 46 mmHg.

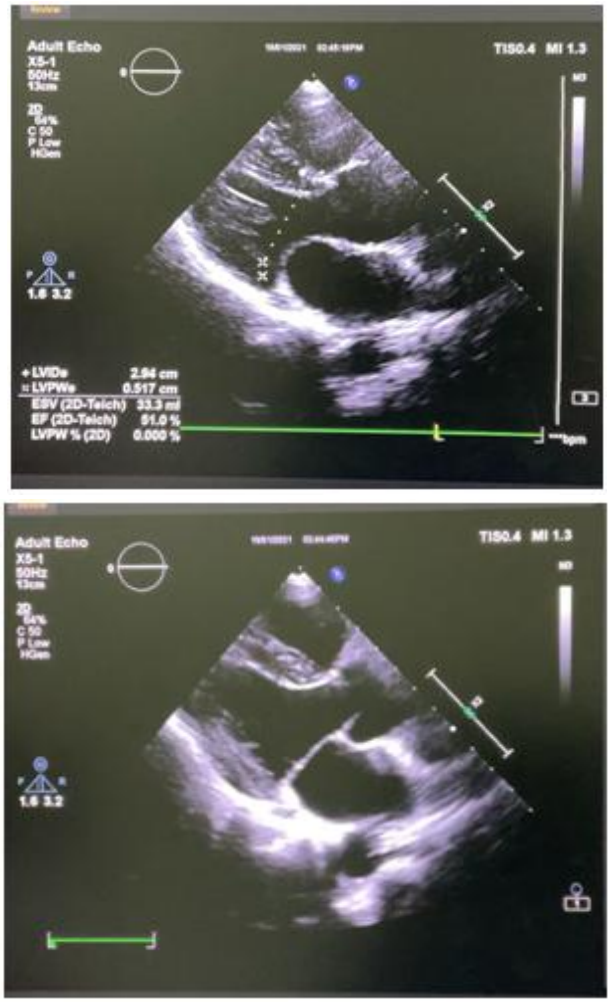


Figure 4

In view of myocarditis most probably due to viral etiology, dengue Ig G, IgM along with other viral serologywas sent which was non reactive. In light of ongoing pandemic, ANTI SARS COV-2 antibody was done which was found to be reactive (22.2). Also in view of COVID induced myocarditis inflammatory markers were sent which were elevated (CRP- 48, LDH- 350). So diagnosis of COVID 19 induced viral myocarditisand heart failure was made.

During the first days of her hospitalisation, the patient remained hypotensive (systolic blood pressure less than 90 mm Hg) and required inotropic support in the first 48 hours. Blood pressure progressively stabilised, although systolic pressure remained less than 100 mm Hg,

Due to decreased Haemoglobin, 1 unit blood transfusion was done. Iron profile was done showed iron deficiency anaemia for which iron correction was given and deworming was done. Cardiac team of the hospital was involved and heart failure directed treatment was started and she was also started on treatment as per COVID protocol.

She improved dramatically, was weaned off from oxygen support, and discharged from hospital after a stay of 12 days. Patient was followed up after a period of 7 days. She is doing well. Repeat echo was done which showed improvement in diastolic function, and improvement in LVEF was 50%.

Limitations

Due to financial constraints, cardiac MRI couldn't be performed. Also As endomyocardial biopsy was not performed, limitations of this report are the lack of the histological demonstration of myocarditis and the absence of viral genome search in the heart. She had no previous history of any cardiac illness and at such young age heart failure couldn't be explained with any other etiology. Except for the first 48 hours during which she required inotropic support, the patient was mainly treated with heart failure–directed medical treatment. However, as described in the literature, viral myocarditis has a wide spectrum of clinical presentations, ranging from life-threatening arrhythmias to advanced heart failure requiring invasive support.

Discussion

COVID-19 cases with cardiac involvement, developing acute myocarditis have been described.[7,8] Heart failure has been appointed as one of the sources of secondary complications in these patients.[8]

There are 2 proposed mechanisms for cardiac involvement: first which involves direct viral infection to myocardium and second is indirect toxicity caused by the systemic infection, which can trigger vasculitis or hypersensitivity reaction.

Viral infections have been widely described as one of the most common infectious causes of myocarditis, especially associated with influenza and parvovirus B-19 infection. However, less is known about the cardiac involvement as a complication of SARS-CoV-2 infection.

Myocarditis results in focal or global myocardial inflammation, necrosis, and eventually ventricular dysfunction. Focal myocarditis is often suspected in patients presenting with chest pain after an influenza like syndrome, with clinical evidence suggesting an acute coronary syndrome on electrocardiography or laboratory testing or with evidence of wall motion abnormalities without evidence of obstructive coronary artery disease on coronary angiography.

In the scenario of the SARS-CoV-2 pandemic, it is important to consider the hypothesis of cardiac involvement, mainly in patients with abrupt deterioration of symptoms despite respiratory support measures, those with unexplained increase in myocardial necrosis markers and in patients with a new dysfunction documented by echocardiography. In face of such a possibility, CMR can be used to search for signs compatible with myocarditis, such as the presence of non-ischemic late enhancement pattern.

References

1. Wu Z, McGoogan JM. Characteristics of and important lessons from the coronavirus disease 2019 (COVID-19) outbreak in China: summary of a report of 72 314 cases from the Chinese Center for Disease Control and Prevention. JAMA. 2020;323(13):1239-42.
2. Kim IC , Kim JY , Kim HA , Han S. COVID-19-related myocarditis in a 21-year-old female patient. Eur Heart J 2020;41:1859.
3. Li YC, Bai WZ, Hashikawa T. The neuroinvasive potential of SARS-CoV2 may be at least partially responsible for the respiratory failure of COVID-19 patients. J Med Virol 2020;92:552–555.
4. Gu J, Han B , Wang J. COVID-19: gastrointestinal manifestations and potential fecal–oral transmission. Gastroenterology 2020;158:1518–1519.
5. Madjid M , Safavi-Naeini P , Solomon SD, Vardeny O. Potential effects of coronaviruses on the cardiovascular system: a review. JAMA Cardiol 2020;doi: 10.1001/jamacardio.2020.1286.
6. Inciardi RM , Lupi L , Zaccone G , Italia L, Raffo M , Tomasoni D, Cani DS, Cerini M , Farina D , Gavazzi E , Maroldi R, Adamo M , Ammirati E , Sinagra G, Lombardi CM, Metra M. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). JAMA Cardiol 2020;5:819–824.
7. Hu H, Ma F, Wei X, Fang Y. Coronavirus fulminant myocarditis saved with glucocorticoid and human immunoglobulin. Eur Heart J. 2020 Mar 16:ehaa190.
8. Rente A, Uezato Junior D, Uezato KM. Coronavirus and the heart | A case report on the evolution of COVID-19 Associated with Cardiological Evolution. Arq Bras Cardiol. 2020;114(5):839-42.
9. De Farias LP, Strabelli DG, Fonseca EK, Loureiro BM, Nomura CH, Sawamura MV. Thoracic tomographic manifestations in symptomatic respiratory patients with COVID-19. Radiol Bras. 2020;53(4):255-61.
10. Fonseca EK, Chate RC, Sasdelli Neto R, Ishikawa WY, Silva MM, Yokoo P, et al. Findings on COVID-19 on magnetic resonance imaging. Radiology: Cardiothoracic Imaging. 2020;2(2). https://doi.org/10.1148/ryct.2020200193.