Myocarditis as an Initial Presentation of COVID-19 Infection in a 70 Years old Bangladeshi Lady: A Case Report and Insights

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Abstract

Viral genesis is the most common cause of myocarditis. COVID-19-associated myocarditis seems to be a notable extra pulmonary manifestation, which may result in the need for a different treatment. There has been no positive Polymerase Chain Reaction (PCR) testing of COVID-19 in heart specimens, thus far. Myocarditis is well known to be caused by viral infections such as Coxsackie virus group B, human herpes virus 6 and parvovirus B19. However, during the current emerging outbreak of COVID-19, there have been few case reports describing myocarditis as a possible presentation. In our case report we describe, cardiac manifestations of COVID-19 in a medical college hospital in Dhaka, Bangladesh. A 70 year old woman without any comorbidities presented with symptoms of COVID-19 later developed fulminant myocarditis. Patient underwent treatment with ionotrope and methylprednisolone that showed reduction in myocardial inflammation and significantly improved myocardial contractility. Although, not described in the literature, we have found conjunctive use of ionotrope and methylprednisolone is effective in patient with COVID-19 fulminant myocarditis.

Keywords: Myocarditis; COVID-19; Ionotrope; Methylprednisolone

Introduction

Since March 2020, we have been facing a pandemic due to the novel corona virus (severe acute respiratory syndrome corona virus 2 - SARS-CoV-2), who's initial cases emerged in the city of Wuhan, in the province of Hubei, China [1-4]. SARS-CoV-2 is a single-stranded RNA virus with fast mutation and recombination, and high similarity to other corona viruses which have appeared in previous years (SARS and MERS) [5]. 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 dyspnoea, hypoxia, major lung involvement in imaging, respiratory failure, shock and multiple organ failure [6]. Chest Computed Tomography (CT) can help to diagnose the disease, mainly in the current pandemic scenario, in which Real-Time Polymerase Chain Reaction (RT-PCR) results from nasal and oropharyngeal swabs can take a few days, although its use as a screening method is not recommended. The most frequently observed CT findings in cases of disease caused by SARS-CoV-2 are ground glass opacities and consolidations in lungs, with a predominantly peripheral distribution, sometimes associated with fine reticulate (forming the so called crazy

paving pattern), vascular thickening and inverted halo signal. Central parenchyma involvement, nodules, cavities, pleural effusion or lymph node enlargement are not frequently observed [7,8]. However, extra pulmonary manifestations of the disease are also increasingly being reported [9-13].

Fulminant myocarditis is a catastrophic illness, with significant myocardial inflammatory compromise, which can lead to death. Its multiple an etiologies include autoimmunity and infections. A viral origin is one of the most common, with multiple microorganisms involved [14]. Cases of heart involvement by the corona virus 2019 disease (COVID-19), developing with acute myocarditis has also been described, mainly in severe cases [2,15]. Chest CT, however, is limited in terms of heart assessment [16]. Thus, these patients with clinically suspected COVID-19 myocarditis have been assessed by other imaging methods, such as echocardiography and Cardiac Magnet Resonance Imaging (CMR) [10]. We present a case of probable fulminant myocarditis secondary to COVID-19.

Case Report

An 70 years old Bangladeshi lady, not known to have hypertension, diabetes mellitus obstructive airway disease or any cardiac disease came to the emergency room with the complaints of fever (102.6°C), worsening dry cough, dyspnoea and a 90% oxygen saturation at home for 3 days. But she denied any hemoptysis, palpitation, chest pain. On examination she was febrile, dypnoeic with respiratory rate 26 breaths per minute. Real-time polymerase chain reaction SARS-CoV-2 identification was positive on a nasal swab sample. Sputum culture did not reveal signs of co-infection. Given the clinical presentation and risk factors for progressing to a severe case, such as age, management chosen was admission and performing chest CT. The CT study revealed (Figure 1) small round ground-glass opacities, with multifocal distribution on both lungs, which corroborated the possibility of COVID-19 among differential diagnoses.

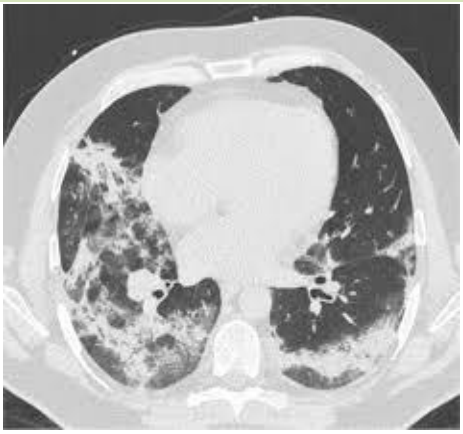
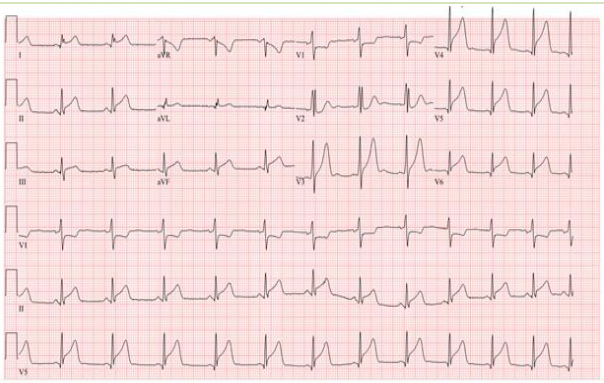
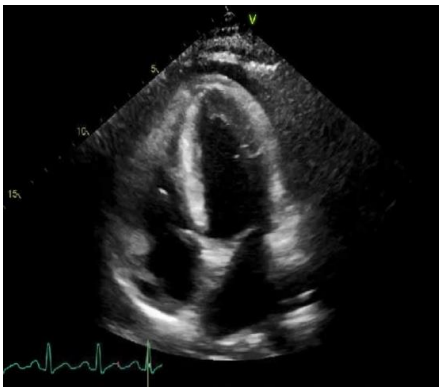


Figure 1: CT chest revealed round ground-glass opacities, with multifocal distribution on both lungs.

Admission lab tests revealed normal complete blood count, CRP, and procalcitonin. Arterial blood gas revealed hypoxia (PaO_2 52 mm of Hg on room air). She was transferred to ICU and started treatment with oxygen and lopinavir/ritonavir based therapy [2]. Days after admission her shortness of breath worsened and she also developed chest tightness and palpitation. Repeat clinical examination revealed pulse 122 beats/min, regular, BP 70/30 mm of Hg, respiratory rate 26 breaths/min. Cytokine storm was suspected and some other investigations were advised. Serum ferittin was 1551 mcg/litre (normal 11 mcg/litre to 307 mcg/litre), LDH was 880 U/L (normal 140-280 U/L). ANA and ANCA's were negative. Troponin I was significantly raised (63pg/mL; normal if <5pg/mL). An electrocardiogram was then performed (Figure 2A) which revealed tachycardia and ST elevation with concavity upwards consistent with myopericarditis but did not show signs of ischemia, and the transthoracic echocardiogram (Figure 2B) revealed a left ventricle with global hypokinesia, with severely reduced systolic function, an ejection fraction of 30%, without valvular heart disease, and mild pericardial effusion (2 mm).



(A)



(B)

Figure 2: (A) ECG showing ST elevation with concavity upwards and transthoracic echocardiogram, (B) left ventricle with global hypokinesia respectively.

Finally, it was concluded that the patient should be diagnosed with fulminant myocarditis with stage B cardiogenic shock and COVID-19 pneumonia. Treatment in the Intensive Care Unit (ICU) consisted of oxygen therapy without mechanical ventilation, methylprednisolone 500 mg/day for 3 days, subcutaneous enoxaparin, intravenous meropenem and linezolid for 10 days. Tablet l Lopinavir/ritonavir (800/200 mg/day was continued. The initial support was performed with norepinephrine and subsequently inotropic support with dobutamine in addition to intravenous frusemide, without the need for circulatory assistance devices. The patient showed a sustained clinical, hemodynamic, and respiratory improvement. After 18 days of hospital stay (10 days in the ICU), the patient was discharged with heart failure management which included bisoprolol 5 mg/day, spironolactone 25 mg/day, and telmisartan 40 mg od and rivaroxaban 10 mg once daily. Before discharge, cardiac biomarkers and echocardiography were repeated which revealed decreased troponin I and improving ejection fraction to 40%.

Discussion

The worldwide COVID-19 pandemic is both a challenge for healthcare, as well as unknown territory with respect to several aspects of SARS-CoV-2 triggered pathophysiology. Apart from the dominant pulmonary symptoms, much less is known about extra pulmonary symptoms and manifestations. In addition to reports on neuronal invasion [10], gastrointestinal manifestation, [11] and alteration of the lymphocyte profile, myocardial injury and other cardiac manifestations is still an emerging field of knowledge [12,13]. With respect to the systemic aspects of a SARS-CoV-2 infection, other systemic inflammatory diseases need to be considered at the time of diagnosis. Among the rheumatic diseases, small-vessel vasculitides especially eosinophilic granulomatosis with polyangitis in particular present with symptoms similar to COVID-19.

COVID-19 cases with cardiac involvement, developing acute myocarditis has been described [2,15]. Heart failure has been appointed as one of the sources of secondary complications in these patients [15]. The analysis of 44,672 confirmed cases of COVID-19 in Wuhan pointed out cardiovascular complications, such as myocarditis (10% of cases), myocardial injury (20%), arrhythmias, (16%) and heart failure and shock (5%) [17-20]. While uncommon, fulminant myocarditis is a malignant condition requiring rapid recognition and treatment due to high morbidity and mortality. Our patient had a clinical picture consistent with acute myocarditis with rapid deterioration leading to cardiogenic shock and need for inotropic support.

In our case, we observe an increase in the markers of myocardial injury with severe systolic dysfunction in the echocardiograph; this implies that the possible myocardial injury mechanisms due to COVID-19 could be, in the first place, by direct injury to the myocytes as a result of the migration of infected macrophages from the lung, as observed in the infiltration of myocardial macrophages in the autopsy samples of patients with SARS [21]. A second probable mechanism is the indirect injury of the myocardium due to the cytokine storm that the infection triggers, demonstrated by the increase of Interleukin (IL) IL-6, IL-10, IL-2 receptor (IL-2R), and Tumor Necrosis Factor (TNF)-a in severe cases [22,23]. A third probable mechanism is the presence of a significant amount of Angiotensin-Converting Enzyme 2 (ACE2) receptors in the myocardium with down regulation which alters the cardio protective effects of angiotensin 1-7, with an increase of TNF-a production associated with the myocardial dysfunction [24]. These mechanisms suggest that the severe inflammatory response is a probable mediator of myocardial compromise in SARS-COV2 cases, although there is a lack of studies that confirm this theory.

There are few fulminant myocarditis cases described in the SARS CoV-2 pandemic. The inflammatory process in this case takes place in the lower respiratory tract, as observed in the chest CT at the time of diagnosis, which could explain a cardiac injury triggered by the inflammatory response. Currently, fulminant myocarditis is not known as a secondary common complication of COVID-19, and therefore a high clinical suspicion is required for an early diagnosis.

Inciardi RM, et al. [13] reported a patient with COVID-19 and myocarditis diagnosed by Cardiac Magnetic Resonace (CMR), who presented with increased troponin, changes in segmental contractions and left ventricular dysfunction on the echocardiogram. The patient was treated with inotropic support, having improved clinically as of the first week after initiation of treatment. Another more severe case was reported by Hu H, et al. [2] who described a patient with the diagnosis of a fulminant myocarditis, along with diffuse myocardial edema and major ventricular dysfunction. The patient received hemodynamic support, steroids and human immunoglobulin, having completely recovered ventricular function and attained normal myocardial lesion markers after 3 weeks. In our case, after treatment, the patient presented clinical and hemodynamic improvement, achieving recovery of systolic function and decreasing pulmonary compromise. The decision to use dual inotropic therapy was substantiated by the persistence of the ScvO2 at <60% despite the use of dobutamine and norepinephrine, with subsequent clinical improvement, without circulatory support. Perhaps the cytokine release syndrome, a hypothesis related to the severity of inflammation in the infection by COVID-19 that generates cardiac involvement, encourages us to use systemic steroids to counteract it. Currently, there are no clinical trials to support this therapeutic approach; the decision was based on the clinical improvement described in case reports and the decrease of mortality observed in a comprehensive support treatment for patients with fulminant myocarditis [2,13,25]. We also administered lopinavir/ritonavir based on their use leading to a shorter stay in the ICU as a secondary outcome.

Like the cases described [2,13] our current case occurred at an older age, there was no history of cardiovascular disease. CMR and endomyocardial biopsy was considered for histological confirmation, but it could not be done due to unavailability. Regarding the background, in a study of 138 patients with COVID-19, [18] it was noted that 11.8% of the patients who died had substantial heart damage without an underlying cardiovascular disease, which implies that the incidence of cardiovascular complications is relevant. Also, in our case, clinical improvement was observed with the use of steroids, in addition to the treatments described so far for COVID-19. It seems that the use of inotropics plus an intravenous steroid helps to improve the hemodynamic

condition of the patient. It also decreases the need for left ventricular assist devices in centers where there is no availability. At present, the evidence is not sufficient and requires further study.

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. Moreover, in suspected arrhythmia and/or myocarditis, lung fields should be carefully assessed, even by CMR, given respiratory asymptomatic or oligosymptomatic individuals can be infected by the new corona virus.

Conclusion

The infection by SARS-COV-2 can affect multiple systems, with mainly lung involvement. However, myocardial injury may be frequently evidenced in patients with COVID-19 with variable clinical manifestation such as acute fulminant myocarditis that requires clinical suspicion for early diagnosis and treatment. Frequent monitoring and follow-up of acute heart failure are required. Complementary tests such as echocardiogram and cardiac magnetic resonance imaging can help diagnostic investigation. Control of progression is indispensable, given there is still no evidence in the literature on the late development of myocardial dysfunction in these patients. The importance of this case lies in the use of steroids and ionotrope as part of the successful therapy, which to date requires further study.

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