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Case Report

Acute Myocarditis Following COVID-19 Infection- A Case Report

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Abstract: COVID-19 has been declared a global pandemic by the World Health Organization and is responsible for hundreds of thousands of deaths worldwide. COVID-19 is caused by SARS-CoV-2, and common clinical symptoms include fever, cough, sore throat, headache, and fatigue. The cardiovascular system is affected in this infection, with complications including myocardial injury, myocarditis, acute myocardial infarction, heart failure, dysrhythmias, and venous thromboembolic events. Current therapies for COVID-19 may interact with cardiovascular medications. Myocardial injury is relatively common in patients with COVID-19, accounting for 7%-23% of cases, and is associated with a higher rate of morbidity and mortality [1]. In patients with COVID infection clinical suspicion is necessary to diagnose myocarditis as an important complication of this infection. Here we present a patient who developed shortness of breath and orthopnea following sore throat and myalgia and was eventually treated with a diagnosis of myocarditis and discharged from the hospital in relatively good general condition.

Keywords: Myocarditis, COVID-19, immune response.

INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a new variant form of coronavirus that is responsible for the coronavirus disease 2019 (COVID-19). Although the virus mainly infects the lung epithelial cells causing respiratory signs and symptoms, there has been an upsurge of cases that presented COVID-19-induced acute myocarditis [2]. It has been suggested that direct viral contact through angiotensin-converting enzyme 2 (ACE-2) signaling pathways might have a role in the myocardial injury. In addition, cytokine release syndrome has been proposed to be the main pathophysiology of COVID-19-induced acute fulminant myocarditis [3].

In this case report, we report a patient with a sore throat and myalgia that developed dyspnea orthopnea and finally diagnosed as myocarditis and COVID -19 infections.

CASE REPORT

A 52-year-old woman has been referred with shortness of breath and orthopnea form 7 days ago. She mentions sore throat and myalgia from 20 days ago that have not improved. She mentions frequent cough and dyspnea at rest that get worse with laying and physical activities. Past medical and drug history was negative except a recent flu like symptoms. On examination, the patient was a 52 years old woman, who has tachypnea and respiratory distress. Jugular veins were engorged. Blood pressure on admission was 130/80 mmHg, heart rate was 140 beats/minute and the patient had a body temperature of 37.5°C and SpO2=89% in room air. Auscultation of lung revealed fine crackle and rhonchi in both lungs; heart auscultation revealed tachycardia and S3 gallop and holosystolic murmur in left lower sternal border and apex. On ECG she had sinus tachycardia and non-specific ST/T changes.

In lab tests, WBC=7000(PMN=%85, Lympohocyte=%15), Hb=18.3, Hct=%52, Plt=90000, BUN=27,Cr=1.6,Na=128,K=5.4,Terigycerid=99,LDL= 116,HDL=23, Cholesterol=159,ESR=6, CRP=50, Troponin=Negative, CKMB=15. She admitted to intensive care unite and undergo echocardiogram. Echo showed moderate left ventricle enlargement, severe left ventricle systolic dysfunction, left ventricle ejection fraction (LVEF) was 15%, global hypokinesia of left ventricle, normal right ventricle size with severe systolic dysfunction, severe central jet mitral regurgitation, severe tricuspid regurgitation, mild aortic regurgitation. The patient undergo treatment with IV furosemide 20 mg three time daily ,nitroglycerin 20-40 $\mu g/min$, captopril 25 mg twice daily, spironolactone 25mg once daily subcutaneous heparin 5000 unit twice daily, pantoprazole 40mg oral daily and oxygen supplement with face mask. She undergo lung HRCT tomorrow and showed multiple ground glass opacities in the both lung highly suggestive of viral pneumonia including COVID-19 is noted (figure2). Then nasopharyngeal PCR was taken, which was positive for COVID-19. She was treated with interferon beta-19 9.6 mU subcutaneous Every other day for 5 days, dexamethasone IV 4mg twice daily, azithromycin 250 mg oral daily and ceftriaxone 1g twice daily for 5 days. She was eventually discharged in relatively good clinical condition.

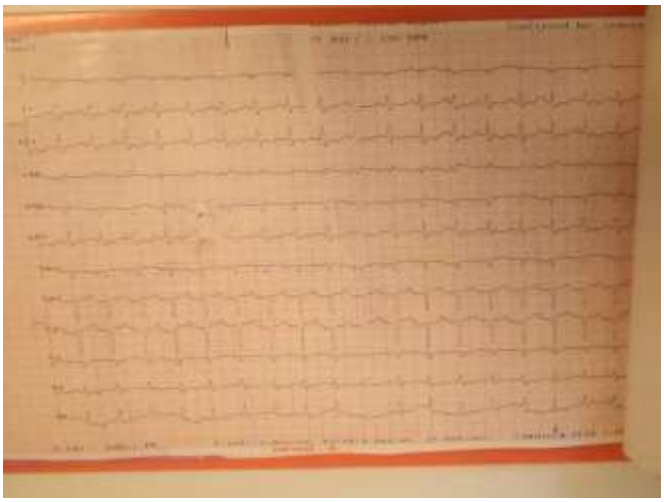

Fig-1: ECG shows sinus tachycardia and non-specific ST/T changes

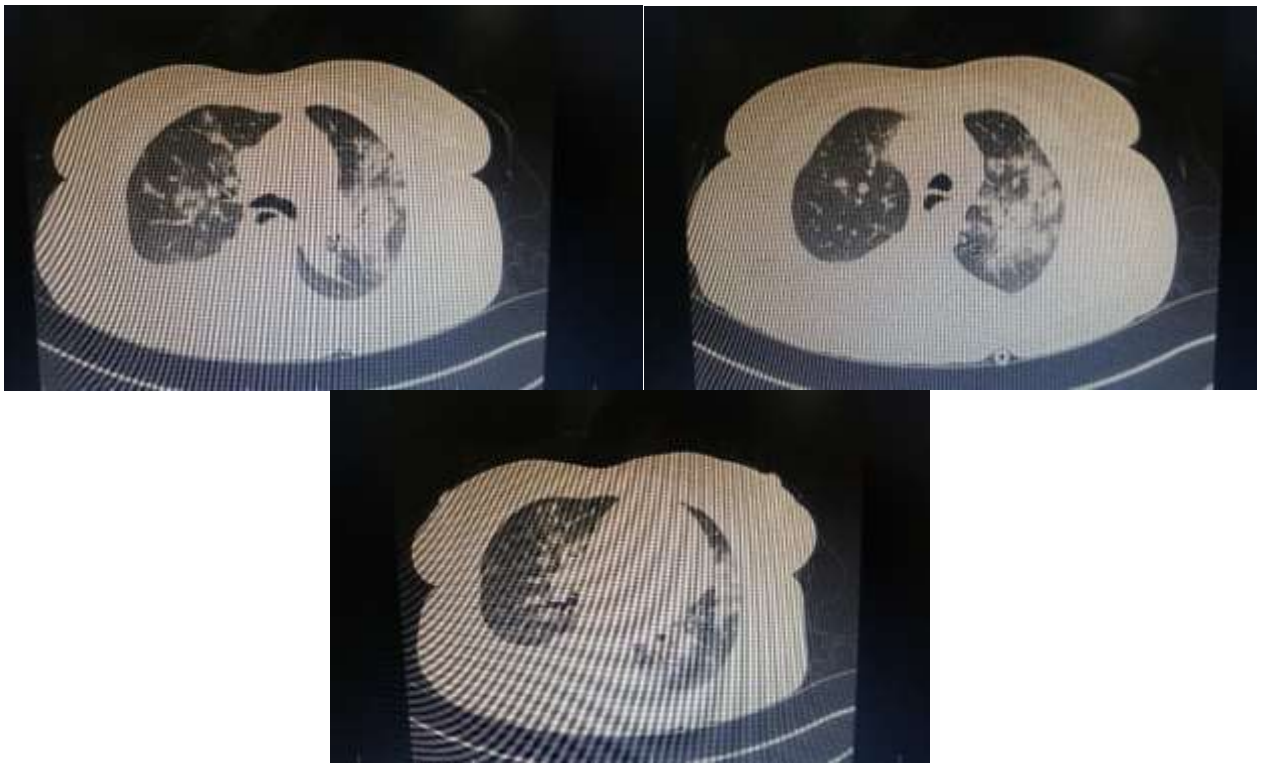

Fig-2: Ground glass opacities in both lungs suggestive of COVID-19 pneumonia

DISCUSSION

We reported a case of COVID-19 infection that referred due to orthopnea and sever dyspnea following CIVID infection.

The pathophysiology of COVID-19–related myocarditis is thought to be a combination of direct viral injury and cardiac damage due to the host's immune response. Diagnosis should be guided by insights from previous coronavirus and other myocarditis experience. The prevalence of myocarditis among COVID-19 patients is unclear, partly because the early reports often lacked the specific diagnostic modalities to assess myocarditis. Some argued that up to 7% of COVID-19 related deaths were attributable to myocarditis [4]. However, this was assumed and not based on confirmatory diagnoses of myocarditis and thus may be an overestimate. In contrast, many laboratories are rationing tests and are first screening for known pathogens resulting in flulike symptoms. It is possible that many cases of COVID-19–related myocarditis were missed due to a lack of SARS-CoV-2 diagnosis. Clinical presentation of SARS-CoV-2 myocarditis varies among cases. Some patients may present with relatively mild symptoms, such as fatigue and dyspnea [5, 6] whereas others report chest pain or chest tightness on exertion [7, 8]. Many patients do deteriorate, showing symptoms of tachycardia and acute-onset heart failure with cardiogenic shock. Results of blood tests from myocarditis patients often show elevated levels of lactate and other inflammatory markers, including C-reactive protein, erythrocyte sedimentation rate, and procalcitonin, which usually are raised in keeping with the clinical presentation of infection. Testing patients for baseline cardiac enzymes (eg, troponin and N-terminal pro–B-type natriuretic peptide [NT-proBNP]) on hospital admission, as cardiac troponin I (cTnI), cardiac troponin T (cTnT), NT-proBNP, and BNP levels usually are elevated in myocarditis due to acute myocardial injury and possible ventricular dilation. Elevations of both troponin and NT-proBNP levels were observed in the COVID-19–related myocarditis cases [8]. Electrocardiogram (ECG) abnormalities commonly seen with pericarditis, such as ST elevation and PR depression, may be observed in myocarditis [9]. Differential diagnoses include acute coronary syndrome, Sepsis-related cardiomyopathy, Stress-induced cardiomyopathy (Takotsubo cardiomyopathy). Diagnostic evaluation for COVID-19–related myocarditis include echocardiogram, cardiovascular magnetic resonance (CMR), Contrast-enhanced cardiac CT and endomyocardial biopsy (EMB) as the definitive diagnostic tool for myocarditis The American Heart Association (AHA) recommends further testing for patients having signs consistent with myocarditis with 1 or more cardiac imaging methods such as echocardiogram or cardiovascular magnetic resonance (CMR) [10]. Management of COVID-19–related myocarditis administration of inotropes and/or vasopressors and mechanical ventilation for cardiogenic shock in patients with fulminant myocarditis, Longer-term management involves mechanical circulatory support such as extracorporeal membrane oxygenation, ventricular assist device, or intra-aortic balloon pump [9]. It is reasonable to withhold or minimize the use of immunosuppression in SARS-CoV-2 patients, especially in the setting of a positive viral genome on EMB. Tocilizumab, an anti–IL-6 receptor monoclonal antibody, is now being tested in a multicenter randomized controlled trial that recruits COVID-19 patients with raised IL-6 levels [11]. Chloroquine and its derivative, hydroxychloroquine and agents used empirically to treat COVID-19, including ritonavir/lopinavir and azithromycin, are known CYP3A4 inhibitors, may cause QTc interval prolongation. Hence, their combination therapy with (hydroxy) chloroquine should be accompanied by QTc interval monitoring [12]. Cautions must be taken for the use of the NSAIDs and QTc-prolonging drugs in COVID-19 patients because these medications might exacerbate cardiac symptoms.

CONCLUSION

Based on the findings, it seems that myocarditis is important complication that may be seen following COVID-19 infection. Clinical status and Simple bedside tests such as serial ECG and cardiac biomarkers can raise suspicion of acute-onset cardiac symptoms. Cardiac imaging techniques such as echocardiography and CMR can be used to aid diagnosis. And definitive diagnosis of myocarditis is obtained via EMB.

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