Case study

CASE STUDY - AN INTERESTING CASE OF COVID19 INDUCED FULMINANT MYOCARDITIS

Abstract

A young patient was presented to the Emergency Department, aged 31 years old, initially presented somewhere else with complaints of mild fever, throat pain, and generalized myalgia. As per suspicion, a test for COVID19 was ordered, which came out to be positive, thus confirming that the patient was suffering from COVID19.

Since the patient's chest CT scan came out to be normal and without any consolidations which are characteristic of COVID19, he was prescribed Augmentin and an antipyretic, advised for strict home isolation, and then sent back home.

However, two days later, the same patient reported to the Emergency Department once again. This time, his symptoms had turned severe. He was now experiencing extreme shortness of breath, an exaggerated cough, and above all, extreme fatigue and exhaustion which was evident from his overall appearance.

Now, the patient was examined once again to see what developments had taken place over this period lasting two days.

Generally, the patient appeared to be severely ill and had both tachycardia and tachypnea. His oxygen saturation levels were 85%, which are low and required him to get oxygen support immediately.

Comment [K1]: tautology

Furthermore, his neck veins were severely congested along with a raised JVP, he had distant heart sounds, and his chest auscultation revealed bibasilar crackles. The left lobe of the liver was also palpable upon palpation. There was, however, no indication of any ascites, or fluid retention anywhere else in the body.

Comment [K2]: tautology

Concluding it medically, this case was a classic presentation of Acute Fulminant Myocarditis which occurs secondarily due to COVID19. The diagnosis took time, but fortunately, the patient had presented to the Emergency at the right time and this led to him getting admitted, evaluated, and treated within the safe period before any other complication took place. Although patients with a pre-existing heart condition or predisposition to develop any heart condition are more inclined towards developing such cases, it can affect almost anyone, owing to the complicated nature of the virus itself.

Key words: Myocarditis, Fulminant,Coronavirus,COVID-19

INTRODUCTION

Severe Acute Respiratory Syndrome-Coronavirus-2 (SARS-CoV-2) continues to pose a constant challenge for humanity to date. This virus does not only have a confusing nature, the long list of complications and diseases that are now arising as a consequence of developing the disease initially are becoming a source of concern for the doctors and the experts who are working to completely eliminate this evil virus from the society.

Cases of COVID19-associate myocarditis were being heard from here and there, but now it has almost become an established fact that COVID19 frequently causes myocarditis of varying degrees in some recovering or suffering patients.[1]

Although explaining myocarditis in terms of the COVID19 era might sound like a difficult idea, it is postulated that it should be diagnosed and evaluated on the same grounds as before. The only addition is the fact that now it occurs in association with COVID19 and not alone.

COVID19-associated myocarditis is seen to occur due to a series of events that follow an initial viral injury and the resultant immune response causing damage to the cardiac walls and muscle. Acute fulminant myocarditis is a rare event that occurs due to the inflammation of the cardiac muscle. When it occurs, it significantly reduces the ability of the heart to pump blood efficiently. Because of this failure of the heart to function properly, acute fulminant myocarditis is seen to have higher than normal mortality rates, ranging from 40-70% approximately.[2]

Based on the classification, cases of –Fulminant Myocarditis are divided on the basis of their histological appearance into lymphocytic, giant cell myocarditis, eosinophilic, and sarcoid heart disease. The lymphocytic form is the most deeply studied one, and is again, further subdivided into infective and non-infective forms. Where the confirmation of a viral etiology was not possible, cardiac muscle biopsy is done. This helped confirm the fact that approximately 78% of all cases of -Fulminant Myocarditis occurred because of a viral etiology. [3]

CASE STUDY

A 31 years old patient presented to the hospital with complaints of having a mild fever, sore throat along with throat pain, and generalized myalgia in his body for the last few days. No other systematic problems or abnormalities were reported. As part of the new hospital treatment protocols, the patient was ordered to get his COVID19 test done. This test came out to be positive and thus, the patient was ordered to go directly into home isolation. Since the patient was stable and well-oriented and had no other respiratory symptoms that could have complicated his case, it was decided to not admit him to the hospital. Moreover, his CT chest had come out clear as well, which meant that his COVID19 infection was mild and had not involved the lungs or the respiratory system. Therefore, the patient returned home with an antipyretic and Augmentin to take care of his sore throat, pain, and fever.

However, the patient returned within two days, this time to the Emergency. Now, the patient had developed full-blown, severe symptoms. He had a severe cough, extreme shortness of breath, and severe fatigue. This condition rendered a complete and detailed examination of the patient.

Examination

On examination, the patient appeared to be extremely exhausted, fatigued and withdrawn from his surroundings. He had tachypnea and tachycardia.

The following points were noted during his systemic examination:

- BP = 104/70 mm of Hg
- PR = 120/min
- Temperature = 39.6 C
- Respiratory Rate = 26/min
- O_2 Saturation = 85%

- The patient had congested neck veins,
- His JVP was raised,
- Distant heart sounds were heard on auscultation,
- Chest examination revealed bibasilar crackles,
- The left lobe of the liver was tender and palpable,
- There were no ascites anywhere,
- The patient was also noticed to have positive symmetrical pitting lower limb edema,
- There was neither any skin rash nor any neurological involvement.

This entire list of examinations was unclear without any background information or new investigations. Therefore, investigations were ordered.

Investigations

The entire investigations along with their findings could be summarized below:

CBC Report:

- HB 14.6 g/dl had dropped to 10.6 g/dl
- PLT = 68,000
- TWBC = 16,700
- MCV = 73
- MCH = 26

Serum/Electrolytes Investigations

- Urea = 16 mmol/lit

- Creatinine = 272micomol/lit
- Na ions = 133 mmol/lit
- K ions = 4.0 mmol/lit
- CRP = 435 mg/lit
- Troponin 237 ng/lit increased next day to 78480 ng/lit
- CPK = 4752 IU/Lit
- Serum Ferritin 1866 microg/lit increased next day to 5988 microg/lit
- BNP = 29400pg/ml
- COVID 19 PCR = Still Positive
- Procalcitonin = More than 100 ng/ml
- Interleukin-6 = 259pg/mL(less than 7.0)

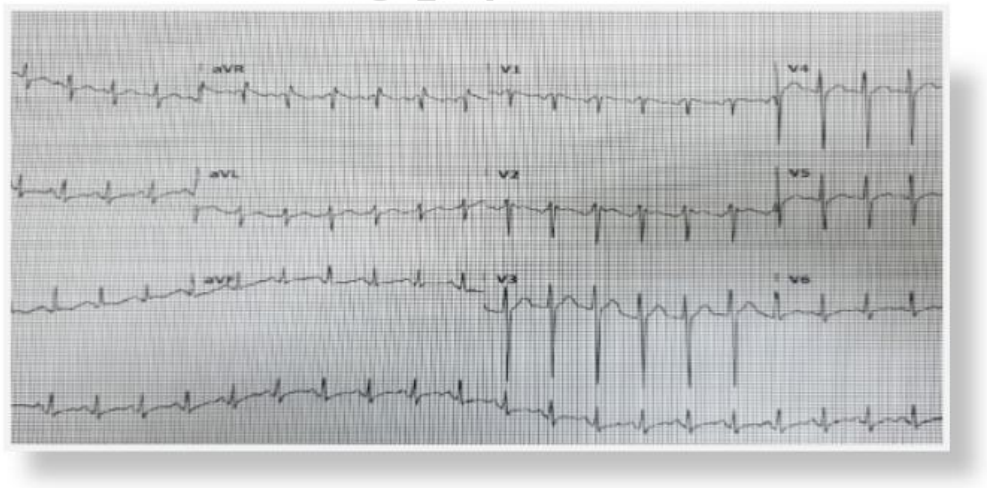

Fig 1: Imaging studies revealed the following results:

ECG = Sinus Tachycardia

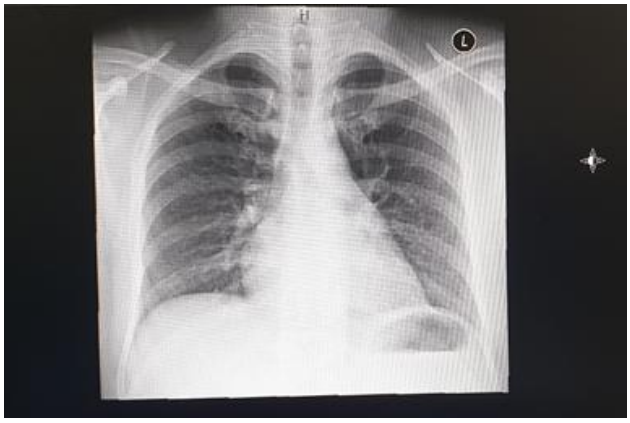

Fig 2: Normal chest x-ray (-First presentation-)

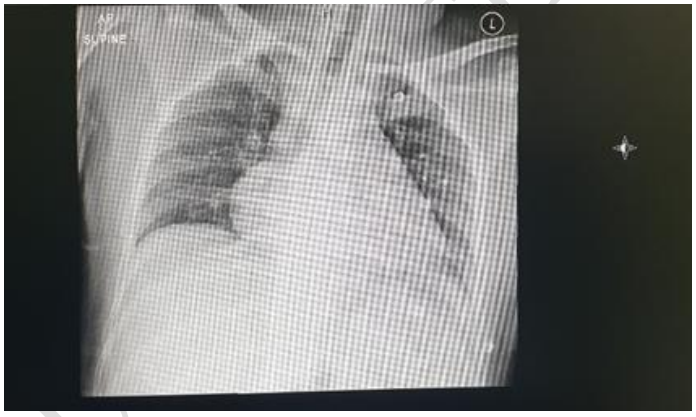

Fig 3: CXR = Cardiomegaly

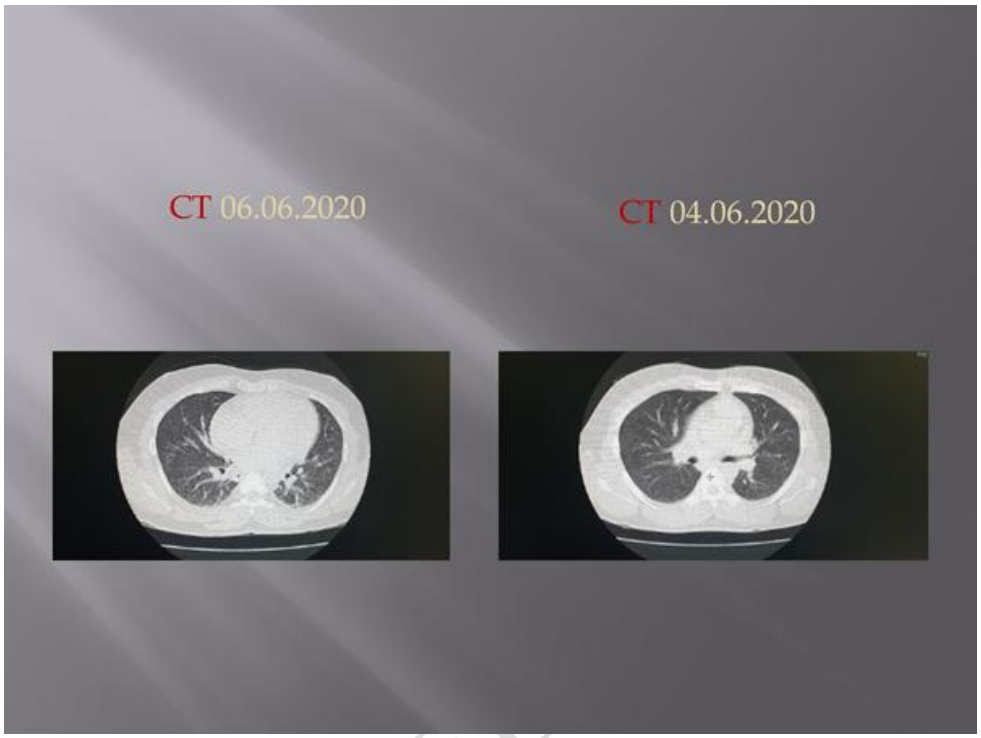

Fig 5: CT HR Chest = No lung involvement

Differential Diagnosis*:*

Based on all the investigations and the examinations done above, it was evident that there was an obvious involvement of the heart in this scenario. Therefore, keeping all the findings in mind, the following differential diagnosis were prepared:

- COVID19 Induced Fulminant Myocarditis
- Acute coronary syndrome
- Sepsis-related cardiomyopathy
- Stress-related cardiomyopathy

Since it was not yet clear which condition was causing the symptoms, it was decided that a cardiac bed should be ordered for the patient. The patient was started on parenteral Furosemide along with constant Oxygen support, which led to some hopeful improvements in the symptoms.

Diagnosis & Management:

The patient was directly admitted to the ICU, where he was confirmed as a case of COVID19Induced Myocarditis. After this confirmation, the patient was started on a targeted therapy consisting of Furosemide, Inotropes, and Vasopressors (Dobutamine and Norepinephrine).

His echocardiogram done at that time revealed Global Dyskinesia with an Ejection Fraction of 20%.

The patient was made to complete his course for COVID19 as well. This regime had been upgraded to Camostat and Favipiravir along with Methylprednisolone for 3 days.

The echo was repeated again on Day 6. It now revealed EF 55% with normal heart size.

On Day 7, the patient had been extubated. He continued to be on Oxygen support (2 L/min) for two more days and then finally discharged in a good and healthy state.

DISCUSSION

COVID19 Induced Fulminant Myocarditis is a recently added condition in the list of the diseases that have been occurring due to the COVID19 infection. [4]

The COVID19 virus has been seen to have a significant impact on cardiovascular health as a whole. The involvement of the heart could occur in COVID19 irrespective of whether there are any significant respiratory symptoms or not. On the same side, viral diseases were found to be the prevailing causes for giving rise to heart-related conditions such as myocarditis, myopericarditis, pericarditis, etc. [5]

Specifically, a cardiac injury that is accompanied by significant troponin leakage is found to be responsible for mortality associated with COVID19. Up to 7-8% of the deaths due to COVID19 were found to occur due to myocarditis arising as a secondary complication. [6]

Myocarditis in COVID19 patients could occur due to several reasons. An exaggerated immune response may give rise to a cytokine storm, which may then cause an increased vascular permeability, apoptosis, and T-cell and antibody responses. Along with these responses, a rise in the cardiac biomarkers was also noticed, which means that the heart was equally involved in the series of changes taking place in this setting. Otherwise, it was also hypothesized that the hypoxia which could have occurred due to COVID19 infection could have given rise to cardiac damage which was significant to cause myocarditis. [7]

The clinical features or the presentation of the patient varies based on the severity of the damage done. The patient could be asymptomatic or he could be in a state of cardiogenic shock. The symptoms were also seen to mimic those of acute or long-standing heart failure.

Fatigue, chest pain on or without exertion, elevated jugular venous pressure, right upper quadrant abdominal discomfort, and peripheral edema were seen to occur in the patients, mimicking right-sided heart failure. The case of the patient discussed above had a similar presentation.

The diagnosis protocol is the same as is followed for any viral disease. The clinical presentation of the patient is very important. In addition to that now, travel history or exposure to sick or already diagnosed contacts, a thorough physical examination, and laboratory tests including COVID-19 PCR and inflammatory and cardiac markers need to be obtained to reach a conclusive diagnosis. [8]

As far as the treatment is concerned, a proper treatment regimen needs to be formulated for this condition. Presently, only supportive treatment is catered to the patient. NSAIDs, Colchicine, Corticosteroids, Intravenous Immunoglobulin (IVIG), and Plasma Exchange therapy stand as the standard mode of treatment for patients struggling with this disease. [9]

With new data and statistics arising, a definite solution to put the myocarditis to rest would surely arise.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

REFERENCES:

1. Chen, C., Zhou, Y., & Wang, D. W. SARS-CoV-2: a potential novel etiology of fulminant myocarditis. Herz, (2020) 45(3), 230–232. https://doi.org/10.1007/s00059-020-04909-z

2. Cooper LT., Jr. Myocarditis. N Engl J Med. 2009;360(15):1526–1538. doi: 10.1056/NEJMra0800028

3. Kuhl U, Pauschinger M, Noutsias M, et al. High prevalence of viral genomes and multiple viral infections in the myocardium of adults with "idiopathic" left ventricular dysfunction. Circulation. 2005;111(7):887–893. doi: 10.1161/01.CIR.0000155616.07901.35

4. Chen N, Zhou M, Dong X, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020 doi: 10.1016/S0140-6736(20)30211-7.

5. Pankuweit S, Moll R, Baandrup U, et al. Prevalence of the parvovirus B19 genome in endomyocardial biopsy specimens. Hum Pathol. 2003;34(5):497–503. doi: 10.1016/s0046-8177(03)00078-9

6. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). JAMA Cardiol. 2020;5(11):1265–1273. [

7. Tavazzi G, Pellegrini C, Maurelli M, Belliato M, Sciutti F, Bottazzi A et al Myocardial localization of coronavirus in COVID-19 cardiogenic shock. Eur J Heart Fail (2020) 22:911–915

8. Khatri, A., & Wallach, F. Coronavirus disease 2019 (COVID-19) presenting as purulent fulminant myopericarditis and cardiac tamponade: A case report and literature review. Heart & lung: The Journal of Critical Care (2020)., 49 (6), 858–863

9. Farina A, Uccello G, Spreafico M, Bassanelli G, Savonitto S. SARS-CoV-2 detection in the pericardial fluid of a patient with cardiac tamponade. Eur J Intern Med. 2020;76:100–101.