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A case of acute myocarditis with no viral prodrome with review of literature

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Abstract

A significant side effect of coronavirus disease 2019 (COVID-19), a disorder brought on by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus, has been identified as myocarditis. Comparing COVID-19 myocarditis to other viral etiologies, it appears to have distinctive inflammatory features. As a wide range of figures have been cited in the literature, it is still unclear how common is COVID-19 myocarditis. However, it appears that the risk of myocarditis rises with more severe infection. Additionally, injection of the mRNA COVID-19 vaccine has been linked to myocarditis development, especially following the second dose. The symptoms of COVID-19 myocarditis range widely, from dyspnea and chest pain to severe heart failure and possibly death. The symptoms of COVID-19 myocarditis range widely, from dyspnea and chest pain to severe heart failure and possibly death. It is crucial to identify any cases of myocarditis, especially those that show with fulminant myocarditis, which might exhibit heart failure symptoms and arrhythmias. Electrocardiograms and serial troponins should be part of the initial work-up for suspected myocarditis. Additional screening should be done if these tests reveal myocardial injury. The most effective tests for myocarditis are endomyocardial biopsy and cardiac magnetic resonance imaging. However, the combination of intravenous immunoglobulins and corticosteroids may be effective, especially in cases of fulminant myocarditis. Treatment for COVID-19 myocarditis is still debatable. Here we present a case acute myocarditis in a young male with no underlying comorbidities without any viral prodrome.

Keywords: fulminant myocarditis, endomyocardial biopsy, IVIG.

Case Presentation

A 22 years old male presented to emergency room with history of sudden onset shortness of breath for 3 hours with feeling of tightness in chest. On arrival patient was afebrile, tachypneic, with 55% saturation at ambient air recorded by pulse oximetry, blood pressure was 150/90 mmHg recorded on right arm in sitting position. Physical examination revealed signs of hypoxemia with bilateral rales on pulmonary auscultation. Patient has no underlying comorbidities with no history of fever,

malaise, myalgias or cough preceding this illness. Nasopharyngeal swab for rapid antigen test and polymerase chain reaction for SARS-CoV-2 taken on day of arrival came positive. Although patient didn't recall any contact with COVID positive person.

Fluid resuscitation initiated and laboratory investigations were obtained. A 12-lead ECG demonstrated sinus tachycardia with no signs of myocardial ischemia.

Investigations

Laboratory investigations revealed neutrophilic predominant leucocytosis ($12x10^9$/L), elevated troponin levels, high CRP (48mg/L), D-dimer (3.84ng/L), CK (176U/L), creatinine (0.71mg/dl), procalcitonin (0.05ng/mL) IL-6 (56pg/mL), lactates of 6.9 mmol/L, NT-proBNP levels were raised (4148pg/ml). HRCT chest was obtained and showed multifocal peribronchial ground glass opacities with few pleuro-parenchymal bands in left lower lobe with CORADS 4. In view of hemodynamic instability and acute myocardial dysfunction transthoracic echocardiography was done and revealed reduced ejection fraction (38%), global hypokinesia with maintained wall thickness, no pericardial effusion.

Serial investigations on subsequent days revealed decreasing trend of levels of inflammatory markers. Cardiac MRI was deferred for a later date.

Differential Diagnosis

Given the clinical scenario and the laboratory investigation it was highly suggestive of myocarditis, so diagnosis of COVID-19 related acute myocarditis was made. No evidence of regional wall motion on echocardiography was detected and 12-lead ECG was also not revealing any evidence of ischemic cardiac disease and in view of hemodynamic instability cardiac angiogram was deferred as per cardiologist opinion.

Treatment

Patient was started on inotropes on arrival and serial cardiac and BP monitoring was done. As pulmonary auscultation was suggestive of cardiogenic pulmonary edema so IV diuretics were added as per BP. Dexamethasone 6mg IV was started on first day till day 5. After obtaining positive test for SARS-CoV-2 antiviral remdesivir was started.

Outcome and Follow-Up

Patient became hemodynamic stable after 2 days. Patient was discharged after 6 days. Patient was advised to start ACE inhibitors and SGLT-2 inhibitors. Follow up echocardiography revealed improvement in ejection fraction after 3 week. Outpatient cardiac MRI has been advised with cardiology follow-up.

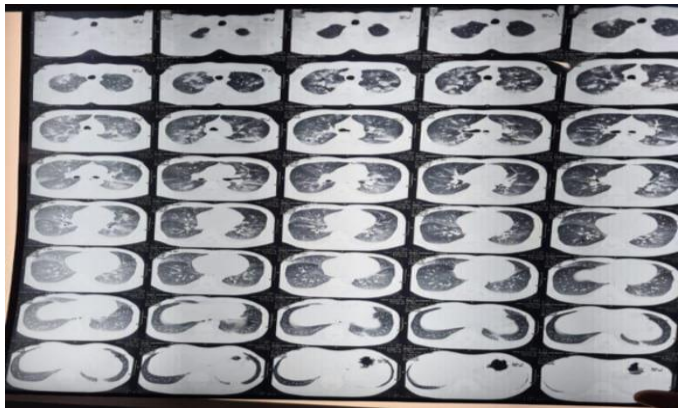

Fig. 1: CT chest shows multifocal peribronchial ground glass opacities with CORADS-4

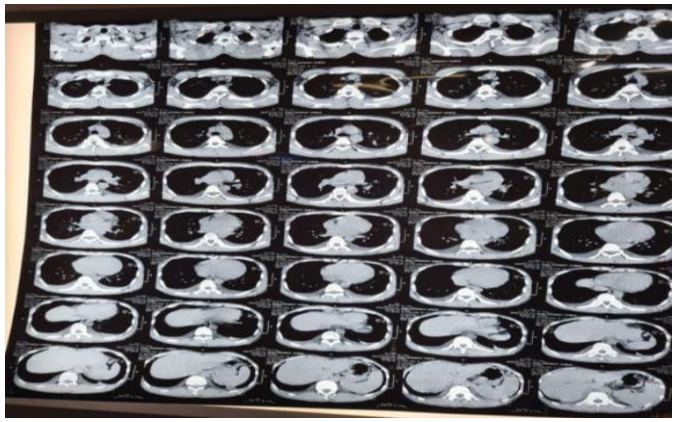

Fig.2: CT chest shows bilateral minimal pleural effusion with cardiomegaly

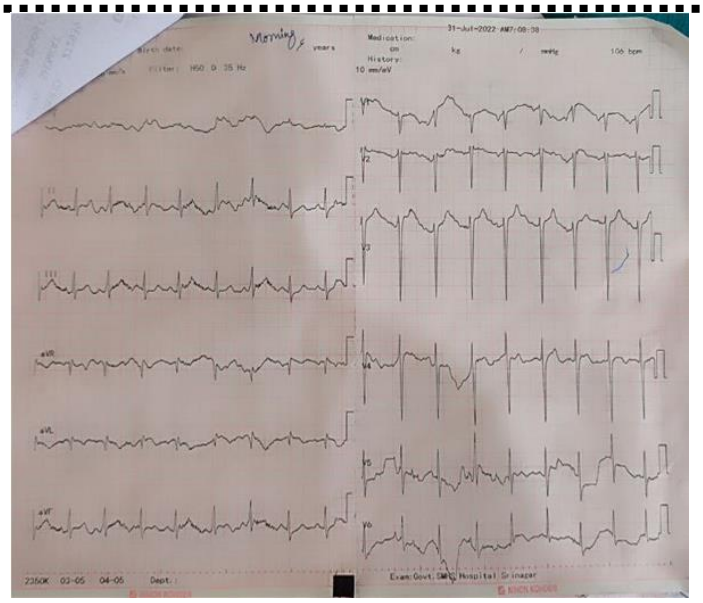

Fig. 3: ECG shows sinus tachycardia with ventricular rate of 106bpm

Introduction

The coronavirus sickness 2019 refers to a pneumonia infection outbreak that started in Wuhan, China, in December 2019. (COVID-19). The severe acute respiratory syndrome coronavirus 2 was identified as the root cause (SARS-CoV-2). The World Health Organization proclaimed COVID19 a global pandemic on March 11, 2020, as a result of the virus' quick global spread. By August 31st, 2021, there have been more than 4.5 million fatalities and over 200 million confirmed cases of COVID19 worldwide.The SARS-CoV-2 virus's ability to bind and enter host cells depends on the spike (S) protein. The S protein consists of two subunits, S1 and S2, where S1 facilitates binding to host cells and S2 orchestrates the fusion of the viral and host cell membranes. The S protein interacts to the receptor angiotensin-converting enzyme (ACE)-2 [5]. Once binding has taken place, the SARS-CoV-2 virus can enter the host cell by fusing its membrane with the host cell. Type 2 trans-membrane serine protease (TMPRSS2), a cell surface enzyme that cleaves ACE-2, mediates the fusing of membranes.Following viral replication and an immunological response after entry into host cells, COVID-19 clinical symptoms and tissue damage result [4].Cough, dyspnea, and fever are the usual signs of pneumonia caused by COVID-19 [8]. But it has recently been discovered that COVID-19 can lead to cardiovascular issues, including myocarditis.The COVID-19 myocarditis illness course can be minor to severe. In order to prevent the myocarditis from progressing to potentially fatal heart failure and arrhythmias, it is crucial for clinicians to identify suspected COVID-19 myocarditis cases and treat them appropriately [9–11]. Additionally, it can result in more ward admissions at a time when hospitals may already be overburdened. This review of the literature intends to explore the pathophysiology, prevalence, presentation, diagnosis, and management of COVID19 myocarditis.

The cause and treatment of viral myocarditis

Inflammation of the heart muscle that results in damage in the absence of ischemia is referred to as myocarditis [12, 13]. Adenovirus, parvovirus B19, Epstein-Barr virus, and CMV are only a few of the many viruses that have been implicated as substantial contributors to the pathogenesis of myocarditis [13–16].The SARS-CoV-2 virus may now also be an important infectious agent for myocarditis, according to recent research. Direct cell injury and immune-mediated cell death are combined to form the pathogenesis of viral myocarditis [12].

High viral replication rates early in viral myocarditis development cause direct cardiomyocyte damage [17]. Toll-like receptors and inflammasomes are activated by the injured cells and proteins produced from them (such as cardiac myosin), which causes the production of pro-inflammatory cytokines [18, 19]. These pro-inflammatory cytokines gradually draw immune cells, such as macrophages, T-lymphocytes, and natural killer cells, to the myocardium. Immune-mediated myocyte

damage is caused by these cells [17]. Additionally, dilated cardiomyopathy and heart failure are finally brought on by interleukin (IL)-1 and IL-17-induced cardiac remodelling and fibrosis [20, 21]. A disturbance in the conduction pathway brought on by myocardial fibrosis increases the probability of arrhythmia development [22].

Proposed pathways for myocarditis caused by COVID-19

As previously reported, the SARS-CoV-2 virus attaches to the ACE2 protein before entering human cells. These proteins can also be detected on cardiomyocytes, even though the respiratory manifestations of COVID-19 are caused by the expression of the ACE2 protein on epithelial cells (type II alveolar cells) of the respiratory tract [23–25]. A COVID-19 patient's myocardium contained SARS-CoV-2 virus particles, according to a case study employing endomyocardial biopsy (EMB) [26]. In addition, autopsy of 20 human heart samples from SARSCoV patients—a virus similar to SARS-CoV2 showed that seven of the hearts had viral particles and had been infiltrated by macrophages [27]. The SARS-CoV-2 virus may therefore potentially infect cardiomyocytes, resulting in viral myocarditis [28]. The identification of SARS-CoV-2 in endothelial cells of several organs, including the heart, during histology [30, 31], is evidence in favour of this concept.

In contrast to patients with normal myocarditis and control groups, several researchers discovered that individuals with COVID-19 had more diffusely dispersed CD68+cells in their hearts [32]. According to Fox et alhypothesis's [32], COVID-19 myocarditis is a unique inflammatory condition distinct from ordinary viral myocarditis because of the difference in immune cells on the histology. There were two hypotheses put out to explain the inflammatory process. First, SARS-CoV-2 can cause endothelial cells in coronary vessels to become infected. This causes macrophages to migrate to these locations and activate complement and induce apoptosis [32]. Second, thrombus development in the coronary arteries brought on by inflammation may result in ischemia myocardial damage [32]. Myocarditis caused by COVID-19 may also be influenced by systemic inflammation.

Additionally, cytokine storm, a potentially fatal disease caused by high elevations in pro-inflammatory cytokines and an unregulated immune response, is primarily mediated by IL-6 in some COVID-19 patients [33, 34]. Due to platelet activation and elevated levels of clotting factors (including factor V and VIII), this systemic inflammation can further raise the likelihood of thrombus formation inside coronary arteries [29, 35]. Additionally, the cytokine storm may cause an aggravation of pre-existing myocarditis and additional cardiac damage [28]. Additionally, myocardial injury may be made worse by hypoxia of the myocardium as a result of higher oxygen demands brought on by infection that cannot be fulfilled because of pneumonia or acute respiratory distress syndrome [36]. Fig. 1 depicts the potential pathogenesis of COVID-19 myocarditis.

Possibility of contracting COVID-19 myocarditis

Uncertainty surrounds the occurrence of myocarditis caused by COVID-19. According to a research, 28% of COVID-19 patients had myocardial damage, which was identified by elevated troponin T levels [37]. According to a meta-analysis, 8% of COVID19 patients experienced myocardial damage, with patients in intensive care having a 13-fold higher frequency [38].Less than 2% of 277 postmortem cases with myocarditis were found to have clinically significant myocarditis, according to Halushkaet al. [39]. The results of this study suggest that the true incidence of COVID-19 myocarditis may be

underreported since some patients may not have any symptoms or only experience minor ones.

In a study of 100 patients who had recently recovered from severe COVID-19, Puntmannet al. discovered that 60% of them had persistent inflammation and that 78% of them had cardiac involvement on cardiac magnetic resonance imaging (cMRI) [40]. Myocarditis is a severe COVID-19 consequence, although it's uncertain how many people will die as a result. Myocardial involvement increases mortality in COVID-19 hospitalised patients, according to published data [41].

According to Qiurong et al. analysis's of 68 COVID-19 patient deaths, 33% of these deaths were caused by a combination of respiratory and cardiac failure, whereas 7% of these deaths were a result of fulminant myocarditis that caused circulatory failure [42]. Based on an analysis of the clinical information that was available to the authors, fulminant myocarditis diagnosis were determined. The most accurate method of identifying myocarditis was not mentioned immune-histological investigation. Due to the increased possibility of incorrect diagnoses, this could alter how reliable these results are. Patients with COVID-19 infection may have a worse prognosis after developing myocarditis, whereas those who do so may experience long-term cardiovascular consequences that need to be further researched. Patients with cardiovascular co-morbidities had a higher risk of developing COVID-19 myocarditis, according to a systematic analysis and case series [43, 44].It's unclear exactly how this happens or what the underlying process is. According to Guo et al. theory's [45], the virus could enter the pulmonary circulation after infecting pneumocytes via endothelial cells that express the ACE2 gene.

Furthermore, it has been shown that failing hearts express more ACE2 proteins than healthy hearts do [45-47]. The SARS-CoV-2 virus may be more readily absorbed in these hearts due to the greater levels of ACE2 there [46]. In individuals with cardiovascular illness, the SARS-CoV-2 virus is likely to come into contact with ACE2 expressing cardiomyocytes because the heart is the first organ that the pulmonary outflow encounters [45]. These patients may be at a higher risk of developing COVID-19-induced myocarditis since SARS-CoV-2 binds to ACE2 to enter cells [28]. Due to a higher frequency of cardiovascular illness in these groups, Black, Asian, and Minority Ethnic (BAME) groups may be more severely impacted by COVID-19-induced myocarditis [48–52]. However, some data point to the expression of ACE2 being lower in people of African heritage, particularly in those with pre-hypertension [52]. More research is needed to determine whether race affects the likelihood of developing COVID-19 myocarditis because the information is conflicting.

Competitors in competitive sports should be especially watchful because myocarditis is linked to sudden cardiac mortality in athletes [53]. 1597 athletes were examined by Daniels et al. for signs of myocarditis caused by COVID19. Of these athletes, 37 (2.3%) had myocarditis according to the COVID-19 classification, and another 28 may have had it. Only five occurrences of COVID-19 myocarditis would have been documented, according to Daniels et al., if cardiac testing had only been performed on patients who had cardiac symptoms [54]. This once further emphasizes the idea that COVID-19's cardiac involvement may be understated because of asymptomatic patients [55]. Another study reported that 31% of the 26 competitive athletes who underwent cMRI had signs of prior myocardial injury, while 15% had myocarditis. The precise incidence of COVID-19 myocarditis is still unknown; however the available research indicates that those who experience a severe

infection are more likely to do so than those who experience a mild illness.

Association between myocarditis and the COVID-19 mRNA vaccination

Myocarditis may arise with the delivery of a COVID-19 mRNA vaccination (both Pfzer and Moderna). According to recent data, myocarditis rates among individuals aged 12-39, with a predominance of young boys, are approximately 12.6 instances per million administrations of the second dose of mRNA vaccines [56]. Two to three days following the second dosage of the vaccination, patients frequently complain of chest discomfort and aberrant ECG readings [56]. Symptoms frequently go away for patients [56]. Since the vaccination began to be distributed in the USA. Diaz et al. found an increase in the mean number of monthly cases of myocarditis/myopericarditis of 10.4 (p0.001) [57].

In addition, numerous case studies of patients who experience acute myocarditis after receiving the COVID-19 mRNA vaccine have been described [58–61]. It was confirmed that none of the investigated vaccine recipients had COVID-19 when they first presented. Six patients who presented after the second dose and one patient who presented after the first dose were tracked by Mouchet al. [58]. Four patients who had received both doses of the mRNA COVID-19 vaccination were tracked by Kim et al. [59]. Shaw et al. [60] monitored four patients: two of whom received the vaccine in two doses and the other two. Both of the patients who arrived after the first dose had already been infected with SARS-CoV-2. Every patient in these investigations had a cMRI for diagnosis. Last but not least, Montgomery et al. [61] monitored 23 male patients, 20 of whom appeared after the second dosage and three after the first dose (all of whom had already contracted SARSCoV-2). Only eight patients in this study had diagnostic cMRI, which reduces the reliability of the findings because most diagnoses would have been made using clinical judgement. In total, these four studies monitored 37 patients: 6 patients with prior COVID-19 infection who presented after the first dose of the mRNA vaccine and 31 patients who presented after the second dosage. This emphasizes the crucial idea that vaccine-induced myocarditis frequently develops following sensitization to SARS-CoV-2.

Thirteen of the 15 children (12–18 years old) in a case series who were hospitalized for myocarditis symptoms such as fever and chest discomfort, displayed cMRI abnormalities that were compatible with myocardial inflammation [62]. All but one of these patients showed up following the second vaccination dosage. This study shows that the delivery of mRNA vaccinations to children may also put them at risk for myocarditis. Children with COVID-19 had a low mortality and intensive care admission rate, according to recent research [63]. Therefore, the risk of myocarditis compared to the risk of severe COVID-19 infection in this age range should be taken into account when recommending the administration of mRNA vaccines to children (under 18 years old).

There is evidence that the COVID-19 mRNA vaccine, especially in cases of prior exposure, can result in myocarditis. Given that the majority of patients appear after receiving the vaccine's second dose or after receiving the first dose if they previously had SARS-CoV-2 infection, a hypersensitive reaction following the earlier exposure may be possible [61, 64]. Due to the two- to three-day delay between vaccination delivery and the onset of symptoms observed in the majority of patients, this reaction may be a delayed-type hypersensitivity reaction [56, 64].

The first dosage of the vaccine may function to sensitize the immune system, and the second dose may cause the

immune system's effector phase to become activated [64]. Myocardial cytokines may be released into the myocardiumm as a result of immune cells that have been activated migrating there. This procedure may prompt additional immune cells to enter the myocardium, causing inflammation and the possibility that the patient could develop myocarditis.

Myocarditis with COVID-19 presentation

Myocarditis typically manifests as heart failure-like symptoms such as dyspnea, orthopnea, and possible chest discomfort [65]. However, COVID-19 myocarditis patients' clinical manifestations can differ from patient to patient. Some individuals report with very minor symptoms as fever, cough, and dyspnea [9, 10, 22, 66, 67]. These symptoms could be brought on by COVID-19 rather than myocarditis. As a result, COVID-19 myocarditis may appear silently in certain patients [22]. Chest pain that may or may not be described as a pressure may be present in some patients [68–70]. This chest pain wasn't accompanied by exhaustion, a cough, or dyspnea in one report [68]. Along with their other symptoms, some patients may also experience palpitations [67, 71].If treatment is delayed or insufficient after initial presentation, patients may deteriorate and show indications of heart failure and hemodynamic compromise [9-11]. Without a history of cardiovascular disease, patients may first arrive with severe cases of new-onset heart failure [71]. This is a fulminant myocarditis presentation, a syndrome marked by abrupt and severe cardiac inflammation that can result in arrhythmias, severe heart failure, or even death [72, 73]. Patients who present with hypotension, ECG alterations (such as ventricular tachycardia, bradyarrhythmias, or ST depression), or clinical symptoms of heart failure including peripheral edoema should be closely monitored by doctors [73].

COVID-19 myocarditis diagnosis

Patients with COVID-19 myocarditis have been observed to have elevated levels of C-reactive protein (CRP), lactate dehydrogenase (LDH), and white blood cell count (WCC) [68-70].As indicators of infection, these blood tests are not specific for myocarditis. In COVID-19 myocarditis, elevated levels of cardiac enzymes, such as troponin, and N-terminal pro-B-type natriuretic peptide (NT-pro-BNP), have also been reported [44, 70]. As a result, it is important to measure the baseline levels of troponin I/T and NT-pro-BNP when a patient with COVID-19 is admitted in order to establish the trend of these tests during the course of the patient's stay [28]. A normal troponin does not rule out myocarditis because certain COVID-19 myocarditis patients may not have an elevated troponin [22, 74].

In fact, myocarditis has a 34% sensitivity for increased troponin I levels [75]. Patients who acquire myocarditis may also experience alterations in their electrocardiogram (ECG). Sinus tachycardia, which is the most frequent change, ST segment elevation/depression, T wave inversions, tachy / bradyarrhythmia, and QT prolongation are among the most common non-specific changes [44, 70]. Therefore, ECG abnormalities cannot be used to diagnose myocarditis, but they can be useful in evaluating the severity of the disease or the possibility of myocardial damage or the occurrence of arrhythmias. However, echocardiography also reveals generalized alterations in myocarditis, such as decreased ejection fraction, pericardial effusion, and hypokinesis across the entire heart wall [74-76]. To rule out further causes of heart failure, such as valvular or congenital reasons, echocardiography can be helpful. The best noninvasive test is cMRI since it has a high sensitivity for detecting myocarditis [77]. When analyzing cMRI pictures, the Lake Louis criterion ought to be applied [78]. To identify

myocardial edoema, hyperemia, myocardial necrosis, and myocardial fibrosis, this criterion combines T2-weighted images with early and late gadolinium enhancement [78–80]. The drawback of cMRI is that it cannot tell whether the inflammation is brought on by an immunological reaction to the virus or by a viral infection of the myocardium [78]. Additionally, the application of cMRI can be restricted in patients who arrive with severe myocarditis leading to cardiogenic shock or hemodynamic instability [77, 81]. These patients may also be mechanically ventilated or suffer from tachyarrhythmias. When confirming the existence of myocarditis in such circumstances, it may be preferable to employ EMB, which is regarded as the gold standard test because it can identify the kind of inflammation [77]. In order to establish the diagnosis of COVID-19-induced myocarditis, samples from the biopsy can be sent for immunohistology and genomic analysis, which can detect SARS-CoV-2 RNA [82]. The Dallas criteria, which defined myocarditis as myocyte necrosis or damage linked to inflammatory infiltrates [83], were previously used to evaluate EMB samples. The Dallas criteria's dependability is in doubt because it has been demonstrated that 50% of virus-positive cases do not meet its requirements [29]. Due to the patchy inflammation present in myocarditis, EMB also has limitations in terms of the risk of infection and sampling mistakes [74]. The Dallas criteria have been strengthened by the addition of an immune-histochemistry criterion [29]. According to this criterion, myocarditis is indicated by the presence of leukocytes (14/mm2), monocytes (4/mm2), and CD3+cells (7/mm2) as well as histological evidence of non-ischemic necrosis [29]. By using these criteria, cMRI may be more sensitive in detecting COVID-19 myocarditis. Due to the infection risk, EMB is probably avoided on COVID-19 patients, and the availability of scans like cMRI has been drastically decreased during the COVID-19 pandemic [70, 84]. Therefore, if hospitals are under pressure from COVID-19, doctors may need to employ a mix of blood tests, ECGs, echocardiograms, and a high clinical suspicion for myocarditis to make a diagnosis.

Taking care of COVID-19 myocarditis

Myocardial inflammation and any potential consequences must be managed as part of the myocarditis treatment. The effectiveness of intravenous immunoglobulins (IVIG) in the treatment of viral myocarditis has been investigated. Immunoglobulins IgG, IgA, and IgM exhibit anti-inflammatory properties while also neutralising and aiding in the removal of pathogens from the myocardium [85].A decrease in inflammatory and viral levels was one of the positive effects of immunoglobulin therapy for biopsy-proven CMV myocarditis, according to Maisch et al. [86]. However, the use of immunoglobin treatment produced mixed results in cases of suspected myocarditis without biopsy evidence of viral infection [86]. Hu et al. successfully treated COVID19 myocarditis with glucocorticoids and immunoglobulin therapy [87].

The evidence is not as strong in favour of using corticosteroids to treat COVID-19 myocarditis. In the absence of viral replication, the corticosteroid prednisolone may be effective in treating viral myocarditis [89]. When viral replication is evident, it is considered that using immunosuppressive drugs like corticosteroids may make acute myocarditis worse [90]. When corticosteroids were used, individuals with COVID-19 myocarditis had better outcomes, according to a systematic analysis by Sawalhaet al. [76].

This comes with the warning that the evidence is unreliable because the review only included 14 case reports. The use of corticosteroid therapy, on the other

hand, does not appear to lower mortality in patients with viral myocarditis, according to other research [91].An anti-IL-6 receptor monoclonal antibody called tocilizumab was tested in conjunction with the antiviral drug favipiravir to treat COVID-19 patients who had experienced cytokine storm [92]. In a trial, Tocilizumab and favipiravir together dramatically decreased inflammation brought on by cytokine storm [92]. The use of this combination therapy may be beneficial because COVID-19 myocarditis may be made worse by cytokine storm [28].

More research is required to evaluate the effects on COVID-19 myocarditis particularly, despite evidence demonstrating the effectiveness of IVIG in the treatment of viral myocarditis. The treatment of COVID-19 now involves the corticosteroid dexamethasone. The effectiveness of Dexamethasone may therefore be evaluated in order to determine whether current therapy is sufficient or whether individuals who develop COVID-19 myocarditis require additional antiviral/anti-inflammatory care.Further treatment is required for patients who develop cardiogenic shock as a result of fulminant myocarditis. Inotropic drugs, such dobutamine, and mechanical support, like intra-aortic balloon pumps or Impella systems, can be utilized to maintain blood pressure in patients with cardiogenic shock [74, 77].

Tachyarrhythmias can be treated with intravenous amiodarone hydrochloride, or pacing or direct current cardioversion may be utilized if the patient is unstable or unresponsive to pharmaceutical intervention [93]. When bradyarrhythmias do occur, they can be treated with intravenous atropine or, if necessary, transcutaneous pacing [93].

Conclusions

A serious side effect of SARS-CoV-2 infection that might make patients' prognoses worse is COVID-19 myocarditis. While some cases might be unimportant or symptomless, it's possible that clinicians will run upon cases that are more serious and need quick treatment. Therefore, understanding how to detect and treat this illness is crucial. Serial troponins and ECGs should be performed whenever possible to check for the emergence of myocarditis or other myocardial injuries. Myocarditis can have vague symptoms that can be confused with COVID-19's respiratory symptoms, making a diagnosis challenging. Given that first tests are generally not very expensive, it is critical to have a low threshold for working up a patient. Patients who show signs of myocardial injury after first evaluation should have further testing, such as echocardiography, cMRIs, and EMB. Clinicians should be on the lookout for any heart failure or arrhythmia symptoms since these may be the fatal symptoms of fulminant myocarditis. Although no effective treatment for COVID-19 myocarditis has been reported, IVIG and corticosteroids together have the potential to lower mortality, especially in cases of fulminant myocarditis.

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