EJCRIM
European Journal
of Case Reports
in Internal Medicine

A CASE OF SEVERE CARDIOMYOPATHY DUE TO COVID-INDUCED MYOCARDITIS, COMPLETELY RESOLVED AFTER COLCHICINE AND IMMUNOGLOBULIN THERAPY

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Received: 02/07/2023 **Accepted:** 10/07/2023 **Published:** 02/08/2023

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Informed consent was obtained from the patient.

How to cite this article: Khine S, Edupuganti S, Bachuwa G. A case of severe cardiomyopathy due to COVID-induced myocarditis, completely resolved after colchicine and immunoglobulin therapy. *EJCRIM* 2023;**10**:doi:10.12890/2023_003877.

ABSTRACT

Coronavirus 19 (COVID-19) is well known for causing acute respiratory distress syndrome. Among other systemic complications, myocarditis is a frequently reported presentation as well as complication. One systematic review reported a 14% mortality rate in patients with COVID-19 myocarditis. Endomyocardial biopsy is a definitive diagnostic test but has been a challenge to perform in most cases of COVID myocarditis due to the contagious nature of the disease. Patients presenting with new cardiomyopathy with troponin leak and arrhythmias, supported by recent COVID-19 diagnosis should be suspected for COVID-induced myocarditis. Supportive treatment has been the mainstay of treatment with limited data on immunotherapy and colchicine. Our case is about a male in his 50s who had a cardiac arrest due to ventricular fibrillations, with a positive COVID-19 test. Further workup showed severe non-ischaemic cardiomyopathy with an EF of 15–20%. He was treated with intravenous immunotherapy and colchicine. A repeat echocardiogram 3 days later showed resolution of cardiomyopathy. Our case report highlights the possible beneficial effects of immunotherapy and colchicine in viral myocarditis.

KEYWORDS

COVID-19 myocarditis, non-ischaemic cardiomyopathy, ventricular tachycardia, colchicine, immunotherapy

LEARNING POINTS

- Myocarditis should be suspected in patients with acute onset cardiomyopathy with troponin leak and no evidence of ischaemia. COVID-19 myocarditis can present with arrhythmia, which could be fatal in some cases.
- Even though supportive management is the mainstay of treatment for COVID-19 myocarditis, there have been reports of benefits of intravenous immunotherapy (IVIG) and colchicine.
- More studies are warranted to explore the beneficial effects of IVIG and colchicine not just in COVID-19 myocarditis, but also in other viral causes of myocarditis he aim of this study is also to raise awareness among healthcare professionals about the Bentall procedure in patients with type A aortic dissection involving the aortic valve.

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European Federation of Internal Medicine

Publisher
smc media srl

INTRODUCTION

Coronavirus 19 (COVID-19) is an infectious disease caused by SARS-CoV-2 virus with over 500 million confirmed cases since the start of the pandemic in 2019, and is responsible for more than 6 million deaths globally[1]. Systemic complications that are frequently documented included acute respiratory failure, myocardial injury, cardiac arrhythmias, heart failure, cardiogenic shock, venous thromboembolism, pulmonary embolism, acute tubular necrosis and acute kidney injury[2]. Multiple case reports have related SARS-CoV-2 infection with myocarditis. Myocarditis is a clinically challenging disease with variable prognosis depending on severity. One systematic review reported a high mortality rate of 14% specifically for patients with COVID-19 myocarditis[2]. The supportive management remains the mainstay of treatment. Antiviral therapy and intravenous immunoglobulin has been suggested but there is little data to support the therapy[3]. We reported a case of COVID-19 myocarditis who presented with the ventricular tachycardia storm and was successfully treated with colchicine and immunoglobulin therapy.

CASE DESCRIPTION

An African-American male in his 50s presented to the Emergency Department after receiving a total of seven shocks from an automatic implantable cardioverter defibrillator (AICD) device. His medical history included hypertension, hyperlipidemia and cardiac arrest, due to ventricular fibrillation status post AICD placement. He had rhinorrhoea, a cough, a sore throat and a low-grade fever over the previous 2 days. He denied any chest pain or shortness of breath. On initial evaluation, he was alert and oriented with no acute distress. He was immediately started on an amiodarone infusion. An electrocardiogram (EKG) was unremarkable with no acute ST changes, and with a corrected QT interval (QTc) of 440. Troponin was 0.373 ng/ml. Electrolytes were unremarkable. Later, the patient decompensated and went into pulseless ventricular tachycardia with a heart rate of 230 beats per minute. Cardiopulmonary resuscitation (CPR) was started. Return of spontaneous circulation was achieved after a few rounds of CPR and external defibrillation; he was intubated and admitted to the intensive care unit. The patient came back positive for SARS-CoV-2. He was started on dexamethasone and remdesivir for the SARS-CoV-2 infection. Troponin jumped up to 12.373 ng/ml and peaked at 148 ng/ml. Procalcitonin was 48.85 µg/L. An echocardiogram showed an ejection fraction (EF) of 15–20% with grade 2 diastolic dysfunction, global hypokinesis and significant wall abnormality. He underwent cardiac catheterisation on day 2, which revealed mild coronary artery disease with severe cardiomyopathy. His most recent echocardiogram two years prior showed an EF of 35–40%. He was presumed to have severe non-ischaemic cardiomyopathy likely due to acute myocarditis from the SARS-CoV-2 infection. On days 3 and 4, the patient continued to have multiple episodes of ventricular tachycardia with a heart rate in the 150s despite being on oral amiodarone, beta blocker and mexiletine. Multidisciplinary discussion was held among a cardiologist, an electrophysiologist and an intensivist, and the patient was started on colchicine and intravenous immunoglobulin therapy for severe COVID-induced cardiomyopathy with ventricular tachycardias. On day 7, episodes of ventricular tachycardia were decreasing. An echocardiogram was repeated and showed improved EF of 50–55% with resolution of cardiomyopathy. Immunoglobulin therapy and colchicine was discontinued. The patient was extubated a few days later and underwent inpatient rehabilitation, and was subsequently discharged after completion of rehabilitation.

DISCUSSION

Since the beginning of the pandemic in 2019, there have been several studies reporting myocardial damage and myocarditis as a complication of severe COVID-19. COVID-19 myocarditis – similar to other viral myocarditis – presents with fever, cough, chest pain, dyspnoea, syncope and palpitations. Other supportive evidence included finding arrhythmia on EKG, cardiac dysfunction on an echocardiogram or cardiac magnetic resonance imaging indicating myocarditis. Ideally, an endomyocardial biopsy should be performed for a definitive diagnosis of myocarditis. However, in cases of COVID-19 myocarditis, biopsy is challenging due to the contagious nature of the infection, the severity of cases and having no influence on treatment decisions[4-5]. Our patient also did not undergo biopsy due to limited resources and his instability. However, several factors were supportive of myocarditis. Firstly, he presented with several episodes of ventricular tachycardia, which subsequently led to cardiac arrest in the setting of normal electrolytes. Secondly, his echocardiogram showed a very low ejection fraction with global hypokinesis, which is one of the features seen in myocarditis cases. In addition, his cardiac catheterisation was negative for any coronary artery lesions and thus ruled out the ischaemic nature of cardiomyopathy. Thirdly, he was noted to have a mild troponin leak of 0.373 ng/mL even before his cardiac arrest. His troponin later spiked at 148 ng/ml which might partially be attributed to his post cardiac arrest state. These findings in the setting of SARS-CoV-2 infection were supportive of COVID-19 myocarditis.

There is no definite guideline for management of myocarditis other than supportive management. There are other therapies such as colchicine, intravenous immunotherapy (IVIg), and corticosteroids that are being used, but these are not widely supported due to limited evidence[6]. Colchicine is an anti-inflammatory agent with the ability to suppress inflammatory cytokines, and NOD-, LRR- and pyrin domain-containing protein 3 (NLRP 3) inflammasome. One case report demonstrated the resolution of symptoms within 48 hours after administration of colchicine in a case of suspected COVID-19 myocarditis[7]. Moreover, a clinical trial has shown the possible beneficial effects of colchicine in treating patients with COVID-19 infection and in reducing the duration of hospitalisation[8]. Colchicine has also been shown

to be beneficial in the treatment of acute myocarditis caused by Epstein-Barr and cytomegalovirus[9]. On the other hand, immunoglobulin therapy has been reported to be beneficial, according to some of the studies. A meta-analysis of 13 studies showed significantly reduced in-hospital mortality and improvement in left ventricular dysfunction in acute myocarditis cases treated with IVIg therapy[10]. However, the data are still insufficient to recommend routine use of either colchicine or IVIg[6]. In our case, the decision to start colchicine and IVIg was made due to the severe nature of the patient's presentation and recurrent ventricular tachycardia despite treatment with anti-arrhythmic agents. Our patient improved rapidly after 2 days of therapy as evident on the echocardiogram, and decreasing episodes of ventricular tachycardia.

Myocarditis is still a clinical challenge due to delay in diagnosis, the wide range of presentations and being a renowned cause of sudden cardiac death. Major clinical studies should be encouraged to explore the beneficial effects of colchicine and immunoglobulin therapy in myocarditis.

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