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Case Report

Ventricular fibrillation storm in COVID 19 responding to steroid therapy: A case report

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Abstract

Cardiac arrhythmias are common in patients of COVID -19 and frequently complicate the clinical course of critically ill patients. Life threatening arrhythmia including ventricular fibrillation less common but are reported to be more common in patients with elevated cardiac troponins. The mechanisms of arrhythmia in COVID 19 are multifactorial and arise from either direct cardiac involvement, from consequences systemic affection or drug interactions. The successful management requires correct identification of the cause. We report a case of VF storm in a patient with COVID 19 who responded to steroid therapy. Controlling the fulminant inflammation may reduce the burden of arrhythmia in appropriate cases.

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Learning objective

- Arrhythmia cause a significant burden of disease in COVID-19 patients.
- Life threatening arrhythmia are more common in critically ill COVID-19 patients.
- The pathophysiology and etiology of arrhythmia in COVID 19 is multifactorial.
- Correct identification of cause is essential for proper management.
- In selected cases reducing the inflammatory status may help reduce the arrhythmia.

Introduction

The cardiac complications of coronavirus disease 2019 (COVID-19) in patients with and without prior cardiovascular disease are well known which include myocarditis, heart failure, and acute coronary syndromes resulting from coronary artery thrombosis or SARS-CoV-2-related plaque ruptures. The viral infection either causes direct damage to the cardiomyocytes or damage by adverse systemic inflammation which leads to myocarditis and is purported as one of the major cause of COVID 19 related cardiovascular complications. The COVID-19 also causes significant burden of arrhythmia in affected patients and they are more common in critically ill COVID -19 patients.1 Atrial fibrillation was the most common cardiac arrhythmia observed in patients with COVID-19 and Ventricular arrhythmia are also reported and malignant arrhythmias, including ventricular tachycardia/ventricular fibrillation, are more common in patients with elevated troponin T levels.2 Elderly who are likely to have various comorbidities such as diabetes, hypertension and obesity comprise the majority with cardiac involvement during severe acute respiratory syndrome coronavirus (SARS-CoV-2) infection.

There could be multitude of factors and causes leading to arrhythmias in COVID-19 patients due to pathologic and metabolic consequences of the disease like hypoxia caused by direct damage of lungs, myocarditis, adverse immune response, myocardial ischemia or strain, electrolyte abnormalities, intravascular volume imbalances and effects of medication used either for prophylaxis or treatment of COVID 19.1 Correct identification of the underlying culprit is crucial for correct management. Herein, we describe the
course of a COVID-19 patient who experienced an electrical storm of ventricular fibrillation (VF) due to myocardial inflammation and responded very well to steroid therapy.

Case report

A 65 year old male hypertensive gentleman was admitted to the emergency department of our hospital with complaints of multiple episodes of sudden onset palpitation followed by unconsciousness from last 2 days. There was no history of chest pain, shortness of breath, headache, vomiting and fever. The medication history did not reveal any history of treatment or prophylactic administration of Hydroxychloroquine or Azithromycin during pandemic time. His physical examination revealed blood pressure of 120/60 Hg, pulse rate of 58 per minute, oxygen saturation of 98% while breathing ambient air and body temperature of 98° Fahrenheit. Systemic examination including cardiovascular and respiratory system did not reveal any abnormalities. His 12 lead ECG revealed sinus rhythm with frequent ventricular ectopics with early coupling (originating from purkinje fibres) with normal QT interval (Fig. 1(a)). His bedside Echocardiography revealed mild concentric left ventricular (LV) hypertrophy with mild LV Systolic dysfunction(LVEF = 50%). The serum electrolytes were normal but the troponin I was elevated. Additionally the serum C-Reactive Protein(44.60 mg/L) and D-dimer values(3172.51 ng/ml) were significantly raised. Other acute phase reactants were also elevated from baseline including serum LDH and serum ferritin. (Suppl. Table 1).

In the emergency room patient suddenly became pulseless and unresponsive which reverted back spontaneously. The patient was shifted to Intensive Care Unit where patient again became unresponsive. The Cardiac Monitor revealed Torsades de pointes (Fig. 1(b)) but again it spontaneously reverted to sinus rhythm. Lignocane infusion and amiodarone infusion were started. After half an hour the patient again developed VF which was reverted to sinus rhythm with 150 J direct current (DC) shock. The patient was immediately shifted to catheterization laboratory where Coronary angiography was done, which revealed non obstructive coronary artery disease. Patient had multiple VF episodes in next 24 h for which five times Cardiac defibrillatory shock was delivered. Repeat troponin samples revealed the rising titre of same. At that point, the family signed comfort care only. At this point of time his COVID-19 RT-PCR was found to be Positive. Treatment was started as per ICMR protocol and all the necessary investigations for COVID-19 were sent. He was put on intravenous dexamethasone considering a possibility of COVID 19 related myocarditis. There were no VF episodes after that. Patient was discharged on sixth day with advice for home isolation.

Fig. 1. (a) The baseline electrocardiogram of patient showing frequent early coupled ventricular ectopics (b) The monitor captured images of torsades-de-pontes and fibrillation.
Discussion

Electrical storm is usually defined as three or more separate episodes of ventricular tachycardia or ventricular fibrillation within 24 hours and it is often life threatening and has been associated with poor prognosis. The underlying etiology varies and it is important to consider triggers like electrolyte disturbances, proarrhythmic drugs, acute coronary syndrome, and structural heart disease and at times endomyocardial biopsy may be needed for diagnosing myocarditis.\(^3,4\)

The hypothesis regarding our patient’s VF storm etiology was acute myocarditis/myocardial injury in the setting of hyper-inflammation (as evidenced by elevated markers of inflammation, Suppl. Table 1), was supported by elevated cardiac troponins, global hypokinesia of left ventricle with mild LV dysfunction, absence of other factors like hypokalemia, hypomagnesemia, bradycardia and long QTc and excellent response to steroids. The pathogenesis of arrhythmia can be multifactorial in COVID-19 (Fig. 2) and the management depends upon the identification of possible etiology.\(^1\) 

In a study of 138 patients with COVID-19, it was noted that 11.8% of
the patients who died had substantial heart damage without an underlying cardiovascular disease, which suggests that the incidence of cardiovascular complication is relevant. Perhaps the cytokine release syndrome, a hypothesis related to the severity of inflammation in the COVID-19 that generates cardiac involvement, encouraged us to use systemic steroids to counteract it. In our case, there were no respiratory symptoms or gastro-intestinal problems, nor was there a history of previous myocardial infarction, and from the beginning the clinical picture was manifest with ventricular arrhythmia. This case report suggests that reducing the inflammatory state in COVID-19 patients can help in managing ventricular arrhythmia. Systemic inflammation predisposes for ventricular arrhythmia indirectly by inducing a hyper-sympathetic state or inhibiting cytochrome p450. Thus, reducing this inflammatory state in COVID-19 patients can mitigate arrhythmic events. However, in such cases treatment with intravenous magnesium and intravenous beta blockers can also be tried considering the fact that magnesium works as membrane stabiliser and is effective in TdP where QT is not prolonged and beta blockers would help curb the adverse effects of high sympathetic state resulting from cytokine storm. We also acknowledge that cardiac magnetic resonance imaging (MRI) would have been an ideal test to arrive at a conclusive diagnosis of myocardial involvement. Cardiac troponin elevation may not be confirmatory as it can rise due to other reasons including systemic inflammation and multiple DC shocks. However, We ruled out other causes of VF storm in the patient (hypoxia, durgs, QT prolongation, ischemia, fluid disturbance etc) and that also leaves us with a support to our diagnosis of myocardial injury/myocarditis as a possible mechanism of VF in presence of circumstantial evidence including raised troponins and markers of systemic inflammation, LV dysfunction with global hypokinesia dramatic response to steroids, and absence of other cause for VT storm.

Our case report suggests that the hyper-inflammatory state with myocardial injury in COVID-19 patients can induce ventricular arrhythmias, which has been reported to cease abruptly following a reduction in inflammation. Hence, checking this fulminant inflammatory condition may prevent or decrease cardiac arrhythmias.

Consent

The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Contributorship

All authors were involved in patient care, drafting and editing of the manuscript.

Data availability statement

The data underlying this article are available in the article and in its online supplementary material.

Declaration of competing interest

None, no relationship to industry.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ihjccr.2021.07.007.

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