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Case Report

Acute Cardiac Failure Complicating a Case of SARS-COVID 19 in a 24-Year-Old Patient

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Abstract

Cardiac complications of SARS-COVID 19 disease have been under-appreciated. We present a case of a young male who had presented with SARS COVID 19 and acute cardiac failure. The reasons for cardiac failure associated with SARS-COVID 19 could be multiple. Identification of the cause of cardiac decompensation through clinical judgment and echocardiographic findings hold the key to successful management in these cases.

Keywords: COVID 19; Acute cardiac failure; Myocarditis; Blood

Abbreviations

COVID 19: Corona Virus Disease 2019; SARS: Severe Acute Respiratory Syndrome; RT: Reverse Transcriptase; PCR: Polymerase Chain Reaction; ECHO: Echocardiogram; BIPAP: BiLevel Positive Airway Pressure; PASP: Pulmonary Artery Systolic Pressure; RAP: Right Atrial Pressure; BNP: Brain Natriuretic Peptide

Introduction

The corona virus pandemic has been declared as a world-wide public health emergency [1]. The majority of the deaths associated with COVID 19 have been attributed to acute respiratory distress syndrome & multi-organ failure [2]. Reports of toxic myocarditis associated with SARS-COV-2 have been few and far in between [3]. However, they have been associated with arrhythmias and in some cases progression into fulminant cardiac failure3. Here we present such a case in which a 24-year-old patient with COVID 19 presented with fulminant cardiac failure.

Case Presentation

A 24-year-old male patient was referred to our hospital during the COVID pandemic from a local hospital. He had a 7-day history of fever and cough and was diagnosed to have COVID 19 using RT PCR. Therapy given in the local hospital included remdesevir, dexamethasone, low molecular weight heparin, empirical antibiotics, and non-invasive ventilation using BiPAP. As the patient's condition deteriorated on the second day, he was referred to us for intensive care.

On admission in our ICU, he was found to be febrile, drowsy, tachypnoeic, saturating at 82% on NIV with a FiO_2 of 1.0 and hypotensive with cold extremities not responding to fluid boluses. It was decided to electively intubate him and initiate invasive ventilation. Modified rapid sequence intubation was performed and controlled ventilation was initiated. Central venous access and arterial line were secured to aid hemodynamic management. Vasopressor support was initiated using noradrenaline which was quickly escalated to include vasopressin and adrenaline because of refractory hypotension. High FiO_2 (1.0) and PEEP were applied to maintain a PaO_2>60 mmHg.

Electrocardiogram done at this point revealed sinus tachycardia with no ST-T abnormalities. Bedside echocardiography revealed a dilated left ventricle (60 mm), ejection fraction of 28%, global left ventricular hypokinesia, PASP of 53 mm HG, RAP of 12 mm HG, moderate TR with impaired right ventricular function. Hematological values of myocardial injury like troponin I & NT-pro-BNP were also elevated (Table 1). Routine blood investigations were sent which demonstrated an elevated neutrophil/lymphocyte ratio with multiorgan failure. Other markers of inflammation like ferritin, IL-6, LDH, and procalcitonin were also elevated.

His CXR revealed extensive bilateral infiltrates with some degree of fluid overload. After sending blood, urine, and endotracheal cultures broad-spectrum antibiotics in the form of meropenem and teicoplanin were started. Other supportive measures in the form of steroids (dexamethasone 4 mg BD), low molecular weight heparin 40 mg BD, vitamin C, and zinc were continued. Remdesevir was discontinued keeping in mind his worsening kidney functions. A restrictive fluid therapy using albumin and furosemide infusions targeting a negative fluid balance was employed. However optimal intravascular volume was ensured by point of care measurement of caval diameter using ultrasound to ensure renal perfusion.

Keeping in mind his rapidly deteriorating clinical condition associated with multiorgan failure, he was enrolled in our institutional plasma trial and was administered 2 units of convalescent plasma 24 hours apart. Signs of clinical recovery were noticed after 24 hours in the form of diminishing inotropic requirements, settling down of tachycardia, clearing of chest x-ray, and improving renal and hepatic functions. Antibiotics were downgraded as his cultures were sterile. As his condition steadily improved, we stopped sedation and extubated him on to a facemask on the 5th day of admission. He was started on oral feeds and other supportive measures like incentive spirometry, chest physiotherapy, and ambulation. He was shifted out of the ICU

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Table 1: Laboratory investigations.

	Day of admission	Day 2	Day 5	Day 7
Haemoglobin (gm/dl)	14		12.8	
WBC count (cells/ mm^3)	46000	32340	24000	12100
N/L ratio	47.6		32	
Total bilirubin(mg/dl)	9.4		5.6	2.4
Albumin(gm/dl)	2.8		3.3	3.1
SGOT(U/L)	82		45	44
SGPT(U/L)	78		43	41
Urea(mg/dl)	78		67	45
Creatinine(mg/dl)	1.8	1.6	1.4	1.1
INR	1.9			
D Dimer(ng/ml)	5209			
Fibrinogen(mg%)	646			
Procalcitonin(ng/ml)	20		8.5	
Ferritin(ng/ml)	1746			
LDH(U/L)	408			
IL 6(pg/ml)	298		100	
NT-proBNP(pg/ml)	12800			
Trop i(ng/ml)	0.572 (<.045)			

on day 7 by which time he was off oxygen therapy. An ECHO repeated the next day revealed a good biventricular function with no regional motion abnormalities. We were able to successfully discharge him on the 10^{th} day of admission.

Discussion

Myocardial disease associated with COVID 19 has been described even during the initial stages of the pandemic in china [4]. Sudden cardiac failure in COVID 19 can be attributed to 3 main causes namely acute coronary syndrome, viral myocarditis, and stress-related cardiomyopathy. Clinical features of these 3 syndromes frequently overlap and extensive evaluation might be needed to distinguish between them. Our patient was young, with no known comorbidities. The regional wall motion abnormalities in our case were global, rather than segmental which makes us believe that the cause of hemodynamic instability was either viral myocarditis or stress-related cardiomyopathy. The only way these 2 conditions could have been definitively distinguished was an endomyocardial biopsy [5] and identification of viral genetic material in the specimen. We didn't perform endomyocardial biopsy since the patient was showing rapid clinical involvement and also keeping in mind the risk of contagion spread.

Management of a patient with SARS COVID 19 presenting with acute cardiac failure should revolve around the etiology of cardiac failure. An acute coronary syndrome should be suspected in cases with known cardiac risk factors, ECG changes, and segmental wall abnormalities in ECHO. Revascularisation or thrombolysis can be planned depending upon the clinical condition of the patient. Management of stress cardiomyopathy and viral myocarditis is supportive which includes hemodynamic support either with inotropes or mechanical circulatory devices, tight control of intravascular volume, management of arrhythmias, and broad-spectrum antibiotics to control sepsis.

Steroids and immunomodulators like toclizumab should be used with caution in these cases especially since they might be associated with severe bacterial infections. In our case we used 2 doses of convalescent plasma early in the course of the disease. We believe the antibodies in convalescent plasma might have been beneficial in managing cytokine storm in our patients. Even though the patient had high levels of IL 6, toclizumab was withheld owing to the elevated procalcitonin levels. To conclude, cardiac involvement in COVID 19 might be more common than previously thought and in some cases can lead to fulminant cardiac failure. The performance of certain diagnostic procedures like coronary angiogram or endomyocardial biopsy might be impeded owing to the fear of the spread of the contagion. Hence,a clear clinical judgement must be made based on the available history, risk factors, markers of inflammation, ECHO findings, and response to therapy.

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