Case Report

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Unusual Presentation of a Covid Patient with MI with Survival after Cardiac Arrest

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Abstract

The novel coronavirus disease (COVID-19) has been declared as a pandemic. With the increasing number of COVID-19 patients, it is necessary to rangeover the factors related to the disease to aid patient management until a definitive vaccine is prepared, As the disease is not limited to the respiratory system alone. COVID-19 has been associated with various cardiac, vascular complications including acute myocardial injury, arrhythmias, myocarditis and venous thromboembolism.

It can expand thrombotic complications that ultimately lead to myocardial infarction even in patients without having a history of underlying heart disease.

Keywords : Pandemic, Covid 19, Myocardial infarction, Cardiac arrest.

Introduction

The "2019 coronavirus (2019-nCoV)," known as the COVID-19, first appeared in Wuhan, is an infectious disease that converted into a pandemic. The characteristics features are fever, cough, and pneumonia, accompanied by severe respiratory syndrome. The most considerable group of patients are either asymptomatic or minimally symptomatic.

Nevertheless, censorious manifestations, including ARDS, vascular events -thrombosis and encephalitis, can be observed. The most common clinical presentation is a respiratory disease as compare to cardiovascular manifestations. About 20%-30% of patients with COVID-19 suffer from cardiovascular involvement. The predominant type of cardiac involvement in COVID-19 is acute heart injury, with a marked rise in cardiac biomarkers. It is postulated that cardiac manifestations, particularly cardiac dysfunction in COVID-19 cases with lung involvement, can be due to viral myocarditis, which mimics Myocardial infarction in clinical presentation and in laboratory data.

Nevertheless, thrombotic events should be kept in mind as the main cause of MI which may mimic

myocarditis in the COVID-19 cases. In this case report, we aim to present a case of COVID-19 that results in myocardial infarction.

Case Report

A 65-year-old male patient with a history of type 2 diabetes mellitus came to ED, presented with complaints of dyspnea on exertion since last 2 days, worsened since the morning (admission day, November 2020) associated with chest pain & diaphoresis. On admission, the patient's arterial oxygen saturation (SpO2) was 80% on room air, 92% on 10 L Oxygen, heart rate of 140/min, blood pressure of 130/90 mmHg. 12 lead ECG was done which showed ST Elevation in lead V2-V4.

His COVID Rapid Antigen test was done at the time of admission which comes out to be positive. Prior to his transfer to Cath lab, patient became unconscious, cardiac monitor showed Ventricular fibrillation followed by asystole, CPR was started as per ACLS protocol, ROSC was achieved. Patient was intubated & was shifted to Cath lab. Angiography findings showed a 100% thrombotic occlusion in proximal LAD (Left anterior descending artery) with a 80% occlusion in distal LCX (Left circumflex artery), Lesion crossed with PTCA wire, thrombo-suction was done & stent was placed. Laboratory investigations of the patient revealed elevated D-dimer; inflammatory markers including the ESR, ferritin, Interleukin, procalcitonin and C-reactive protein (CRP) were raised due to the hypercoagulable state secondary to COVID-19.

Chest X Ray showed non homogenous & ground glass opacities in bilateral mid & lower zones. HRCT chest showed areas of consolidation in both middle & lower lobes (Corads-6, CT severity scoring, 20/40), B/L pleural effusion with associated interlobular septal thickening in both lower lobes-h/o recent cardiac arrest noted. He was started on dual antiplatelet therapy consisting of clopidogrel (75 mg) and aspirin (75 mg) Remdesivir, Toclizumab as well as high dose of antibiotics, Steroid therapy (methylprednisolone 40 mg, intravenous injection), 2 Units of Plasma Transfusion given to the patient. Despite of all this treatment, patient's general condition detoriates on day 7of hospitalization, Patient had developed sudden bradycardia followed by asystole, CPR was again started as per ACLS protocol, ROSC again achieved.ST elevations were noted on telemetry. The patient's electrocardiogram showed inferior lead ST-segment elevations. Her troponin I peaked at 5.5 ng/ml.

Again patient was underwent percutaneous intervention with a drug-eluting stent for the pathology with the thrombolysis. Repeat Complete Blood Count showed leukocytes 28,000/cumm (normal range 4000-11,000 cumm), S.Creatinine (6mg/dl), decreased urine output, hypotension, Antibiotics were modified along with vasopressors &inotropes support, 1 cycle of hemodialysis was done. Subsequently, Patient's Covid 19 report found to be Negative on 15th day &subsequently Lymphocyte counts showed decreasing trend. He had been intubated for approx. 20 days. Multiple Chest X-rays were obtained to evaluate the lung involvement, General condition of the patient improved day by day & patient was extubated. After 30 days of prolonged hospitalization, patient was discharged from the hospital.

On admission ECG showed ant wall MI, Chest Xray showed non homogenous & ground glass opacities in B/L mid & lower zones with endotracheal tube in situ. x-ray on Admission-showing B/L ground glass opacities, with endotracheal tube in situ. HRCT Chest showing B/L Consolidation.

Discussion

It is believed that myocardial injury in COVID-19 patients is produced by numerous mechanism is chemic injury, coronary thrombotic complications because of increased platelet activity, endothelial dysfunction, vessel stasis, hypoxia-induced injury, and cytokine storm. The thrombotic events can develop due to the primary effect of COVID-19 or the secondary consequences of the viral infection, including hypoxia and critical illness.

In this case, there was no prior history of underlying heart disease. Due to the severity of the symptoms and paraclinical data, emergent invasive angiography was performed. Catheterization findings showed the presence of fresh clot inside the coronary artery lumen, which results in obstruction of vessel. Preliminary documents indicated that affected patients may suffer from hemostatic impairments. In a study by Guo et al., COVID-19 patients with diabetes mellitus had higher mortality (10.81%) secondary to severe COVID-19. In another study, hypertension (98%) and diabetes (69%) were prevalent amongst some patients requiring mechanical ventilation.

By the end of the follow-up, (31%) patient had died within 6 days (median) after hospitalization. All these risk factors are affiliated with severe COVID-19 infection and high mortality. In our

case, the patient had an extended hospital stay due to a severe COVID-19 infection with complications due to several of the comorbidities, which are underlying risk factor; however, he survived.

Conclusion

In the population having some prior comorbidities such as Diabetes or hypertension, In addition to Respiratory system, COVID-19 can affect other vital system also via different mechanisms like in this case, Covid 19 affects cardiovascular system, mechanism although not clearly understood, it can increase thrombotic complications because of endothelial dysfunction, increased platelet activity, and vessel stasis that ultimately lead to Myocardial Infarction & Cardiac Arrest.

Conflict of Interest: None declared

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