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CASE REPORT

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COVID-19 with non-obstructive coronary artery disease in a young adult

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ABSTRACT

Systemic inflammatory response due to cytokine storm in severe COVID-19 cases can lead to acute myocardial infarction, also affecting the younger population, without significant risk factors. We present the case of a 36-year-old male with morbid obesity and well-controlled asthma who had developed COVID-19-induced acute respiratory distress syndrome requiring mechanical ventilation and, subsequently, extracorporeal membrane oxygenation (ECMO) who developed myocardial infarction on Day 10 of admission and died on Day 15 of admission due to sequelae of severe COVID-19 infection. In young patients with COVID-19-induced respiratory infection, severe inflammatory response can lead to acute coronary syndrome in absence of obstructive lesions or plaque ruptures.

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STEMI; COVID-19; complications; young

1. Introduction

The novel coronavirus disease termed as COVID-19 by the World Health Organization (WHO) was declared a pandemic on 11 March 2020. Since then, it has affected more than 7 million people worldwide [1]. Cardiovascular disease had been established as a risk factor for inpatient hospitalizations and mortality even in non-pandemic, pre-COVID era because of varied immune responses to infection and increased metabolic demands secondary to viral sepsis [2]. Although myocardial infarction has become a recognized complication given the systemic inflammatory response generated by severe COVID-19, it is a rare presentation in relatively younger individuals. Here, we are reporting a case of a 36-year-old male without significant cardiovascular risk factors except morbid obesity who was COVID-19 positive and admitted with severe acute respiratory distress syndrome (ARDS), requiring extracorporeal membrane oxygenation (ECMO), who subsequently developed new-onset ST-elevation myocardial infarction during his hospitalization and died.

2. Case presentation

A 36-year-old male, with a history of morbid obesity with body mass index of 46.5 kg/m^2 and well-controlled asthma, presented to outpatient clinic initially with shortness of breath, cough, myalgias and significant contact history with COVID-19 positive family members and was tested positive for infection and was discharged home with instructions of self-isolation. However, the patient presented to the emergency department two days later with worsening respiratory status with oxygen saturation in the 60s on room air and increased work of breathing requiring intubation and mechanical ventilation on. On examination, he had bilateral coarse breath sounds on auscultation. Laboratory workup on presentation was significant for thrombocytopenia of 147,000, elevated C reactive protein levels of 21 mg/dl, and high D-dimer levels of 2199 ng/ml and a chest x-ray consistent with multifocal pneumonia and repeat nasopharyngeal polymerase chain reaction (PCR) of COVID-19 positive (Figure 1). The patient had no leukocytosis, no lymphopenia, hemoglobin and hematocrit were within normal limits. Electrocardiogram (EKG) on presentation revealed sinus tachycardia without any ischemic changes (Figure 2). The patient was started on broad-spectrum antibiotics for community-acquired pneumonia. Uranalysis, urine cultures, sputum cultures and blood cultures were all negative. On Day 5, due to severe acute respiratory distress syndrome and worsening hypoxia not improving with proning, the patient was put on venous-venous extracorporeal membrane oxygenation (VVECMO). On Day 10 of admission, the ST elevations were observed on a cardiac monitor; EKG was obtained which showed ST elevations on anterolateral leads and troponin level of 0.24 (Figure 3). Transthoracic echocardiography (TTE) was attempted with no windows visible and emergent transesophageal echocardiography (TEE) was performed which showed preserved LV

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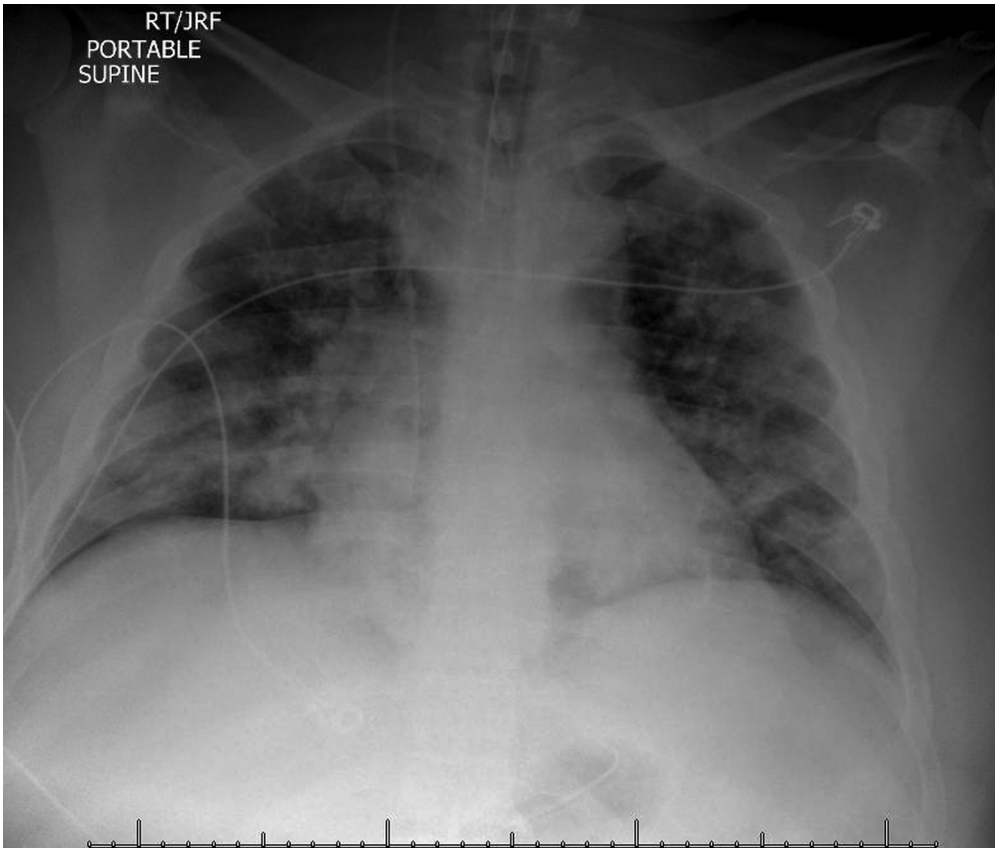

Figure 1. Chest x-ray showing multifocal pneumonia.

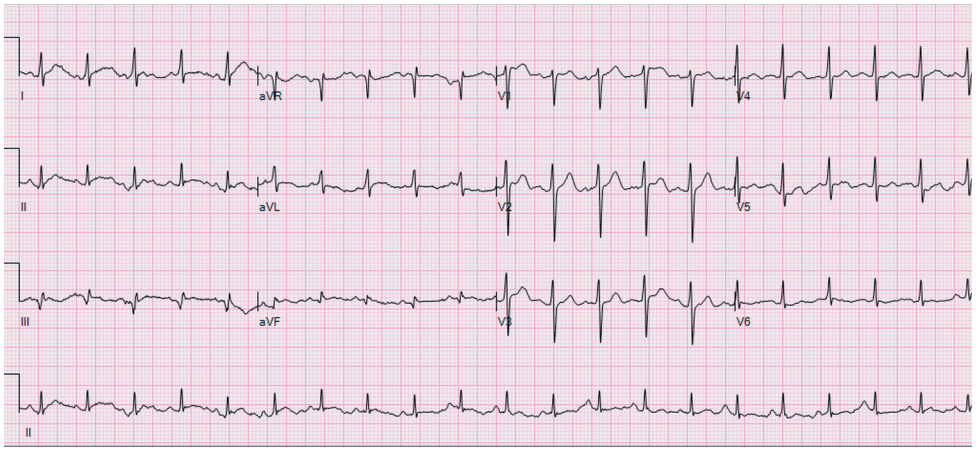

Figure 2. Electrocardiogram (EKG) on presentation showing sinus tachycardia without any ischemic changes.

systolic function, no pericarditis, no overt wall motion abnormalities. The study was limited due to body habitus.

The patient underwent emergent coronary angiography, left heart catheterization and left ventriculography which revealed no obstructive lesions, mean aortic pressure of 80 mmHg, left ventricular diastolic pressure of 20 mmHg, mildly hypokinetic anterolateral and apical walls of the left ventricle and mildly hyperkinetic inferobasal wall of left ventricle. No intervention was done. The patient was started on aspirin and high-dose statin. The patient subsequently developed worsening leukocytosis, lactic acidosis, and refractory hyperkalemia requiring continuous renal replacement therapy and maximum vasopressors support due to complication of ischemic bowel disease. The patient was transitioned to comfort care after palliative discussion with family and died on Day 15 of admission.

3. Discussion

COVID-19 is associated with multiple cardiopulmonary complications such as acute myocardial infarction, myocarditis, heart failure, arrhythmia, and

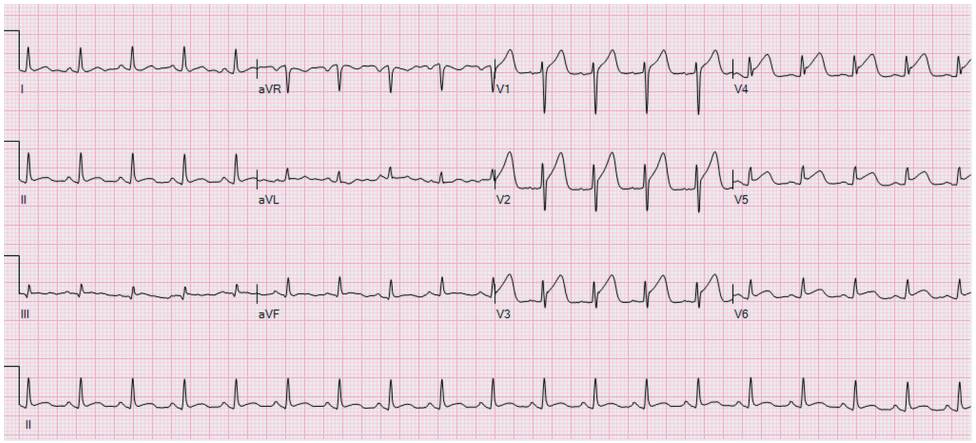

Figure 3. Electrocardiogram (EKG) revealing ST elevations on anterolateral leads.

venous thromboembolic disease, but these complications are relatively rare in younger patients [3].

The features of our case were, however, novel in the sense that our patient had an anterolateral STEMI with mildly elevated troponin levels (0.24) and no obstructive lesions which are pathognomonic of acute coronary syndrome. A recent case report described a 61-year-old female with inferior STEMI who had significantly elevated troponin levels and subtotal occlusion of right coronary artery [4]. Inciardi et al. reported another case with diffuse ST elevations in a 53-year-old male with biventricular and apical hypokinesis [5]. Our patient was relatively younger and lacked significant co-morbidities. In a review, obesity was a major risk factor for severe COVID-19 infection; 88.2% of non-survivors had a body mass index >25 kg/m^2 [6]. Thromboembolic risk is higher for obese patients and therefore, there is a greater chance of patients progressing to severe infection and for thromboembolic event.

The postulated mechanisms include direct vascular injury, hypoxia-induced injury, and viral invasion. In the setting of severe infection with ARDS, stress-induced adrenergic discharge, coronary spasm, endothelial dysfunction, and systemic inflammatory response due to cytokine storm in severe COVID-19 cases leading to atherosclerotic plaque rupture were the probable triggering factors leading to myocardial infarction [7]. In case of non-occlusive lesions, disruption of atheromatous plaque is toted as the cause of the infarction [8]. The criteria for diagnosing myocardial infarction with non-obstructive coronary arteries (MINOCA) included features consistent with an acute myocardial infarction (AMI), non-obstructive coronary arteries on angiography, and no clinically overt specific cause for the acute presentation; all these criteria were fulfilled by the patient [9].

In a case review of 28 patients with STEMI, the mean age of the participants was 68 ± 11 years; 89.33% were with localized ST elevations and 14.3% developed STEMI during hospitalization. All patients underwent urgent angiography and only 11 patients (39.3%) did not reveal obstructive coronary artery disease. It also reported mortality of 39.3% [10]. In a letter to the editor, Banglore et al. reported data of 18 patients with ST-segment elevation, 10 patients presented with ST-segment elevation and eight developed during the hospitalization. Of half the patients who underwent coronary angiography, two-thirds had obstructive disease. The mortality rate was high as 13 patients died during hospitalization (half of those with myocardial infarction (MI) and 90% of those with non-coronary myocardial injury) [11].

The mortality is very high in patients requiring ECMO for treatment of COVID-19 ARDS. Three studies reported mortality outcome of patients requiring ECMO. Out of 16 patients on ECMO, 15 died with 94% mortality [12–14]. Additionally, the in-hospital mortality is high for patients with sepsis in addition to acute myocardial infarction as compared to patients without infarction (48.3% vs. 17.7%) regardless of COVID-19 infection [15].

In COVID-19 patients with STEMI due to non-obstructive lesions, the mortality is reported to be very high. Obesity is also considered as a risk factor for severe COVID-19 infection with increased risk of thromboemolic events. However, ECMO itself is associated with a high mortality rate in COVID-19 patients based on the studies reviewed.

4. Conclusion

COVID-19-induced pneumonia has widespread complications targeting multiple systems, especially myocardial infarction. COVID-19 patients with acute STEMI and underlying non-obstructive lesions and patients requiring ECMO have very high inpatient mortality rate. There is an emerging need to streamline these presentations to aid the health care being provided to COVID-19 patients.

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Informed Consent

Informed consent for patient information and images to be published had been provided.

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