ORIGINAL RESEARCH PAPER

General Medicine

ACUTE CORONARY SYNDROME AND COVID 19 ARDS: A RARE CASE REPORT OF REFRACTORY HYPERCOAGULOPATHY.

KEY WORDS: Covid 19 ARDS, Hypercoagulable state, Acute Coronary Syndrome, Cytokine storm.

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ABSTRACT

Novel coronavirus disease 2019 (COVID-19) is known to cause severe bilateral pneumonia and acute respiratory distress syndrome (ARDS), it is also presenting as severe hypercoagulable state, leading to serious complications such as pulmonary embolism (PE),[4] cerebral infarction,[5,6] or venous thromboembolism (VTE).[7]Less common manifestations include renal artery thrombosis,[8] acute myocardial injury,[9] and mesenteric ischemia.[10,11]

Here we present a case of 40 year old non diabetic patient came with chief complaints of fever,dry cough and generalized bodyache which was diagnosed as having covid 19 pneumonia by RTPCR testing the next day. Patient had recurrent fever spikes, desaturation, tachycardia, Raised HbA1c on the third day. He was started on T. Favipinavir, followed by Inj.Remdesivir. Patient had worsening symptoms and landed up in cytokine storm on the 7th day. After giving 2 injections of Tocilizumab and 2 Convalescent plasma transfusions patient improved but had complications of fungal infections, post covid coagulopathy avascular necrosis of Femur Head and new onset diabetes mellitus. After being asymptomatic for 1 year patient had episode of Acute coronary syndrome. Patient was managed with Inj.Tenecteplase and underwent Coronary angiography which was suggestive of Thrombotic single vessel coronary artery disease of left anterior descending artery (LAD). As Pharmacoinvasive approach patient underwent percutaneous transluminal coronary angioplasty to LAD and was discharged asymptomatic. Patient had no cardiac or respiratory complaints on follow up.

Case Study

A 40-year-old male, came to emergency department with history of fever, dry cough and generalized bodyache for 2 days. His past medical history was unremarkable. His mother had history of CVA and father was diabetic. On second day patient had fever spike of 103 degree F, HbA1c was 7.6%, fasting blood sugars 210mg/dl and post prandial 233mg/dl and polymerase chain reaction (PCR)-based test for SARS-CoV-2 result was positive. As per the guidelines patient was treated with Tab.Favipiravir and antibiotics. On day 4, patient developed tachypnoea respiratory rate 30 breaths per minute with 96% peripheral oxygen saturation (SpO2) with 2 L/min oxygen with a non-rebreather mask. Blood gas analysis with a non-rebreather mask showed a pH of 7.426, PaCO2 28.3 mmHg, PaO2 97.1 mmHg. C-reactive protein CRP- 22.3 mg/L. He was started on inj. Methyl prednisone, low molecular weight heparin and inj Remdesivir with blood sugar level monitoring. On day 7, patient developed respiratory distress, with respiratory rate 45/min, restlessness, patient was desaturating on O2,15 L/min and was shifted to Non-invasive ventilation (NIV), injection Meropenam and Tecoplanin for 14 days. Blood gas analysis revealed pH 7.4, pCO2 40mm Hg, pO2 96 mmHg, sO2 97% (FiO2-40%, NIV), C-reactive protein was raised to 44mg/L and IL-6 levels 203. With raised inflammatory markers patient was suspected to have cytokine storm. A decision to give first dose injection tocilizumab followed by conavalscent o+ plasma was considered. On day 8, patient was unable to maintain saturation on NIV (60% of H2O). second dose of inj. Tocilizumab and convalescent plasma was given. Day 9 and 10, similar treatment was continued for next 2 days and patients NIV requirement was decreased to overnight support. O2 requirement gradually decreased to 6 litres/min. On day 13, repeat RTPCR test was done, which came out to be negative and patient was shifted to isolation room to prevent secondary infection. On day 16, patient was shifted to ward on O2 by bag and mask ventilation. Patient was discharged on with normal room air saturation. After discharge patient develop post covid complications of fungal infection tenia cruris for which he was treated with oral antifungal tab Itraconazole and local ointment. Also patient developed erythematous plaques with rash on the back, which was diagnosed as post covid coagulopathy (ISTH DIC SCORE 5). patient had platelet count of 81000/mm3, fibrinogen 0.9 gm/L, prothrombin time of 11 seconds and INR of 1.6. Patient was followed up closely with anticoagulation and tapering doses of steroids. Post 4 months discharge patient developed hip pain on walking on care evaluation and MRI Imagining it was diagnosed as avascular necrosis of the Femur Head. Patient underwent Total Hip Arthroplasty after 2 months of Diagnosis and failure of conservative approach.

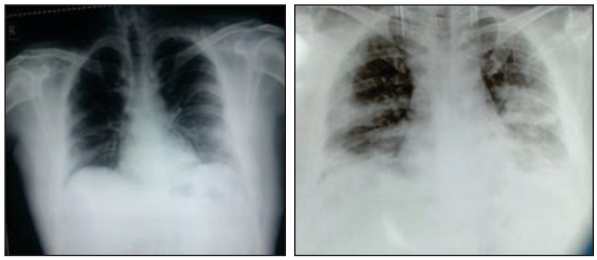

Fig 1.(a) DAY 7 **Fig1.(b) On Discharge**

Fig 1.(a) Xray Chest AP view suggestive of covid 19 bilateral pneumonia (b) X Ray Chest AP view complete resolution of covid 19 pneumonia on discharge

After being asymptomatic for 1 year, patient developed typical retrosternal chest pain along with sweating and diaphoresis. On doing electrocardiograph, evidence of acute coronary syndrome Anteroseptal Myocardial infarction (Fig 2) was noted.

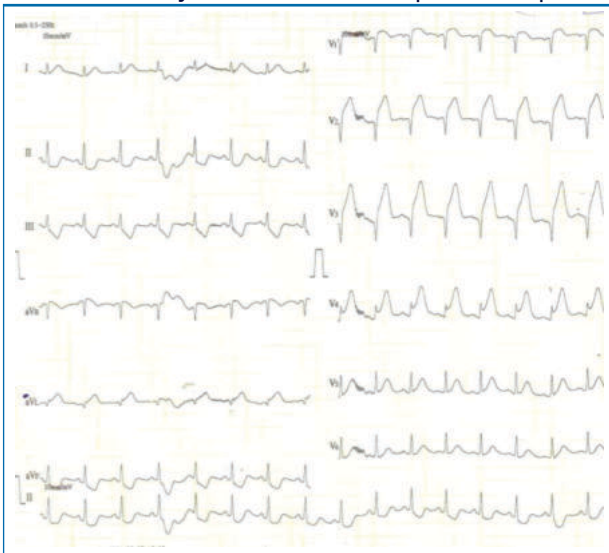

Fig 2. ECG suggestive of ACS Anteroseptal Wall Myocardial Infarction (ST elevation from V1-V4)

Patient was managed with Inj.Tenecteplase and underwent Coronary angiography which was suggestive of Thrombotic single vessel coronary artery disease of left anterior descending artery (LAD). As Pharmacoinvasive approach patient underwent percutaneous transluminal coronary angioplasty to LAD and was discharged asymptomatic. Patient had no cardiac or respiratory complaints on follow up.

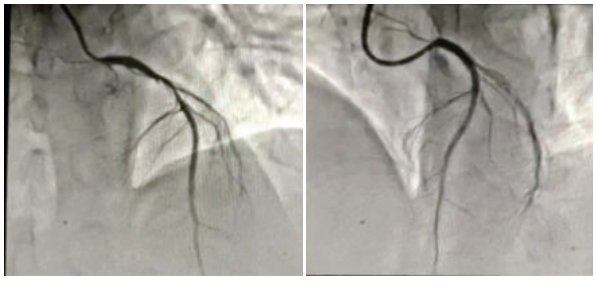

Fig 3 (a). LAO Cranial Projection on Coronary Angiography showing 80% Thrombotic Occlusion of Left Anterior Descending (LAD) Artery. (b) Post Coronary Angioplasty Status showing TIMI III Flow in LAD

CONCLUSION:

Coagulation abnormalities are typically found in patients with severe COVID-19 and are associated with intense inflammation. Elevated D-dimer and fibrinogen levels are the hallmark laboratory findings of COVID-19-associated coagulopathy (CAC) [1,2]. D-dimer levels greatly increase due to the release of urokinase-type plasminogen activator by alveolar macrophages, causing upregulation of local fibrinolysis in alveoli. The second mechanism is direct infection of endothelial cells, leading to a massive release of plasminogen activators [8]. In sepsis-induced coagulopathy (SIC) and disseminated intravascular coagulation (DIC), there is excessive production of PAI1 so that fibrinolysis is often suppressed, and, as an implication, D-dimer levels are usually not as high as in CAC. A relationship between COVID-19 infection and cardiovascular disease is well established. Studies have shown that patients infected with SARS-CoV-2 in the setting of preexisting cardiovascular disease have increased risk of severe disease and death.24 One study demonstrated an 8% risk of acute cardiac injury in patients with COVID-19, with a 13-fold higher incidence in critically ill patients. Notably, nearly all of these studies used biomarkers and/or EKG to diagnose acute myocardial injury[3]. Radiology can play a key role in differentiating between these entities, as evidenced by the cardiac MRI findings of this patient. Given the different treatment strategies for myocarditis versus coronary thrombosis, results of catheter angiography and cardiac imaging may serve as a critical branch point in the treatment of patients presenting with cardiac symptoms in the setting of COVID-19 infection. Cardiac involvement can be due to viral injury of cardiac myocytes. COVID-19 causes systemic inflammation and increased coronary blood flow which can lead to rupture of plaque due to increased shear stress[12]. Our patient fortunately did well, and he was successfully discharged on long-term anticoagulation.

CONCLUSION:

Physicians must be aware of the fact that cardiovascular complications must be considered in the COVID-19 patients, even with minimal risk factors for heart disease. COVID-19-associated coagulopathy frequently occurs in severe COVID-19 infection. Progressive CAC will lead to massive arterial thrombosis in the form of Acute Coronary Syndrome. Here we present A rare case of refractory hyper coagulopathy in covid 19 pneumonia presenting as Post covid coagulopathy, Avascular Necrosis of Femur Head and Acute coronary syndrome.

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