Medical and Research Publications
International Open Access

Case Report **Journal of MAR Pulmonology (Volume 3 Issue 5)**

Acute Refractory Hypoxemia Due to Pulmonary Saddle Embolism in a COVID -19 Patient with ARDS. Past Present and Future and Review of Literature.

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Received Date: September 07, 2021

Published date: October 01, 2021

Abstract

Patient: Male, 43-year-Old

Final Diagnoses: Bilateral Saddle Embolism, COVID-19, ARDS

Symptoms: Hypotension, tachycardia, hypoxemia,

Medication: Alteplase (rTPA), Remdesivir and Dexamethasone, norepinephrine, vasopressin.

Clinical Procedure: Streptokinase infusion

Specialty: Pulmonary & Critical Care Medicine

Objective: Unusual clinical course

MeSH Keywords: *pulmonary saddle embolism, hypoxemia, endotracheal intubation, Alteplase, ARDS, COVID-19*

Background:

We report the case of a COVID-19 patient's survival after presentation with acute hypoxemic respiratory failure and refractive hypoxemia aggravated by a saddle pulmonary embolism. We treated the patient with Streptokinase 100 mg infusion, Remdesivir and dexamethasone which are standard COVID-19 therapy at the time this case was written. We intubated the patient due to increased hypoxic respiratory failure, he was treated using ARDS Net protocol of low tidal volume and high peep with sedation protocol, rotoprone and paralytics due to ventilator desynchrony with plateau pressures of equal or less than 30 mmHg. After being 15 days on the ventilator, the patient had a tracheostomy performed and feeding tube placed, he was downgrade from intensive care to the Rehabilitation Unit for continuation of treatment.

Hypercoagulability is described as one of the compilations to COVID-19 infection, contributing to the patient's hypoxia. Many of these patients can have pulmonary embolism, deep vein thrombosis, ischemic stroke and myocardial infarction. The initial laboratory findings at the beginning of the infection can present, D-dimer and fibrinogen levels as increased, while prothrombin time, activated partial prothrombin time, and platelet counts are often normal. It is believed, there is a large cytokine released and increased inflammatory response resulting from specific interactions between the body's immune response and coagulation system. Acute saddle thromboembolism can be fatal due to its location between the lungs two main arteries and the restriction on oxygen flow. The embolism restricts the lung perfusion, in the presence of COVID-19 hypoxic respiratory failure, it can cause refractory hypoxemia, right ventricular dysfunction, decreased perfusion, increase V/Q mismatch. These events lead to impaired cardiac output and cardiopulmonary collapse leading to hemodynamic collapse and cardiac arrest. In a situation where the patient has significant VQ mismatch and refractory hypoxemia, improvement of the right ventricular function and increased in cardiac output can be a lifesaving procedure and should be the immediate goal of therapy. As improving the cardiac output is actively impaired due to the embolus impeding the right ventricular cardiac outflow. The pathogenesis and spectrum of COVID-19 is still being studied, acute inflammatory dysregulation leading to probable hypercoagulability is believed to be a component.

Case Report:

43-year-old male presented to the emergency room after practicing social distancing due to COVID-19. The patient complaints, of cough, fever, and progressive SOB for the past two weeks. Patient did not get vaccinated. Patient lives in an endemic area with the Delta genetic variant (B.1.617.2, AY.1, AY.2, AY.3), believed to cause more infection and spread faster than previous variants, as indicated by the CDC. This patient was seen in the emergency room, he presented with increased oxygen requirements and low saturations of 85% at room air that corrected with supplemental oxygen 5 liters to 93%. PCR testing

confirmed the diagnoses of COVID-19 viral infection. Chest radiograph, revealed bilateral ground glass opacities, characteristics of COVID-19 pneumonia. Despite the initiation of steroids, Remdesivir and antibiotics, his overall condition continued to decline. A chest tomography angiogram was ordered, it showed bilateral pulmonary embolism. Laboratory testing provided, white blood cell count (WBC) was 22 x103 , Beta Natriuretic Peptide (BNP) of 850 pg./ML, Ferritin of 4554 ng/L and a D dimer > 20. The rest of the chemistries and liver functions were initially normal. The patient was tachycardic and despite further fluid administration and increased oxygen supplementation, he required mechanical intubation. Patient was placed on the ventilator and received Alteplase. Patient was in both hypotensive and hypoxic shock due to the size of the embolus. His initial mechanical ventilation settings were on assist control mode 16 with a PEEP of 10 and 100% of oxygen. His Arterial Blood Gasses (Abg), were 7.13, pCO2 53 pO2 54 %. The PEEP was titrated to 18, the patient was paralyzed with Rocuronium and subsequently placed prone on a rotoprone bed on high PEEP with low tidal volume and plateau of 30, his PO2 improved to 139. Patient also required norepinephrine at 30 mcg/min and Vasopressin infusion. Overnight saturation improved and over the course of the next 15 days, his chest radiographs gradually improved. The patient had a tracheostomy and percutaneous endogastric tube and was discharged from the acute hospital to a long-term care facility. The patient's bilateral venous doppler was negative, confirming there were no additional emboli. The patient had no immediate bleeding or other complications from the administration of TPA to treat the bilateral pulmonary embolism.

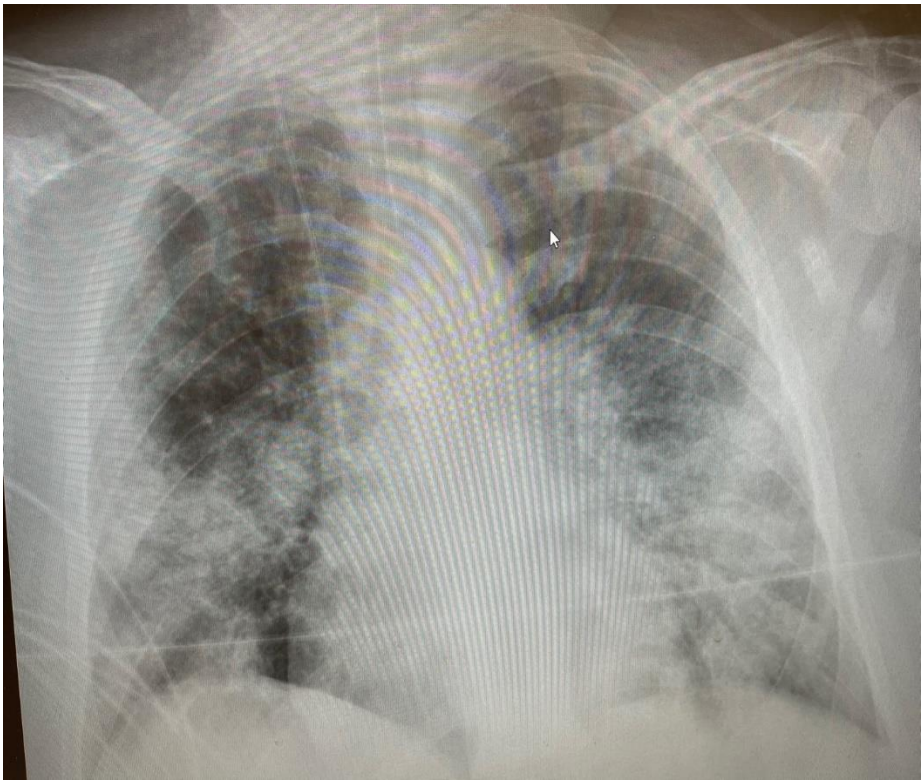

Figure 1. Chest Radiograph demonstrated bilateral opacities with bilateral infiltrates significant for acute respiratory distress syndrome (ARDS) due to Corona virus 2019 Disease.

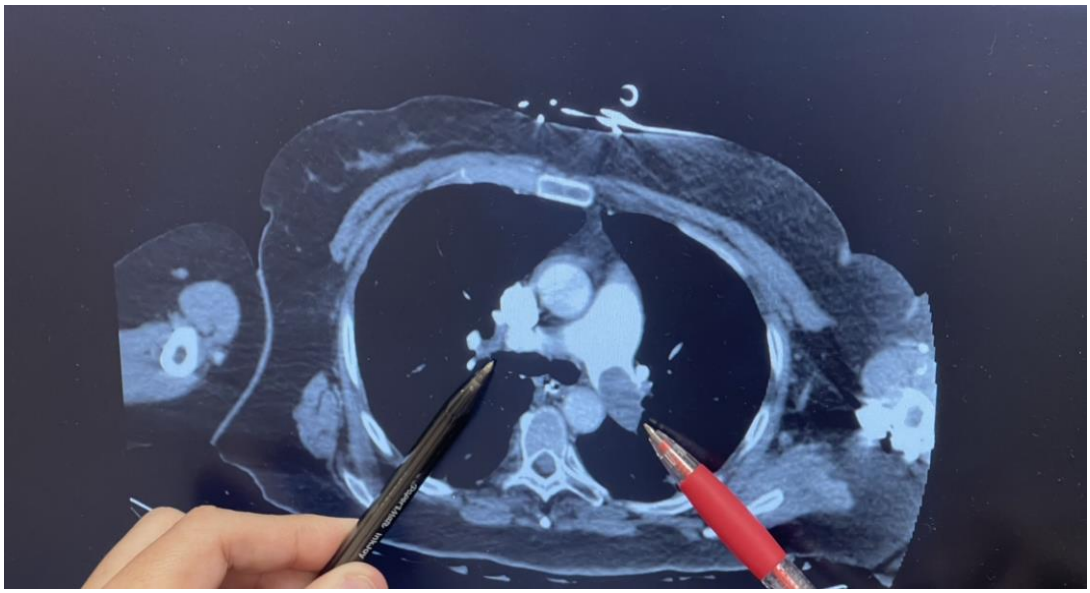

Figure 2. Image of a 43-year-old man with COVID-19 Pneumonia. The CTA shows evidence of bilateral Pulmonary embolism demonstrating filling defects in the segmental areas.

Conclusion:

Respiratory infections have been described as a risk factor in development of pulmonary embolism. Studies have shown that COVID-19 patients who had chest tomography angiograms were reported to have up to 30 % pulmonary embolism. This patient population is unique in that the proinflammatory effects, cytokine release and large body mass index, (BMI), with chronic comorbidities are all risk factors for thromboembolism. Some studies have shown that high level of C reactive protein and D dimers are associated with PE in COVID-19. Ruling out the presence of pulmonary embolism in these patients must be considered when patients have a decline in lung function and increased refractory hypoxemia due to loss of compliance and acute ARDS. The Logic of Thrombolysis with tissue plasminogen activator (tPA) is an established treatment strategy for patients that are at high-risk for pulmonary embolism (PE) and signs of hemodynamic instability, right ventricular strain, shock and right ventricular failure. The use of tPA in corona virus disease 2019 (COVID-19) patients with pulmonary embolism PE and acute respiratory distress syndrome (ARDS) may be of benefit due to the unusually high incidence of pulmonary embolism and pulmonary thrombosis, particularly microvascular thrombosis, which are thought to contribute significantly to hypoxemia. It may also decrease the effects of extravascular and intra-alveolar fibrin deposition described in ARDS. Small case series have reported transient improvements in oxygenation without significant bleeding from systemic fibrinolytic therapy in patients with ARDS and COVID-19. Studies of ARDS have shown a PF ratio of < 100 mm Hg is associated with a mortality rate of 53%, and 40% for a ratio of 100–300. This group, therefore, had a very poor prognosis

but current mortality rates for COVID-19 after ICU admission are even higher, being 88% in a large series. During the H1N11 flu pandemic there were also centers that reported increased thrombotic risk in patients with severe acute respiratory distress syndrome.

It is logical to use tPA in patients with severe PaO2/FiO2 ratios as they have higher mortality and are in a hypercoagulable state with an incidence of venous thrombosis (VT) and PE that exceeds that seen in other pneumonias. In addition, there is gross inflammation of endothelium leading to inflammation-driven local pulmonary thrombosis in medium and small vessels. Large and small thrombi cause vascular shunting and hypoxemia which may be relieved by clot lysis. In addition, inflammation results in a vascular leak allowing extravascular fibrin formation in lung parenchyma tissue and in alveoli with associated hypofibrinolytic shutdown. During the H1N11 flu pandemic there were also centers that reported increased thrombotic risk in patients with severe acute respiratory distress syndrome. As more patients with Delta variant, described by the CDC as being two-fold more contagious than previous variants, or more virulent strains develop along with the increasing BMI of our population. It is conceivable and probable that incidence of thromboembolism may be higher and subsequently be a major cause of morbidly and mortality. Prompt exclusion with tomography and therapy with thrombolytics and anticoagulation is imperative to improve survival and improve V/Q mismatch.

On patients with no evidence of pulmonary embolism, prophylactic anticoagulation with low molecular weight heparin will be of benefit to prevent micro embolism. The possibility a catheter or surgical thrombectomy has been described in the literature but with the vast numbers of cases with COVID-19 and acute respiratory failure we decided to use alteplase infusion to expedite therapy and minimize complications.

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