Case Report

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Unusual Presentation of a Covid Patient with Thrombo-Embolism with Survival

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Abstract

Corona virus disease 2019 (COVID-19) is a viral illness caused by a novel corona virus, severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) pandemic that has affected >188 countries, involved >24 million people, and caused >840,000 deaths. COVID-19, in its severe form, presents as acute respiratory distress syndrome (ARDS), shock, and multiorgan failure. Thrombotic microangiopathy of the lungs and kidneys has been observed in these patients. Elevated D-dimer levels have been observed in people with serious COVID-19 illness, and this could be helpful in guiding treatment with anticoagulation in these patients.

Keywords: COVID-19 illness, and this could be helpful in guiding treatment with anticoagulation in these patients.

Introduction

SARS-CoV-2 enters cells by endocytosis through angiotensin-converting enzyme 2 (ACE2) receptors, which are present in type 2 alveolar cells of the lung, myocardium, and proximal renal tubules. COVID-19 manifestations can range from asymptomatic infections to multiorgan failure and death. The respiratory system is involved in almost all cases. Additionally, there have been many cases of COVID-19 like illness where patients display cardinal features of COVID-19 but a negative nasal PCR test. On repeat/more intensive testing, these individuals are found to have COVID-19 and thus represent the false-negative fraction of testing. In both proven COVID-19 and COVID-19 like illness, fever, cough, dyspnea, chest pain, and oxygen desaturation upon exertion are observed. These symptoms have been associated with increased D-dimer levels and pulmonary microangiopathy on necropsy. The kidneys can be involved, with a similar path physiology, presenting with hematuria and rapidly progressive renal failure with oliguria and death. We present the case of a COVID-19 patient

who developed acute worsening of the illness but then improved rapidly with anticoagulation. This case highlights the multisystem involvement of SARS-CoV-2, raising the possibility of early and intensive anticoagulation as a supportive treatment measure.

Case Report

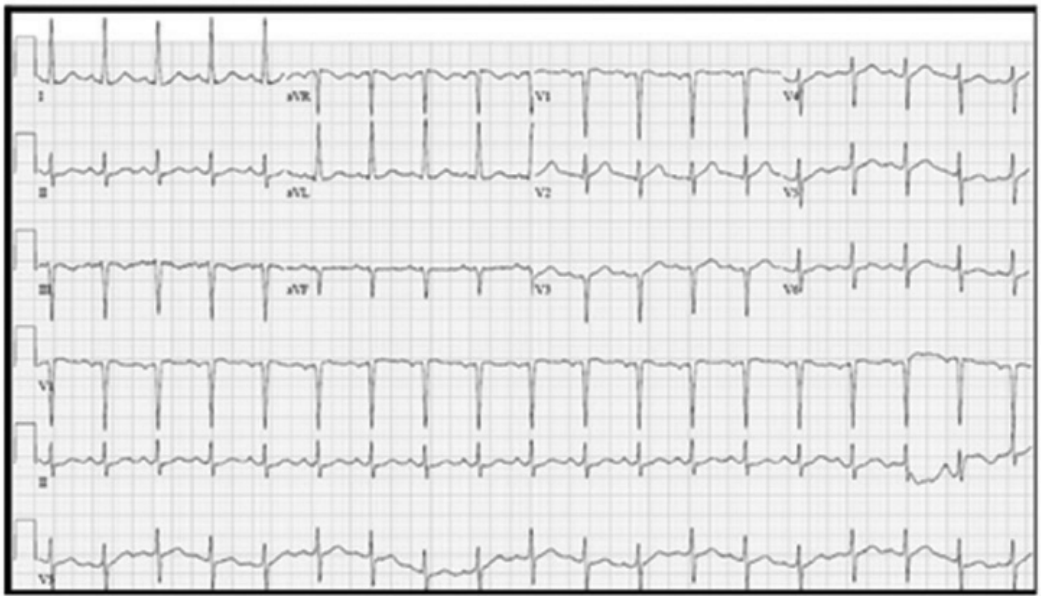
A 53-year-old female with a past medical history of uncontrolled type 2 diabetes mellitus, hypertension, and hyperlipidemia presented to the hospital with weakness, shortness of breath, and diarrhea. Five days earlier, she was tested positive for COVID-19 and was treated for COVID-19 related pneumonia in the hospital for a total of 5 days. She was discharged from that hospital but returned to the ER of our hospital on the same day since she was feeling weak and short of breath after returning home, and her oxygen saturation on pulse oximetry at home was in the range of 80–85%.

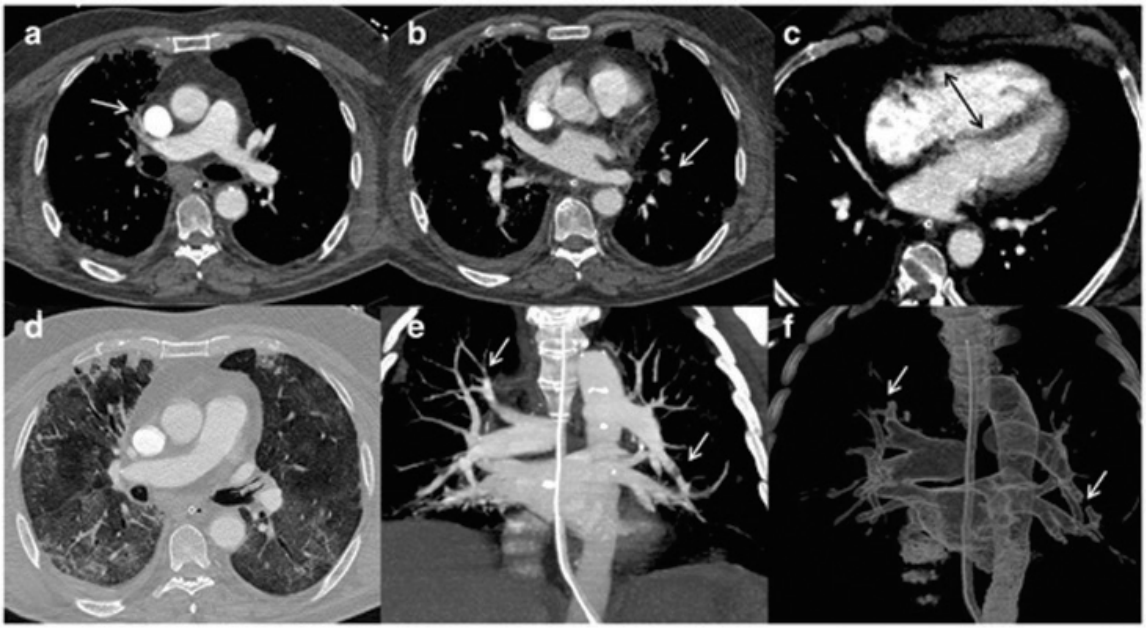
In our ER, she was febrile and tachycardic, and required 4 L/min oxygen via nasal cannula to maintain an oxygen saturation >92%. Laboratory workup showed elevated levels of: serum creatinine 1.14 mg/dL (normal 0.85–1.25 mg/dL; patient's baseline 0.8 mg/dL), lactate dehydrogenase (LDH) 393 U/L (normal 7–55 U/L), ferritin 724 ng/mL (normal 11–307 ng/mL), C-reactive protein (CRP) 44.8 mg/L (normal <10 mg/L), D-dimer 0.67 mg/L (normal <0.5 mg/L), and interleukin (IL)-6 48 pg/mL (normal ≤1.8 pg/mL). Chest X-ray showed patchy bilateral airspace disease, and treatment was initiated with 1 L of intravenous fluid bolus, vancomycin, cefepime, and azithromycin.

Two days later, the patient became tachypneic and hypoxic, requiring a nonrebreather mask. Her respiratory status continued to worsen, and she developed acute hypoxic respiratory failure, for which she was intubated and placed in the prone position in the intensive care unit (ICU). Her inflammatory and coagulation markers continued to worsen, with ferritin 4,848 ng/mL (normal 11–307 ng/mL), LDH 679 U/L (normal 7–55 U/L), CRP 400.79 mg/L (normal <10 mg/L), and D-dimer 4.85 mg/L (normal <0.5 mg/L). She was diagnosed with cytokine release syndrome (CRS) related to COVID-19. Treatment was provided, i.e., corticosteroids, 2 rounds of tocilizumab, and 1 dose of convalescent plasma. One day later, she developed deep vein thrombosis (DVT) in the right internal jugular and the left brachial veins, and anticoagulation was started with an intravenous heparin infusion.

Segmental left lower lobe and right upper lobe PE. Segmental bilateral embolisms (arrows) can be appreciated in axial (a, b) and oblique MIP and VR images (e, f) over a moderate-severe (score 3) pulmonary involvement (d). Signs of right cardiac overload (black arrow) with interventricular septum shifting towards the left ventricle are shown in c.

In the subsequent days of treatment with anticoagulation, the patient's clinical status started to improve, with resolution of the respiratory failure and shock. Anticoagulant was changed to apixaban, Her PaO_2/FiO_2 ratio started to improve and paralytic agents were stopped. She was ultimately extubated to supplemental oxygen





inhalation via a nasal cannula, and then discharged to a skilled nursing facility for rehabilitation.

Discussion

The respiratory system is the most commonly involved, characterized by acute respiratory distress syndrome (ARDS), but systemic involvement leading to shock and multiorgan failure can also be observed, and the prognosis is poor.

The most common laboratory abnormalities seen in these patients are neutrophilia, lymphopenia, thrombocytopenia, and elevated serum creatinine, C-reactive protein (CRP), ferritin, prothrombin time (PT), and D-dimer.

Complications related to coagulation abnormalities, including pulmonary and renal microangiopathy, arterial and venous thromboembolism presenting as acute ischemic stroke, DVT and pulmonary embolism, and arterial and venous catheter thrombosis are being reported. These complications are referred to as COVID-19 associated coagulopathy (CAC). They are most likely due to the profound inflammatory cytokine response associated with the illness, including the increased expression of IL-1, IL-6, and tumor necrosis factor (TNF)-α. IL-6 initiates coagulation activation by inducing tissue factor expression on endothelial cells, and IL-1 and TNF-α suppress the endogenous anticoagulant pathways. CAC is postulated to be a cause of ARDS by forming fibrin-platelet microthrombi in the pulmonary microcirculation and parenchyma.

TMA manifests as thrombosis from endothelial involvement of arteriolar and capillary wall, leading to hemolytic anemia, thrombocytopenia, and multiorgan failure. Clotting in CVVH circuits is already a known complication, but an increasing number of cases are being reported in COVID-19 patients and treated with therapeutic anticoagulation. These complications are found to be associated with an increased incidence of death.

The severity of COVID-19 and the associated mortality is observed to be higher in patients with elevated D-dimer levels. D-dimer is a fibrin-degradation product whose levels are increased by the breakdown of thrombi. It acts as a marker for the activation of widespread coagulation and fibrinolysis. COVID-19 patients with elevated D-dimer (i.e., a 3- to 4-fold increase) should be considered for hospitalization, even in the absence of other symptoms, because this signifies an increased generation of thrombin. Elevated D-dimer in COVID-19 patients with sudden respiratory insufficiency should always raise the concern of pulmonary embolism.

The International Society on Thrombosis and Haemostasis (ISTH) has identified the preliminary phase of sepsis-associated disseminated intravascular coagulopathy as "sepsis-induced coagulopathy" (SIC), and patients meeting the diagnostic criteria for SIC have a proven benefit from anticoagulant therapy

The role of heparin anticoagulation in CAC was evaluated in a study showing a 20% reduction in mortality in patients with severe COVID-19 (and a SIC score ≥4) with a D-dimer value >3 mg/L given prophylactic-dose heparin anticoagulation. Also in

our patient, the D-dimer level started increasing as the disease progressed, but then decreased within days after starting anticoagulation, and the patient experienced a rapid improvement in pulmonary and renal function.

Conclusion

COVID-19 has affected >188 countries, and it presents as sepsis, acute respiratory distress syndrome, and multiorgan failure in its most severe form. Coagulopathy including thrombotic microangiopathy of multiple organs has been observed in COVID-19 patients. Elevated D-dimer levels could hint towards a poor prognosis unless timely treatment with high-dose anticoagulation is initiated.Anticoagulation could be an essential treatment of COVID-19 patients.

Conflict of Interest: None Declared

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