CASE REPORT

A 22-year-old COVID-19 positive male with no prior medical history presented to the emergency department with pulmonary infarction

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ABSTRACT
Rapidly growing evidence has now shown a high incidence of venous thrombosis in patients with severe acute respiratory syndrome secondary to novel coronavirus 2, a disease now named COVID-19. Accumulating case reports and series have also shown a higher prevalence of arterial thrombosis in these patients as well. Although the pathophysiology remains unknown but likely multifactorial – including endothelitis from direct viral damage and an underlying hyper-inflammatory state, arterial and venous thrombosis occurrence does not appear to be linked with underlying classic risk factors for venous thromboembolism and may present in healthy patients without significant comorbidities. We present a case of a 22-year-old healthy patient with COVID-19 who developed a pulmonary embolism with a pulmonary infarction, a complication that results from arterial and venous thrombosis of the pulmonary vascular supply resulting in tissue necrosis.

1. Introduction
Coronavirus disease 2019 (COVID-19) is a viral illness caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). SARS-CoV-2 infection may be asymptomatic or it may cause a wide spectrum of symptoms ranging from respiratory tract infections to overwhelming sepsis. Prothrombotic coagulation abnormalities and thromboembolism is a frequent complication seen in critically ill patients with COVID-19, and these complications may contribute to morbidity and mortality [1].

Accumulating evidence suggests that hospitalized patients with COVID-19 may have a high incidence of venous [2] and arterial thrombosis [3], including those receiving standard thromboprophylaxis. A multicenter retrospective cohort study showed no evidence of arterial or venous thrombosis in any of the COVID-19 outpatients; however, it showed arterial and venous thrombosis in the hospitalized patients with a higher incidence in the ICU patients [3]. We present an unusual case of a 22-year-old male diagnosed with COVID-19 as an outpatient, presented 3 weeks later with dyspnea, after resolving all his flu-like symptoms, found to have a pulmonary infarction.

2. Case presentation
A 22-year-old year Hispanic male with no past medical history came with complaints of shortness of breath and pleuritic chest pain for 2 days along with hemoptysis. He described sharp chest pain worsened with deep inspiration as well as two episodes of hemoptysis. The patient was tested positive for COVID-19 infection 3 weeks prior to presentation after he had symptoms of fatigue, fever, cough, and watery diarrhea. At that time, he self-quarantined and did not require hospitalization or oxygen supplementation. He reported that he was tested again 5 days prior to his presentation and was told that the result was negative. The patient denied any family history of blood clots. He is a non-smoker, did not have any recent surgeries, and is not taking any medications. In the ED, he was tachycardic with a heart rate of 120, normotensive, saturating 100% on room air. On laboratory analysis, troponin negative, d-dimer 3669 ng/ml, BNP <15, fibrinogen 653 mg/dl, aPTT 32, INR 1.1. CBC and metabolic panels were within normal limits, The Rapid COVID-19 test came back positive. EKG showed sinus tachycardia. CXR showed wedge-shaped infarct (Figure 1), CTA of the chest showed bilateral pulmonary emboli right more than left with right lower lobe pulmonary infarction (Figures 2–3). The patient was admitted to the ICU for monitoring. No thrombolyis was required as the patient was hemodynamically stable. The patient was started on high-range heparin infusion (18 units/kg/hr). The venous duplex showed no evidence of deep venous thrombosis. Echocardiogram showed normal EF 60%, McConnell’s sign but no evidence of right
heart strain. The patient was monitored in the ICU for 48 hours on high range heparin then the patient was transferred to the telemetry floor and discharged on rivaroxaban.

3. Discussion

Severe Acute Respiratory Syndrome due to COVID-19 pneumonia was found to be associated with arterial and venous thrombosis. Many case series in different countries around the world showed a high incidence of VTE in hospitalized COVID-19 patients, even those receiving anticoagulants thromboprophylaxis [1–4]. Isaac et al. showed arterial thrombosis, including MI, stroke, and limb ischemia occurring in approximately 4% of critically ill COVID-19 patients [5]. The risk of thromboembolism in non hospitalized COVID-19 patients remains unknown.

The pathophysiology of thrombosis in COVID-19 Patients is still unclear. Endotheliitis, hypercoagulability, and hyperviscosity are described in different studies. Endotheliitis is caused by direct viral infection, facilitated by the overexpression of angiotensin-converting enzyme receptor 2, to the endothelial cells with inflammatory cell infiltration was described in Ackermann et al. study. Angiogenesis was also an unexpected finding that was found in this study distinguishing COVID-19 histopathology from Influenza A (H1N1) [6]. Zhang et al. found that hypercoagulability markers as D-dimers, fibrinogen, and factor VIII were found to be elevated in most participants. Protein c, protein S, and antithrombin deficiency were detected in all participants [7]. Systemic extrapulmonary hyper-inflammation and hypercytokinemia are thought to be a cause of hyperviscosity [5,8]. Microthrombi in the lung arteries were described in a case series of autopsy reports from patients with severe acute respiratory syndrome due to COVID-19 [9]. This finding of in situ thrombosis supports the arterial thrombosis and the unusual high prevalence of PE in COVID-19 patients without evidence of DVT [10]. Our 22 year old patient with no known risk factor developed pulmonary embolism with pulmonary infarction without evidence of DVT, supports the possibility of in situ venous and arterial thrombosis.

The proper prophylactic anticoagulation regimen for COVID-19 patients is still under investigations through multiple clinical trials. These clinical trials compare different regimens and doses of heparin, LMWH, and DOAC regarding the mortality and rates of thromboembolic events in hospitalized patients with COVID-19 [11–13]. Interestingly, the INSPIRATION trial also investigates atorvastatin besides LMWH and heparin, giving the anti-inflammatory and antithrombotic effects of statins [13].
4. Conclusion

COVID-19 has been associated with thromboembolic disease in inpatient and outpatient settings. Our case emphasizes the need for further studies for a better understanding of pathophysiology and the appropriate anticoagulant prophylactic and therapeutic regimens in COVID-19 patients.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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