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## COVID-19: A Potential Risk Factor for Acute Pulmonary Embolism

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**ABSTRACT:** A COVID-19–positive patient presented with pleuritic chest pain and cough and was found to have acute pulmonary embolisms (APEs). There has been an increase in observational reports of venous thromboembolic events in patients who are positive for COVID-19, especially in the setting of elevated inflammatory markers. The possibility of COVID-19 as an independent risk factor for APEs should be further explored in this novel pandemic.

## INTRODUCTION

Acute infections are associated with an increased risk of venous thromboembolic events (VTEs) due to a transient heightened inflammatory state.<sup>1</sup> This pathophysiology has been linked to excessive cytokine production (cytokine storm) that results in increased hypercoagulability, an unregulated immune response that can lead to systemic complications such as disseminated intravascular coagulation (DIC) and even death. Some data suggest that predisposition to VTEs and acute pulmonary embolisms (APEs) may be due to this inflammatory response and DIC, which has been associated with the severity of infection.<sup>3</sup>

Observational data have described patients positive for COVID-19 (the SARS-CoV-2 virus), noting that they have presented with mild symptoms of fever and productive cough as well as acute respiratory distress syndrome (ARDS) with other serious complications, including VTEs and APEs.<sup>1,3</sup> Though this may suggest a possible association between cytokine storm and DIC, it is still unclear why infections due to SARS-CoV-2, versus other diseases, lead to a higher incidence of thromboembolic events despite illness severity.<sup>5</sup>

We describe a case of a patient with COVID-19 who presented with chest pain and cough and was found to have an APE. It could be possible that COVID-19 infections may be predisposing factors for VTEs.

## CASE DESCRIPTION

A 41-year-old man presented to the emergency department with chest pain and a dry cough. His medical history included asthma, which was well controlled with an albuterol inhaler as

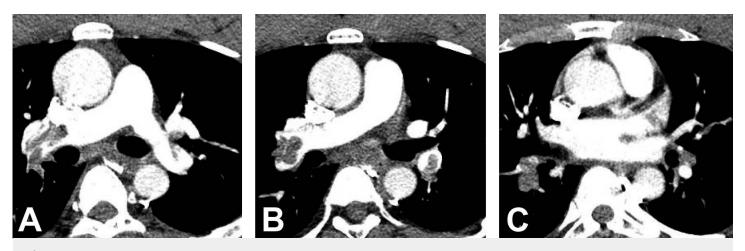


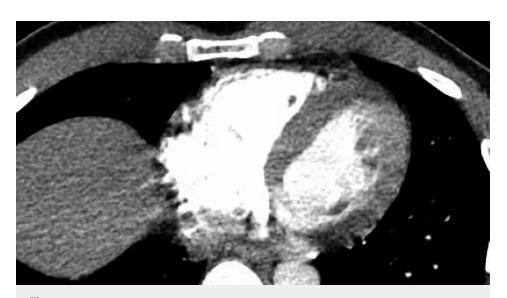
Figure 1.

Computed tomography angiography of chest showing (A) bilateral proximal, (B) segmental, and (C) subsegmental pulmonary embolisms.

needed. The chest pain began 2 days prior and was pleuritic and unresponsive to acetaminophen and albuterol. He also stated that his wife had a similar cough. Triage vitals were within normal limits, with an oxygen saturation of 98%, and physical exam demonstrated normal cardiac and pulmonary auscultation. Baseline electrocardiogram showed an incomplete right bundle branch block and no other abnormalities. Initial chest x-ray was normal. Laboratory work was remarkable for elevated D-dimer of 8,051 (normal upper limit is 230). Subsequent computed tomography angiogram (CTA) of the chest revealed extensive bilateral pulmonary embolisms, right heart strain, and ground glass opacities concerning for an atypical infectious process (Figures 1-3). The patient was found to be positive for COVID-19 via nasopharyngeal swab (PCR test). He denied having fevers, dyspnea, recent travel, surgeries, or a history of clotting disorders or malignancies. He was admitted to medical telemetry and fully anticoagulated with enoxaparin. Cardiac enzymes, clotting factors, and other lab work were normal. A transthoracic echocardiogram showed normal right ventricular size with no evidence of elevated pulmonary artery pressures or other abnormalities (Online videos 1-3). Lower-extremity duplexes were negative for deep venous thrombosis (DVTs). A CT of the abdomen and pelvis did not reveal evidence of malignancy. The patient was discharged with oral rivaroxaban without further complications.

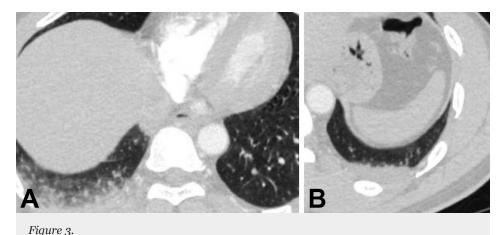
## DISCUSSION

The novel coronavirus, known as SARS-CoV-2 or COVID-19, is a contagious respiratory illness responsible for a global pandemic that, as of spring 2000, has yet to be under control. Its clinical spectrum may range from a mild pneumonia to ARDS secondary to a cytokine



## *Figure 2.* Computed tomography angiography of the chest showing right-to-left ventricular size ratio of approximately 0.9 concerning for early signs of right heart strain.

storm. Although our patient had mild symptoms without hypoxia, he had bilateral pulmonary embolism in addition to ground glass opacities, which is the hallmark of COVID 19. No major predisposing risk factors for APE were identified in our patient, which suggests that even mild-tomoderate COVID-19 manifestations can precipitate acute VTE. Other countries with earlier exposure to this novel virus have reported COVID-19 pneumonia associated with APE in the absence of risk factors and have suggested a causal relationship.<sup>1,2</sup> It is important to consider pulmonary embolism in the differential of not only deteriorating COVID-19 patients but also those with milder presentations, as in our patient. This can have serious



Computed tomography angiography of chest showing bilateral ground glass opacities concerning for an atypical infectious process.

management implications, such as therapeutic anticoagulation, to prevent future decompensation.

Despite this, there is limited data of thromboembolic risk in patients with COVID-19, especially those without other risk factors. Anecdotally, clinicians have reported patients with COVID-19-precipitated ARDS leading to subsequent decompensation with further hypoxia and right ventricle dysfunction. Although this may result from other causes, including high positive end expiratory pressure, pulmonary embolism and other venous thromboembolic disease must remain in the differential given their occurrence even in low-risk presentations, as with our patient.

## CONCLUSION

We described a rare case of concomitant bilateral pulmonary embolism and COVID-19 in the setting of no other predisposing risk factors. Special attention needs to be paid to the possibility of COVID-19 being an independent risk factor for acute venous thromboembolisms. Further investigations into the casual relationship are necessary in this novel pandemic.

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#### Conflict of Interest Disclosure:

The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

#### Keywords:

computed tomography angiogram, acute pulmonary embolism, venous thromboembolism, coronavirus, acute respiratory distress syndrome

### REFERENCES

- Rotzingera DC, Beigelman-Aubrya C, von Garnierb C, Qanadli SD. Letter to the Editors in Chief: Pulmonary embolism in patients with COVID-19: Time to change the paradigm of computed tomography. Thromb Research [Internet]. 2020 Apr 11 [cited 2020 Apr 23];190:58-9. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7151364/ pdf/main.pdf.
- Danzi B, Loffi M, Galeazzi G, Gherbesi E. Acute pulmonary embolism and COVID-19 pneumonia: a random association? Eur Heart J. 2020 Mar 30;ehaa254. Online ahead of print.
- Smeeth L, Cook C, Thomas S, Hall A, Hubbard R, Vallance P. Risk of deep vein thrombosis and pulmonary embolism after acute infection in a community setting. Lancet. 2006 Apr 1;367(9516):1075-9.
- 4. Becattini C, Agnelli G. Pathogenesis of venous thromboembolism. Curr Opin Pulm Med. 2002 Sep;8(5):360-4.
- Schmidt M, Horvath-Puho E, Thomsen RW, Smeeth L, Sorensen HT. Acute infections and venous thromboembolism. J Intern Med. 2012 Jun;271(6): 608-18.
- Kyrle PA, Eichinger S. Deep vein thrombosis. Lancet. 2005 Mar26-Apr 1;365(9465):1163-74.