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Pulmonary Vascular Disease

SESSION TITLE: Medical Student/Resident Pulmonary Vascular Disease Posters

SESSION TYPE: Med Student/Res Case Rep Poster

PRESENTED ON: October 18-21, 2020

PULMONARY EMBOLISM AND RIGHT HEART STRAIN PATTERN IN COVID-19: AN INCIDENTAL FINDING OR A PERSISTENT PATTERN?

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INTRODUCTION: Coronavirus disease 2019 (COVID-19) is presumed to be a prothrombotic state however, there is no concrete evidence supporting therapeutic anticoagulation in this disease. Here, we present a case of a 67-year old male diagnosed with COVID-19 who developed an acute pulmonary embolism and right ventricular strain pattern during hospitalization and was treated with therapeutic Lovenox.

CASE PRESENTATION: A 67-year-old male who had tested positive for COVID-19 one week ago presented to the hospital with shortness of breath, fever (T 102.8), and generalized body aches. His oxygen saturation worsened, and he was put on the nasal cannula, later transitioning to non-rebreather, and finally intubation for not maintaining saturation. Computerized tomography (CT) scan of the chest on admission revealed bilateral, bibasilar and sub-pleural ground-glass opacities. (Figure 1) One week later, he became hypotensive, tachycardiac, and hypoxic on the same ventilator settings. Bedside echocardiogram revealed right ventricular dilatation and akinesis of the mid free wall. (Figure 2) A provisional diagnosis of pulmonary embolism was made and he was treated with therapeutic Lovenox. CT scan of the chest with contrast confirmed the diagnosis of pulmonary embolism. (Figure 3) His hospital course was complicated by pneumonia treated which was treated with antibiotics. He was discharged to a nursing home with a tracheostomy tube and Eliquis on the third week of hospitalization.

DISCUSSION: Emerging as a cluster of pneumonia cases in Wuhan, China in December 2019, COVID-19 has spread across the world. It attaches to angiotensin-converting enzyme 2 expressed in the lungs, heart, and gastrointestinal tract. [1] Widespread microvascular thrombi in the pulmonary circulation as evidenced by autopsy studies cause profound hypoxia by a ventilation-perfusion mismatch in the lungs. The proposed mechanism of COVID-19 thrombosis includes hypercoagulable state and cytokine-mediated damage. [2] It is believed that 50 % of these patients have elevated D-dimer levels that in combination with elevated prothrombin time and reduction in fibrinogen level have been associated with increased mortality.[3] Some enterprises are therapeutically anticoagulating these patients based on these coagulation parameters while others are not.

CONCLUSIONS: COVID-19 increases the incidence of microvascular and microvascular thrombotic complications. Worsening respiratory status not explained by radiological changes in the lung fields, and especially in conjunction with high titers of D-dimers, should raise the suspicion for pulmonary embolism. More studies are needed to evaluate the need for therapeutic anticoagulation in these patients without objective evidence of thrombosis.

Reference #1: Yang XL, Wang XG, Hu B, Zhang L, Zhang W, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature*. 2020. <https://doi.org/10.1038/s41586-020-2012-7>.

Reference #2: Mei H., Hu Y. (2020) Characteristics, causes, diagnosis and treatment of coagulation dysfunction in patients with COVID-19. *Zhonghua Xue Ye Xue Za Zhi* 41:E002.

Reference #3: ang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. *Journal of Thrombosis and Haemostasis*. 2020 Apr 1.

DISCLOSURES: No relevant relationships by John Madara, source=Web Response

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DOI: <http://dx.doi.org/10.1016/j.chest.2020.08.1836>

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