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Pulmonary Embolism: A Serious Risk in COVID-19

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Editorial

COVID-19 is an acute, complex condition associated with SARS-CoV-2 infection, characterized by interstitial pneumonia and acute respiratory distress syndrome in its most extreme form. Infections with COVID-19 are linked to a lot of coagulation system activation. This so-called COVID-19 coagulopathy has been linked to a poor prognosis and increased mortality. Furthermore, COVID-19 infection is linked to a high risk of venous thromboembolism, particularly acute pulmonary embolism (PE). Pulmonary embolism is a life-threatening disease that accounts for 15% of in-hospital deaths in the United States per year. Right-sided heart failure, obstructive shock, hypoxia, and death in the most serious cases cause clinical deterioration. Pulmonary embolism is still not a well-known complication of acute respiratory distress syndrome (ARDS); the serious condition and ensuing extended hospital stay reflect a hypercoagulable state in general. The early initiation of anticoagulant therapy in patients with COVID-19 who are at risk of pulmonary embolism (PE) may improve prognosis.

On the other hand, ARDS caused by COVID 19 infection tends to be a more complicated scenario. Thrombotic complications such as pulmonary embolism and venous thromboembolism have been confirmed to be more common in recent observational studies. Autopsy results from SARS-CoV-19 and MERS patients revealed pulmonary microvascular thrombosis. While pneumonia complicated by ARDS remains the most common cause of hypoxia in COVID-19, new evidence suggests that venous thromboembolism may also play a role, contributing to the complexity of acute PE management.

During this pandemic, the best clinical approach must consider the patient's risk of decompensation, the possible advantage of intervention, the risk of provider exposure and infection, and the availability of hospital services. According to preliminary studies, thrombocytopenia, elevated D-dimer, prolonged prothrombin duration, and disseminated intravascular coagulation was found in COVID-19 patients' coagulation disorders. When computed tomography angiography (CTA) is perforated, pulmonary embolism in hospitalized COVID-19 patients is diagnosed based on the clinical observation of rapid worsening of respiratory insufficiency and blood oxygenation that is out of proportion to the extent of pulmonary infiltration, as well as evidence of pulmonary vessel occlusions, which are generally interpreted as caused by pulmonary emboli.

Patients with COVID 19 pneumonia who are sick enough to be admitted to the hospital have several clinical symptoms in common, such as immobility, acute respiratory failure, inflammation status, and analytical parameters like high D-dimers which make the diagnosis of PE difficult. Both patients who had a CT scan while in the hospital to rule out PE had experienced respiratory worsening not due to other factors or an increase in D-dimer that was not consistent with other inflammatory markers.

Patients with COVID-19 had elevated D-dimer values in up to 43% of cases, with higher values seen in patients with more serious disease. As a result, determining whom to examine for co-morbid pulmonary embolism (PE) in the case of COVID-19 infection is extremely difficult. It is not easy to tell the difference between pulmonary thrombi and pulmonary emboli because their pathogenesis and treatment are arguably distinct. The patient's inflammatory markers and clinical condition improved a few days after receiving prompt COVID-19 and PE treatment with hydroxychloroquine, solumedrol, and heparin.

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