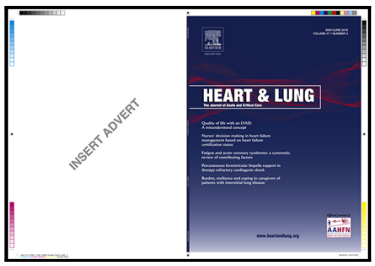

Comment on: High incidence of pulmonary thromboembolism in hospitalized SARS-CoV-2 infected patients despite thromboprophylaxis

FNU Mehak , FNU Deepak ,
Gianeshwaree Alias Rachna Panjwani

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Correspondance: 1. FNU Mehak

Institute: Ghulam Muhammad Mahar Medical college, Sukkur

Department: Medicine

City: Sukkur, Pakistan

Email: Mehaklund99@gmail.com

2. FNU Deepak

Institute: Ghulam Muhammad Mahar Medical college, Sukkur

Department: Medicine

City: Sukkur, Pakistan

Email: Deepakdembra9@gmail.com

3. Gianeshwaree Alias Rachna Panjwani

Institute: Ghulam Muhammad Mahar Medical college, Sukkur

Department: Medicine

City: Lahore, Pakistan

Email: rachnakukreja7@gmail.com

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Letter:
To the Editor:
The article "High incidence of pulmonary thromboembolism in hospitalized SARS-CoV-2 infected patients despite thromboprophylaxis" by El-Qutob et al. was reviewed with great enthusiasm.[1] It was fortunate to read this article, and the author's efforts are to be applauded.

Despite thromboprophylaxis, we concur with the conclusion that individuals infected with SARS-CoV-2 have a high risk of developing pulmonary embolism, as diagnosed by CT pulmonary angiography. However, we are compelled to mention additional points that would enhance the quality of this article and expand previous knowledge.

First, the potential for reporting bias and imprecise verifiable evidence in a retrospective cohort study design raises significant doubts about the study's veracity. Despite the widespread belief that endothelial injury, decreased flow/stasis, and a prothrombotic state all contribute to the development of thrombosis, the precise nature of these risk factors remains unknown.[2] Prevalence estimates for PE ranged from 23% to 57% among patients with severe COVID-19 infection, according to statistics from early French observations. PE imaging findings were also strongly associated with the need for MV. When persistent hemodynamic compromise, evidence of survival benefits, and improved long-term outcomes are present, thrombolysis is recommended as an urgent treatment option for PE. It's debatable whether thrombolysis can be performed without causing damage to the heart and blood vessels. Moreover, refractory hypoxia is not viewed as a typical indication for thrombolysis, which carries the risk of intracerebral hemorrhage; therefore not without risk.[2,3] The authors should have clarified the pathogenesis underlying this phenomenon. Such as a 2021 study by Yash et al.[3] reported that By triggering both thrombosis and inflammation, COVID-19 makes people more prone to bleeding disorders (SARS-CoV-2) (SARS-CoV-2). The SARS-CoV-2 virus initially infects the alveolar epithelium by binding to the ACE2 receptor, which stimulates the release of excessive pro-inflammatory cytokines (IL-6 and TNF) and chemokines (IL-8, CXCL8, CCL2 and CCL3).[4] Neutrophils, macrophages, and epithelium awaken simultaneously. Infection-induced activation and dysfunction of endothelial cells via the ACE2 receptor induce a coagulation cascade that generates thrombin and fibrin clots. Platelet activation and the protease-activated receptor signalling pathway exacerbate lesion formation and inflammatory processes.[3,4]

Antiphospholipid antibodies have been linked to an increased risk of thrombosis in some patients with severe COVID-19. Inflammation and chemokine storms raise the risk of thromboembolism. Pregnancy and surgery increased our patient's risk for thromboembolism. Authors have argued that the biomarker D-dimer for poor prognosis should not be used to determine anticoagulant dosage. It was discovered that clinical criteria and D-dimer levels are significant in the diagnosis of VTE. Multi-detector CTPA and ultrasound compression are the imaging methods for PE and DVT, respectively. Using the YEARS diagnostic algorithm, which considers clinical signs of DVT, hemoptysis, and whether PE is the most likely diagnosis, PE can be safely ruled out in patients with a suspected PE.[5]

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