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Thrombosis and COVID-19 pneumonia: the clot thickens!

Laura C. Price[1,2], Colm McCabe[1,2], Ben Garfield[1] and Stephen J. Wort[1,2]

Affiliations: [1]Royal Brompton and Harefield NHS Trust, London, UK. [2]National Heart and Lung Institute, Imperial College, London, UK.

Correspondence: Laura C. Price, Royal Brompton and Harefield NHS Trust, Sydney Street, London, SW3 6NP, UK. E-mail laura.price@rbht.nhs.uk

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Pulmonary thrombosis appears to be common in COVID-19 pneumonia and takes two forms, proximal pulmonary emboli and/or distal thrombosis. The possible mechanisms and clinical implications are discussed. https://bit.ly/372Xdhw

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At the end of last year, a novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), resulted in an acute respiratory illness epidemic in Wuhan, China [1, 2]. The World Health Organization (WHO) termed this illness coronavirus disease 2019 (COVID-19). The coronavirus family have been shown to enter cells through binding angiotensin-converting enzyme 2 (ACE-2), found mainly on alveolar epithelium and endothelium. Activation of endothelial cells is thought to be the primary driver for the increasingly recognised complication of thrombosis. Viral inclusion bodies have been identified in endothelial cells in a variety of organs, from the lung to the gastrointestinal tract [3]. The immune dysregulation characteristic of severe COVID-19 infection may be initiated by "pyroptosis", a particularly pro-inflammatory form of apoptosis initially described in macrophages [4], with rapid viral replication leading to massive release of inflammatory mediators. One of the most consistent findings is that of a raised D-dimer level. Although many inflammatory processes can influence D-dimer levels, it almost certainly reflects, to some extent, intravascular thrombosis in patients with COVID-19 [5, 6]. In the early studies emerging from China, an elevated D-dimer level (>1000 $ng \cdot mL^{-1}$) at admission was associated with increased risk of in-hospital death [7]. An elevated D-dimer continues to be one of the most consistent markers of poor outcome [8].