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Pulmonary infarcts in COVID-19

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SARS-CoV-2 binds to the angiotensin-converting enzyme 2 (ACE2) receptors found on vascular endothelial cells among others.[1] Goshua et al. reported that von Willebrand factor is released by activated vascular endothelial cells and modulates platelet adhesiveness and aggregation; it was most elevated in sickest COVID-19 patients.[2] Thus, the endothelial cell infection leads to a hypercoaguable state providing a rationale for the extent of microthrombi seen in those who succumbed to the disease.

Chest computer tomography (CT) allows the noninvasive evaluation of pulmonary emboli and sequela of endothelial injury. Pulmonary emboli and Hamptons humps (Fig. 1) are known CT findings associated with vascular clotting and parenchymal ischemia and/or infarction.[3] Larger pulmonary emboli are relatively rare compared to the microthrombotic disease reported in COVID-19.[1] A recent study found pulmonary emboli in 20% of 51 COVID-19 positive patients and 19% of COVID-19 negative patients.[4] However, microthrombi were identified in 90% of autopsies performed on patients with COVID-19.[5]

Hampton's humps representing ischemic or infarcted lung are found in less than 36% of patients with pulmonary emboli who do not have COVID-19 infection.[6] The lung is spared in many patients due to its dual blood supply with a bronchial circulation that connects to the pulmonary arterial circulation and responds to ischemia by increasing its volume 300% following pulmonary embolus.[7] In contrast, Lax et al. found infarcts at autopsy in 8 of 11(81%) of patients with COVID-19 related pulmonary thrombosis.[8]

The reason for increased infarcts in patients with COVID-19 is likely multifactorial. First, there is a larger clot burden overall increasing the likelihood for infarct or ischemia.[1] The micro-thrombotic disease of COVID-19 effects vessels smaller than 3 mm, which are more often associated with infarction than large central embolic disease.[7] Embolic disease spares the bronchial circulation allowing it to compensate for diminished pulmonary arterial supply. In COVID-19, the bronchial endothelial cells are also vulnerable to infection and may be compromised.

Autopsies of COVID-19 patients demonstrate a heavy clot burden with significant infarct and ischemia. PE's are not diagnosed more frequently in COVID-19 likely because the micro thrombi are too small. Increased pulmonary infarcts and ischemia were identified at autopsy and should be visible on CT as Hampton's humps. Zhao et al. demonstrated peripheral opacities in 87 of 101 patients (86%) who were infected with SARS-CoV-2.[9] VQ scan and Dual Energy CT could

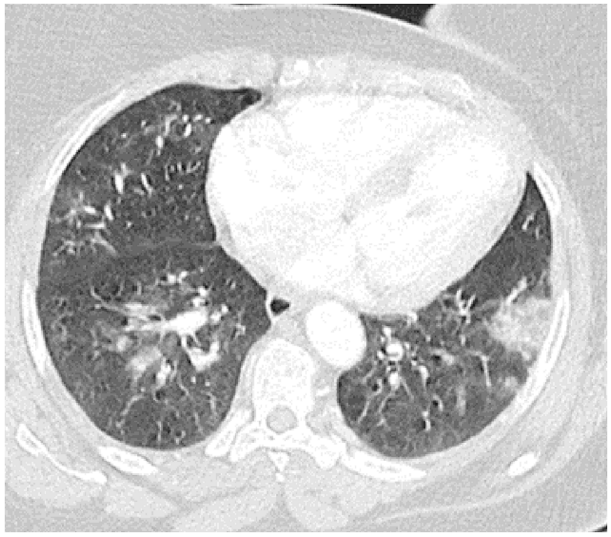

Fig. 1. COVID-19 positive patient with Hampton's hump in the left lower lobe.

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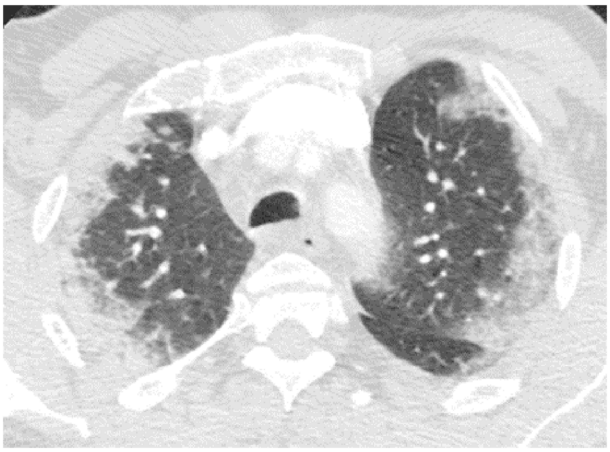

Fig. 2. COVID-19 positive patient with extensive peripheral consolidation.

potentially prove if the peripheral opacities of COVID-19 (Fig. 2) represent ischemia suspected with micro thrombotic disease.[10–14]

Declaration of competing interest

Mary Salvatore received lecture fees from Genentech and Boehringer Ingelheim and serves as a consultant for Genentech and Boehringer Ingelheim and has received a research grant from Genentech and Boehringer Ingelheim.

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