Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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The authors' response to "A more realistic relationship between covid 19 and hemopneumothorax"

Thank you for your comments on our case report and providing us with the opportunity to discuss the possible relationship between COVID-19 and hemopneumothorax. As new information about this disease surfaces daily, we as clinicians must strive to consider novel possibilities for pathophysiology.

The comments propose that COVID-19 would be more likely to result in spontaneous hemothorax via pulmonary emboli (PE) and the subsequent anticoagulant therapy. Indeed, COVID-19 is well known for predisposing patients to thrombotic disease, both in venous and arterial circulations [1]. PE can then lead to both spontaneous pneumothorax (SP) and spontaneous hemopneumothorax (SHP) by way of pulmonary infarction and cavitating lesions [2,3]. Pulmonary infarction is a less common complication of PE, with an incidence ranging from 16 to 31% [4,5]. From the documented cases of SP complicating infarction, the time interval from embolus to pneumothorax may be as long as 5 weeks [2]. Thus, it does seem feasible that a PE would lead to SHP, especially in subacute presentations of COVID-19.

However, because SHP is primarily associated with SP, it will be of interest to note from future studies if patients with COVID-19 have a higher risk of developing SP than the general population, which has an overall incidence of 17–24 per 100,000 in males and 1–6 per 100,000 in females [6]. The mechanism of SP formation in COVID-19 patients is uncertain. It is speculated that the severe inflammatory response gives rise to SP due to degenerative changes in the lung parenchyma [7]. Histologic examinations show diffuse alveolar damage with cellular fibromyxoid exudates, which lead to formation of cystic lesions and alveolar rupture [8].

Currently, there have been multiple case reports of SP and COVID-19, but only a single retrospective review from China citing a 1% incidence [9], which is likely an underestimation as only 99 patients were included and the review was published in the early months of the pandemic. If it is shown that COVID-19 patients have an increased risk of SP, it would follow that these patients may also be at increased risk of SHP. Therefore, it is possible that our case of SHP was due to the inflammation caused by COVID-19, especially since the patient was not previously diagnosed with PE, nor was the patient anticoagulated [10].

Multiple other studies of clinical characteristics have been performed, but none have specifically addressed the rates of SP. We look forward to future research into the clinical presentations of this novel disease.

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Prior presentations

None.

Declaration of Competing Interest

The views expressed in this case report are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government. We are military service members. This work was prepared as part of our official duties. Title 17 U.S.C. 105 provides that "Copyright protection under this title is not available for any work of the United States Government." Title 17 U.S.C. 101 defines a United States Government work as a work prepared by a military service member or employee of the United States Government as part of that person's official duties.

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