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.

Clinical Imaging 88 (2022) 78–79

Contents lists available at ScienceDirect

Clinical Imaging

journal homepage: www.elsevier.com/locate/clinimag

Cardiothoracic Imaging

Pulmonary cavitation in patients with COVID-19

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ARTICLE INFO

Keywords:
COVID-19
Viral infections
Pulmonary cavitation
Computed tomography

To the Editor:

We read with great interest the well-written letter to the editor by Ozgül et al.,[1] who reported that they have observed an increase in the incidence of cavitary lung lesions in patients with COVID-19 infectio4n. The most common cause of these lesions was secondary bacterial infection, mainly by *Staphylococcus aureus*. The authors concluded that secondary bacterial infection should beconsidered first in determining the etiologies of cavitary lesions in patients with COVID-19 pneumonia, with prompt microbiological testing performed for definitive diagnosis.[1]

We would like to highlight that the literature reflects consensus that lung injuries caused by COVID-19 do not cavitate, and that the finding of cavitation should direct the search for alternative diagnoses, or even overlap with other diseases, especially associated secondary infections. None of the published cases demonstrates adequately that pulmonary cavitation is attributable to COVID-19 pneumonia.[2]

Recent studies have demonstrated that pulmonary co-infection by other agents is not uncommon in the COVID-19 context. Complications such as necrotizing pneumonia and subsequent cavitation of lung lesions may occur. Fungal diseases and tuberculosis (Fig. 1A) are other important infections in this context.[3,4] Tadolini et al.[4] reported the detection of cavitary lesions in23 (47%) of 49 patients with current or former tuberculosis and COVID-19.

Pulmonary infarction also should be included in the differential diagnosis of cavitary lesions in patients with COVID-19 (Fig. 1B). Lung cavitation following pulmonary embolism and infarction is an uncommon finding described in patients with diseases other than COVID-19.[2,5] The two types of cavitary pulmonary infarction are bland infarction, caused by aseptic necrosis in the absence of infection, and septic infarction, caused by the superinfection of dead lung tissue. Infected pulmonary infarction leads to cavitation more rapidly than does bland infarction with aseptic necrosis, and infectious cavitary infarction has a higher mortality rate and requires an aggressive approach to improve the outcome.[2,5]

These cavitations must be differentiated from cystic changes, manifesting as small air-containing spaces inside areas of ground-glass opacity (Fig. 1C).[6] The pathophysiology of these cystic airspaces in areas of infiltrates remains unclear. Shi et al.[7] suggested that they can be explained by infection-generated damage to the alveolar walls, which leads to pneumatoceles. Ye et al.[8] hypothesized that they are related to physiological space dilation, are cross-sections of bronchiolectasis, or are associated with consolidation resorption. Other authors have described the development of bulla or emphysema in consolidation areas. In addition, the rupture of cysts, although rare, may cause spontaneous pneumothorax and/or pneumomediastinum.[6]

In conclusion, the presence of cavitation is considered to be an atypical finding of COVID-19, suggesting alternative diagnoses. Clinicians must consider that cavitation may be due to unspecified secondary infection, tuberculosis, fungal disease, or pulmonary infarction caused directly by embolic or thrombotic vascular disease related to COVID-19.

Declaration of competing interest

The authors declare that they have no conflict of interest.

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https://doi.org/10.1016/j.clinimag.2021.04.038
Received 21 April 2021; Accepted 27 April 2021
Available online 7 May 2021

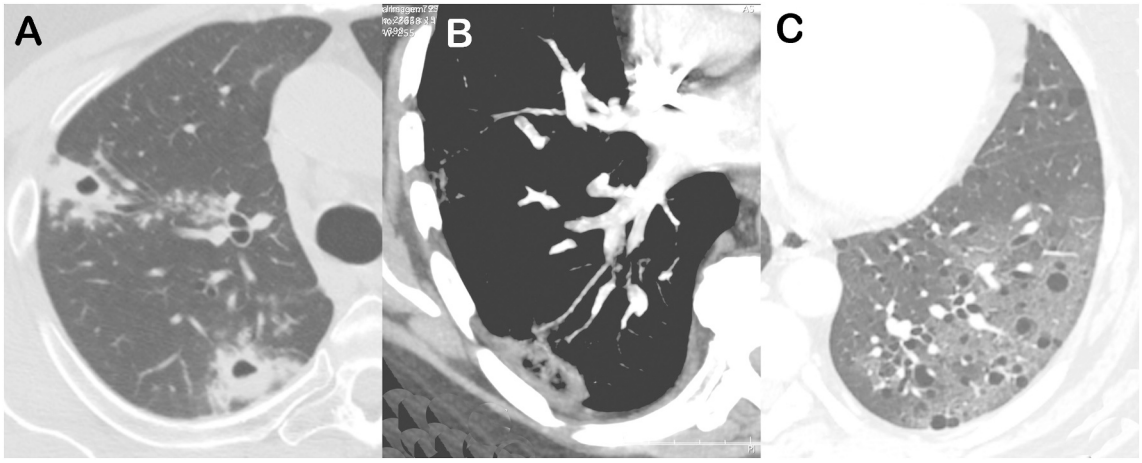

Fig. 1. Cavitary lesions and cystic airspaces in areas of infiltrates in three patients withCOVID-19 confirmed by reverse-transcription polymerase chain reaction. (A) A 51-year-old man with pulmonary tuberculosis. (B) A 66-year-old man with pulmonary thromboembolism and pulmonary infarction. (C) A 47-year-old man with cystic airspaces inside areas of ground-glass opacity.

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