The lung cancer may occur prior to coincidentally with or after many years of diagnosis of PAP. In both PAP and adenocarcinoma, the alveoli are filled with a low-density material, the alveolar septa thicken but the underlying parenchymal architecture remains normal. Solid lung cancers causing PAP have not been extensively studied. As the patient was otherwise tolerating his antiretroviral therapy over two years with a normal CD4 count, considering the slow progression of the disease, the PAP was likely secondary to malignancy. The importance of diagnosing the etiology for the management of a disease cannot be overemphasized especially in a rare disorder like PAP. However, due to the complex pathophysiology of PAP, diagnostic modalities to differentiate the underlying cause in presence of two potential causes of secondary PAP are lacking. The presentation of retroviral disease with adenocarcinoma of lung with PAP is extremely rare and to the best of our knowledge, this is the first reported case in India.

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Cystic airspaces associated with COVID-19 pneumonia

Sir,

We retrospectively reviewed chest computed tomography (CT) images of 43 adult nonsmoking patients with COVID-19-related pneumonia. All patients’ COVID-19 diagnoses were confirmed by positive results of reverse-transcriptase polymerase chain reaction assays of specimens collected on nasopharyngeal swabs. The patients underwent chest CT in March/April 2020 at a private hospital in Rio de Janeiro, Brazil, at a median of 7 (range, 3–13) days after symptom onset. No patient was on mechanical ventilation at the time of the CT examination. Our Institutional Review Board approved the study, and all patients provided written informed consent.

Although several reports have described CT features of COVID-19-related pneumonia, few have highlighted the presence of cystic changes, manifesting as small air-containing spaces associated with other findings in the lung parenchyma. Cystic airspaces associated with COVID-19–related pneumonia have been described as the air bubble sign, vacuoles, round cystic changes, and...
and the cavity sign. All reports on them describe their intermingling with areas of lung parenchyma infiltrates.

We observed small cystic airspaces in the subpleural region and along the peribronchovascular interstitium in infiltrated lung areas in our series [Figure 1]. Thirteen (30.2%) patients presented cystic changes; five (11.6%) patients had cystic airspaces only in the subpleural region and along the peribronchovascular interstitium, and eight (18.6%) patients had cystic airspaces admixed with areas of opacity in the lung parenchyma. Most air-containing spaces had diameters <5 mm, and these spaces were distributed predominantly in the lower lobes.

The pathophysiology of these cystic airspaces in areas of infiltrates remains unclear. Shi et al. suggested that they can be explained by infection-generated damage to the alveolar walls, which leads to pneumatoceles. Ye et al. hypothesized that they are related to physiological space dilation, are cross-sections of bronchiolectasis, or are associated with consolidation resorption. Zhou et al. observed cystic airspaces (which they referred to as vacuolar signs) often in the advanced phase of COVID-19 pneumonia (8–14 days after symptom onset), associated with interstitial involvement and accompanied by repair changes. Other authors have described the development of bulla or emphysema in consolidation areas. We assume that the cystic airspaces may be related to small bulla or emphysema.

Cystic airspaces associated with areas of opacity have not been described often in association with other viral infections. Thus, their presence in areas of infiltrates in association with other features of COVID-19 pneumonia may increase the specificity of the diagnosis in patients for whom confirmatory testing has not been performed. In addition, the rupture of cysts, although rare, may cause spontaneous pneumothorax and/or pneumomediastinum.

In conclusion, air-containing spaces in the lungs are not uncommon CT features of COVID-19–related pneumonia. They may occur in the subpleural region and along the peribronchovascular interstitium in areas of infiltrates. Radiologists should be aware that these cystic changes may be present in association with other features suggestive of COVID-19-related pneumonia. Inappropriate epidemiological and clinical settings, these findings may aid diagnosis.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Falsely low values of oxygen saturation measured by pulse oximetry in patients with coronavirus disease 2019

Sir,

Digital pulse oximetry is a rapid noninvasive test and is used to estimate arterial oxygen saturation. However, falsely low readings are common due to a range of causes including motion artifact, hypotension, nail polish, darker skin pigmentation, and venous pulsations. We recently encountered a number of patients with coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) with falsely low oxygen saturation detected by digital pulse oximetry.

Following the first confirmed COVID-19 case on March 11 in Turkey, our hospital started to serve as a coronavirus pandemic hospital on April 1, 2020. Three hundred and forty-five patients were hospitalized to Istanbul University of Health Sciences Gaziosmanpasa Research and Training Hospital between April 1, 2020, and April 14, 2020. Among these, we identified 17 patients who had a discordance of oxygen saturation measured by digital pulse oximetry and arterial blood gas analysis. This study was approved by our institutional ethics committee (Approvement protocol number: 58/06.05.2020).

The mean (standard deviation [SD]) age was 65.7 ± 17.0 years, and 10 of 17 patients (58.8%) were men. Nine of these 17 patients had fever. Hypotension and tachycardia were not observed in all the patients. Laboratory parameters showed elevated aspartate transaminase (median: 46 U/L, interquartile range [IQR]: 34–58), ferritin (median: 447 ng/mL, IQR: 237–1119), D-dimer (median: 1062 ng/mL, IQR: 774–1387), C-reactive protein (mean ± SD: 141.4 ± 103.1 mg/L), fibrinogen levels (mean ± SD: 382.6 ± 72.0 mg/dL), and low lymphocyte count (mean ± SD: 790 ± 409 cells/µL). The findings of oxygen saturation levels measured by pulse oximetry and arterial blood gas analysis are shown in Table 1. Repeated measurements of oxygen saturation by different pulse oximetry devices were still falsely low in all the patients. Hypertension and chronic ischemic heart disease were the most common comorbidities. In addition, one of them was diagnosed with acute pulmonary thromboembolism. Eleven patients were treated by therapeutic dose enoxaparin, whereas 6 were treated by prophylactic dose enoxaparin. Among the 17 patients, 8 (47%) remained hospitalized at the final study follow-up date, 7 (41.1%) were discharged alive, and the remaining 2 (11.9%) were transferred to the intensive care unit.

The relationship between inflammation due to viral infection and hypercoagulation has already been known. [1] Similarly, extensive intravascular microthrombosis was observed in autopsy series including four deceased cases with COVID-19. [2] Endothelial cell involvement has been suggested a possible reason for impaired microcirculatory function in different vascular beds. [3,4] Ciceri et al. have recently proposed a new hypothesis called “microvascular COVID-19 lung vessels obstructive thromboinflammatory syndrome (MicroCLOTS).” [5] This hypothesis was based on the following findings. First, SARS-CoV-2 enters into endothelial cell through the receptor angiotensin-converting enzyme 2. Second, replication of the virus causes release of pro-inflammatory cytokines and activation of macrophages and the complement cascade in endothelial cells. Activation of complement cascade triggers further immune response, tissue injury, and microvascular thrombosis. Third, the progression of endothelial damage with microvascular thrombosis can spread locally in the lung and potentially extends to the microvascular bed of several organs. In our case series,