

## Case Report

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# Severe covid-19 infection caused rapid multiple pneumatoceles development due to hyper inflammation and accelerated pulmonary fibrosis

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### Abstract

A 70-year-old man was admitted to the internal medicine department complaining of productive cough for three days, severe dyspnea and desaturation with oxygen saturations of 87-89%. On arrival he was diagnosed with severe Covid 19 infection and started treatment with dexamethasone and remdesivir. Mechanical ventilation was installed 7 days after admission. Chest x-ray and chest CT scan issued 3 weeks after admission revealed several large pneumatoceles.

We describe the development of multiple pneumatoceles in severe Covid-19 infection with Adult Respiratory Distress Syndrome (ARDS).

**Keywords:** Pneumatocelle; ARDS; Covid-19; Pulmonary fibrosis.

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### Introduction

There are few reports on Covid-19 diseases describing pneumatocele development [1]. Pneumatoceles were found on X-ray and especially on the CT scan of the chest [2-4]. Covid-19 infection is associated with pneumonia, hyper inflammation process and accelerated fibrosis of the lungs, which is much more common than in other causes of pneumonias from other etiology [5]. Chemokines are elevated in COVID-19 and they stimulate development of lung fibrosis [6,7]. Due to rapid fibrosis—and because of stretched lung tissue, development of pneumatocele is not uncommon. The reasons for this might include the development of ischemic parenchymal damage lung fibrosis, low lung compliance, bronchiolar necrosis and inflammatory exudate in the airway [2-4,8,9]. Recent histological examinations reported bilateral diffuse alveolar destruction with cellular fibromyxoid exudates in COVID-19 [4,9].

Pneumatocelle lesions may form as reaction to cellular fibromyxoid filtrations, which form one way pass-like valve in the bronchus. Here we present a case of severe fibrosis and pneumatoceles development.

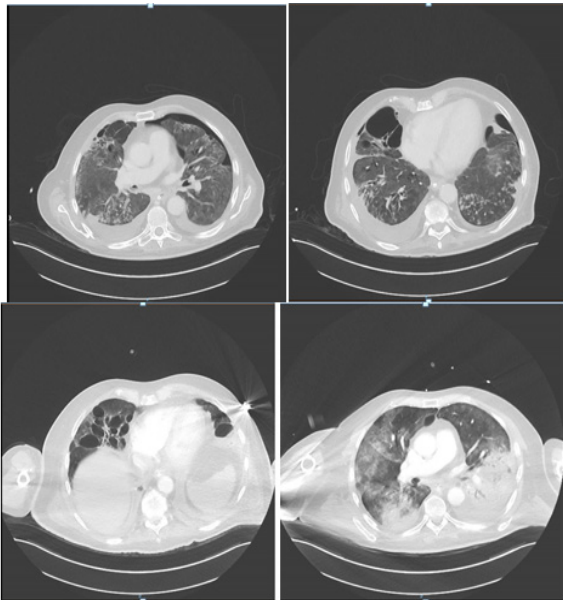
### Case presentation

A 70-year-old man was admitted to ICU in the internal medicine department due to shortness of breath, productive cough for three days before admission and desaturation with of SpO<sub>2</sub> of 89%. He was diagnosed as suffering from severe Covid 19 infection. His previous medical history was unremarkable with no history of prior medication use. Prominent sings on examinations were dyspnea with respiratory rates of 28 per minute, tachycardia 92 per minute, temperature-38.2, cyanosis, rale and crackles on auscultations in both lungs. Chest X-ray on arrival exhibited patchy opacifications in both lungs.

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Blood test showed Hb-13, WBC-3400, 18% lymphocytes (lymphopenia). CRP was 14 (normal values < 0.5). Urea 55 Creatinine 1.3, additional blood work were unremarkable.

Treatment with Remdesivir and dexamethasone 6 mg Intravenous (IV) was initiated. The patient was treated with supplemental oxygen with improvement of SpO<sub>2</sub> to 96%. Within the following 4 days the patients' medical status improved with normalization of body temperature and cyanosis which disappeared. In addition, Respiratory rates decreased to 18 per minute. Laboratory tests showed improvement in CRP decreasing to a value of 10, however, the other blood work did not show any change. Although the patient exhibited partial improvement in medical status, 7 days after admission the patient developed severe respiratory distress and mechanical ventilation was initiated. On recurrent X ray there were more opacifications and large regions with lung fibrosis. CT scan of the lung (Figure 1) showed adult respiratory distress picture with several large (more than 5 cm in diameter) pneumatoceles. The patient continued the treatment with remdesivir for 10 days. Dexamethasone (IV) was continued and the dose was elevated. Blood cultures were negative however because of possibility of co-existing bacterial pneumonia treatment with ceftriaxone and azenil were added on the 11 day after admission. The patient state improved and deteriorated periodically. He was several times extubated and after several days intubated again because of respiratory instability.



**Figure 1:** Four CT scan pictures show bilateral, patchy, ground-glass opacities and consolidations, and multiple pneumatoceles which involves large areas of the lungs

He received several times different antibiotics including imipenem and piperacillin with tazobactam because of different bacteria – klebsiella pneumonia, pseudomonas aeruginosa in the patient's sputum test.

Recurrent CT and X-ray showed severe aggravation in lung fibrosis and significant enlargement of several pneumatoceles with pneumothorax. The patient was referred for lung transplantation after stabilization of his condition.

It is important to emphasize that during 15 weeks of mechanical ventilation the patient lost 10% of his weight mostly due to muscle atrophy including diaphragm (measured by US). Although the patient received treatment of hyperalimentation (by nasogastric tube) blood analysis showed hypoalbuminemia of 1.5 mg/dl because of severe catabolic state. 16 weeks after admission the patient died.

## Discussion

Searching the literature on Covid-19 disease there are few reports describing pneumatocele development [1-4,8]. Pneumatoceles were found on X-ray and especially on the CT scan of the chest. Covid-19 infection is associated with pneumonia, hyperinflammation process and accelerated fibrosis of the lungs, which is much more common than in other causes of pneumonias from other etiology. Chemokines are elevated in COVID-19 and they stimulate development of lung fibrosis [1,2]. Due to rapid fibrosis –and because of stretched lung tissue, development of pneumatocele is not uncommon. The reasons for this might include the development of ischemic parenchymal damage lung fibrosis, low lung compliance, and inflammatory exudate in the airway [7-9].

Recent histological examinations reported bilateral diffuse alveolar destruction with cellular fibromyxoid exudates in COVID-19 [11]. Pneumatoceles lesions together with rapid lung fibrosis may critically reduce effective lung alveolar area and insufficient tissue oxygenation which led to inability of winning from mechanical ventilation, recurrent infections and lethality.

Current report presents an unusual Covid-19 course of the disease which started with a viral phase including clinical status of dyspnea, desaturation with SpO<sub>2</sub> of 89% and fever which deteriorated to severe hyperinflammation state and ARDS indicating the need for mechanical ventilation. The established treatment by remdesivir and dexamethasone and later addition of antibiotics (according to various bacteria) didn't cessate the process. Patient developed severe fibrosis of the lung with ruptures of the fibrotic tissue which caused pneumatoceles to development with critical reduction of the pulmonary alveolar net. All attempts to extubate the patient were unsuccessful. Another explanation is barotrauma in mechanically ventilated patients; however, the ventilation was done on low pressures. The frequency of barotrauma is 6.5 percent in non covid-19 patients [4] however in Covid-19 patients it seems to be more. The incidence of pneumatoceles and pneumothorax is 10% according to the study of Shi H on 81 patients in Wuhan, China [8].

## Conclusion

In conclusion, current report of Covid-19 describes severe respiratory distress which caused lung fibrosis, pneumatoceles and failure from discontinuation of mechanical ventilation.

Mechanical ventilation which is necessary, because of ARDS may be an additional reason by causing barotrauma for pneumatocele development in patients with Covid-19.

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## Declarations

Conflict of interest: None to declare.

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