Pneumomediastinum: A Rare Complication of COVID-19

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Spontaneous pneumomediastinum is an uncommon complication of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pneumonia. Recently, several cases were reported in the literature. In our case, we report the history of an elderly patient with spontaneous pneumomediastinum 2 weeks after coronavirus disease 2019 (COVID-19) infection, which was resolved gradually.

PATIENT DESCRIPTION

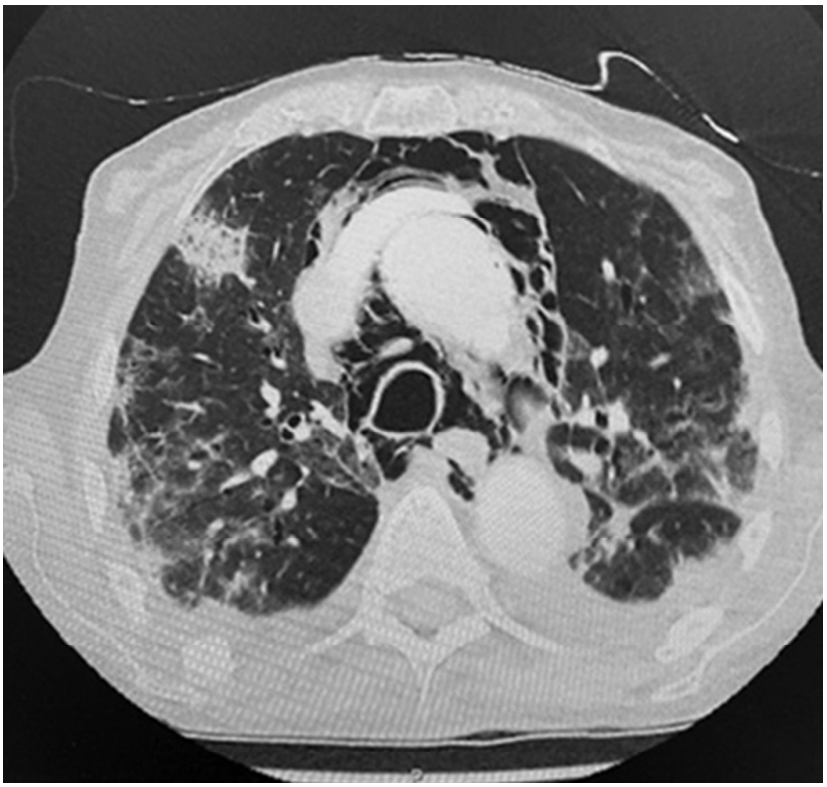
A 90-year-old healthy male, non-smoker, with no chronic diseases or any underlying lung disease, and no continual medications was admitted to our hospital because of dyspnea, fever, and dry cough. Several days before admission, the patient was diagnosed with COVID-19. His vital signs were: blood pressure 187/78, pulse 103/minute, respiratory rate 24/minute, temperature 38.2°C. On admission he was dyspneic and asthenic, his laboratory examination showed an oxygen saturation of 85%, D-dimer 3.18 µg/ml (normal range 0.2–0.5 µg/ml) C-reactive protein 70, procalcitonin 0.64 µg/ml (normal range 0–0.50 µg/ml) IL-6 11.26 pg/ml (normal range 0.0–7.0 pg/ml), hemoglobin 9.0, white blood cell count 12,000, and creatinine 1.7 improved to 1.0. The other laboratory tests were all within the normal rang.

Chest X-ray on admission showed minimal findings. The patient started treatment with antiviral as remdesvir, corticosteroids, vitamin D, enoxaparin, azithromycin, and ceftriaxone as well as supplemental oxygen, inhalation, and physical therapy. The patient did not need mechanical ventilation or other invasive procedure, such as a central venous line.

Two weeks after admission, the patient presented with atypical chest pain and breathlessness. An echocardiograph was normal and serial chest X-ray showed multiple opacities according to COVID-19 infection. Air could not be seen with certainty in the mediastinum [Figure 1]; therefore, a computed tomography (CT) scan was performed to rule out pulmonary embolism, a finding that is also common in COVID-19.

Surprisingly a huge and clear pneumomediastinum was found on the CT [Figure 1]. There were no other precipitating factors to cause pneumomediastinum other than COVID-19 pneumonia. During his hospital stay the patient's clinical situation improved gradually and a clear regression in pneumomediastinum was noted.

Figure 1. Chest X-ray with multiple opacities according to severity of COVID-19 infection



COMMENT

Pneumomediastinum is a rare condition in which air is present in the mediastinum, the overall incidence of 1 in every 25,000 cases in ages 5–34 years, but in COVID-19 is yet unknown. It more commonly affects males and frequently occurs as a result of blunt trauma or esophageal perforation [1]. It is also a well-known complication of barotrauma of the chest including positive airway pressure and mechanical ventilation. Most of forms of pneumomediastinum that occurred during COVID-19 pneumonia were due to an invasive procedure such as mechanical ventilation. In patients who did not undergo an invasive procedure, occurrences were very rare. Only four cases have been reported to date [2-4].

Other associated risk factors include smoking and pre-existing lung parenchymal and airways disease [1]. The mechanism of this complication is the ability of SARS-CoV-2 to infect type I and II pneumocytes, disrupting alveolar membrane integrity and leading to alveolar rupture and leakage of air into interstitial tissue, as well as severe hypoxemia increasing respiratory effort that can cause pneumomediastinum [5].

Spontaneous pneumomediastinum is usually self-limiting with no required interventions only conservative treatment [2]. Rarely, in cases of tension pneumomediastinum, severe cardiopulmonary compromise can occur. Recently several cases have appeared in the medical literature, especially in the years 2020–2021. Our case is the tenth. It is speculated that there will be many more cases in the future with this complication.

CONCLUSIONS

If a patient with COVID-18 suddenly develops acute dyspnea, a chest X-ray or chest CT must be performed as soon as possible to rule out an uncommon complication such as spontaneous pneumomediastinum, a dangerous complication of COVID-19 infection.

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Capsule

Capsid Inhibition with lenacapavir in multidrug-resistant HIV-1 Infection

Patients with multidrug-resistant human immunodeficiency virus type 1 (HIV-1) infection have limited treatment options. Lenacapavir is a first-in-class capsid inhibitor that showed substantial antiviral activity in a phase 1b study. In this phase 3 trial, **Segal-Maurer** and colleagues enrolled patients with multidrug-resistant HIV-1 infection in two cohorts, according to the change in the plasma HIV-1 RNA level between the screening and cohort-selection visits. In cohort 1, patients were first randomly assigned in a 2:1 ratio to receive oral lenacapavir or placebo in addition to their failing therapy for 14 days. During the maintenance period, starting on day 15, patients in the lenacapavir group received subcutaneous lenacapavir once every 6 months, and those in the placebo group received oral lenacapavir, followed by subcutaneous lenacapavir; both groups also received optimized background therapy. In cohort 2, all the patients received open-label oral lenacapavir with optimized background therapy on days 1 through 14; subcutaneous lenacapavir was then administered once every 6 months starting on day 15. The primary end point was the percentage of patients in cohort 1 who had a decrease of at least 0.5 $\log_{10}$ copies per milliliter in the viral load by day 15; a key secondary end point was a viral load of less than 50 copies per milliliter at week 26. A total of 72 patients were enrolled, with 36 in each cohort. In cohort 1, a decrease of at least 0.5 $\log_{10}$ copies per milliliter in the viral load by day 15 was observed in 21 of 24 patients (88%) in the lenacapavir group and in 2 of 12 patients (17%) in the placebo group (absolute difference 71 percentage points, 95% confidence interval 35–90). At week 26, a viral load of less than 50 copies per milliliter was reported in 81% of the patients in cohort 1 and in 83% in cohort 2, with a least-squares mean increase in the CD4+ count of 75 and 104 cells per cubic millimeter, respectively. No serious adverse events related to lenacapavir were identified. In both cohorts, lenacapavir-related capsid substitutions that were associated with decreased susceptibility developed in 8 patients during the maintenance period (6 with M66I substitutions).

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