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Case Report

Pneumopericardium in COVID-19 Infection

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ABSTRACT

Pneumopericardium is a rare medical condition that occurs following trauma, surgery, or other medical interventions. The presence of pneumopericardium after COVID-19 pneumonia has been reported in some cases, and it has been explained that most cases could be self-limited. Here, we describe a 51-year-old man afflicted by pneumopericardium with COVID-19 infection. The patient had pneumopericardium and massive pericardial effusions, necessitating surgical strategies such as pericardial windows. This case highlights the potential severity of COVID-19. We also suggest that cardiologists pay attention to the possibility of pneumopericardium in cases with COVID-19 infection. ***(Iranian Heart Journal 2023; 24(1): 97-103)***

KEYWORDS: COVID-19, Pneumopericardium, Pericardial effusion

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Pneumopericardium is an uncommon alteration defined by the presence of free air in the pericardial cavity preceded by trauma, surgery, or other medical interventions, human immunodeficiency virus (HIV), tuberculosis (TB), and gastro-pericardial fistulae. This disease is mostly presented in preterm children and young men, with chest pain as the most frequent symptom. Spontaneous pneumopericardium could be caused by severe bouts of coughs or a fistulous communication between the pericardium and an infected contiguous organ. [1] The main diagnostic method of spontaneous pneumopericardium is chest radiography. [2] We herein describe a 51-year-old man afflicted by pneumopericardium with COVID-19 pneumonia.

Case Presentation

A 51-year-old man was admitted to our hospital with new progressive dyspnea (from New York Heart Association functional class I to III). His dyspnea had started 3 weeks before admission, and he also complained of pleuritic chest pain, non-productive coughs (since 2 weeks before admission), and orthopnea (since 1 week before admission). During the preceding 6 months, he mentioned no history of weight loss or sweating. In his past medical history, no specific finding was mentioned except for an opium addiction of 12 years' duration. On admission, the patient was awake and alert. He was pale, with an oral temperature of 37 °C, a respiration rate of 20 breaths per minute, an oxygen saturation level of 86% on room air, a blood pressure of 100/60 mm Hg, and a heart rate of 90 beats per minute.

Examinations also showed negative pulses paradox. Further examinations in the neck area demonstrated distended jugular veins. Thorax examinations revealed significantly decreased sounds at the base of both lungs and also muffled heart sounds. Peripheral pulses were also full and symmetric, but pitting edema in the lower limbs (2+) was observed. No lymphadenopathy was detected.

A 12-lead electrocardiogram demonstrated sinus rhythm, low voltage in the limb leads, ST depression in V_1–V_6, and no evidence of electrical alternans. Posterior-anterior chest X-ray showed an increased cardiothoracic ratio and evidence of bilateral pleural effusions (Fig. 1).

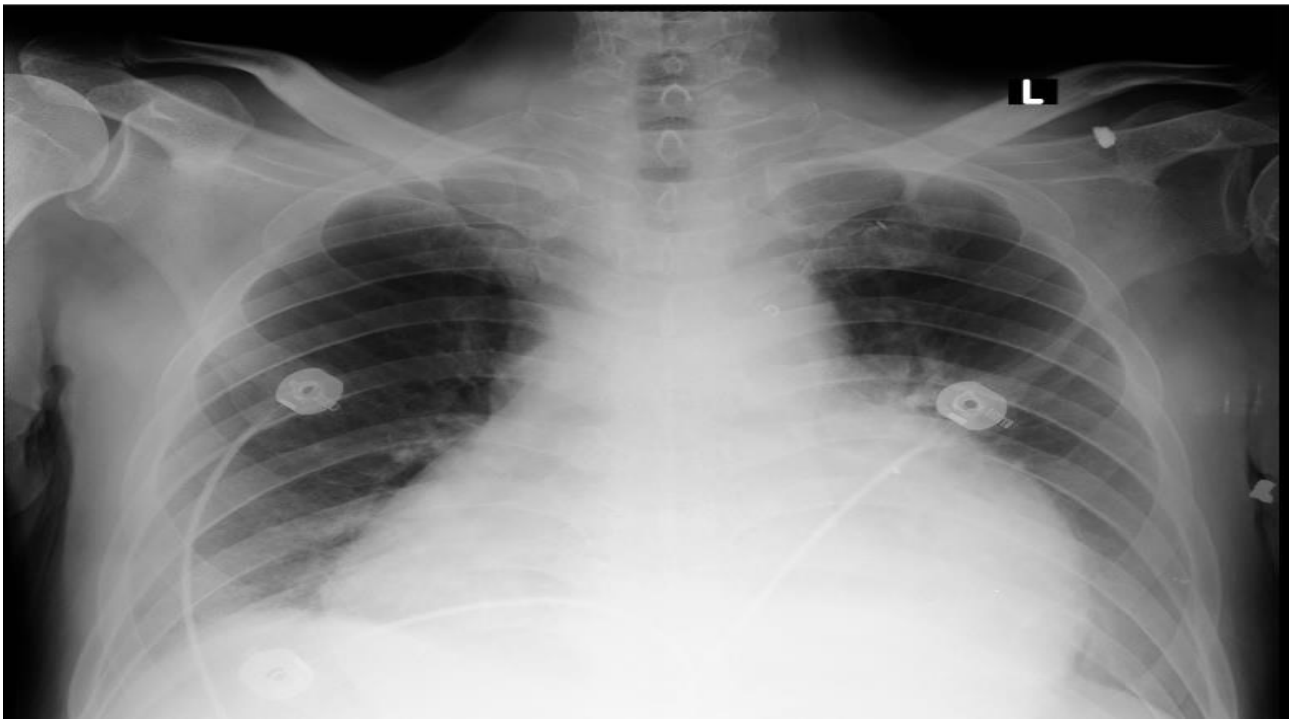

Figure 1: The image presents the patient's chest X-ray in the posterior-anterior view.

Echocardiography, performed in the emergency department, demonstrated a left ventricular ejection fraction of 45% and severe pericardial effusions with abnormal appearance; nonetheless, no collapse in the right atrium and right ventricle was observed. Additionally, the inferior vena cava was dilated without collapse.

The patient underwent neck and abdominopelvic ultrasonography for the evaluation of any possible tumors, but no evidence of malignancy was detected. Bilateral pleural effusions were observed, and 20 mL of a yellow liquid was removed under the guidance of ultrasonography for diagnostic evaluations from the left pleural space. Pleural fluid analysis was exudative, and no blood was detected.

Initial laboratory data showed a white blood cell count of 8700, a neutrophil count of 70%, a lymphocyte count of 20%, a urea level of 23, a creatinine level of 0.9, a hemoglobin level of 11, a mean corpuscular volume of 88.8, a platelet count of 178 000, an erythrocyte sedimentation rate of 15, a C-reactive protein level of 1+, venous blood gas (pH=7.42 PCO_2=40, and HCO_3=26,3), a thyroid-stimulating hormone level of 1.6, negative viral markers, negative troponin, a total iron-binding capacity of 255, a ferritin level of 203, a negative blood culture, negative 2ME, a negative Wright-Coombs test, negative PPD, no peripheral blood smear, no evidence of malignancy, and negative anti-HIV antibodies.

The polymerase chain reaction (PCR) test for COVID-19 was checked for the patient because of the COVID-19 pandemic and the presence of dyspnea. The result was positive, suggesting infection with COVID-19. Therefore, the patient was transferred to an isolated room in the coronary care unit. Afterward, thoracic multidetector computed tomography (MDCT) with contrast was performed for the patient, with the results showing no evidence of pulmonary thromboembolism and peribronchovascular infiltration. Patchy consolidations were depicted basally on both sides. In addition, massive pericardial effusions, bubbles of gas, and mild bilateral pleural effusions were also reported. Furthermore, prominent mediastinal lymph nodes were detected (maximum diameter =12 mm) in the precarinal and aortopulmonary window (Video 1).

Echocardiography was then repeated due to MDCT findings; it showed effusive constrictive pericarditis physiology, pneumopericardium, thick pericardial effusions, and bilateral pleural effusions.

The cardiologist recommended a surgical approach to pericardial drainage, biopsy, and bilateral pleural effusion diagnostic drainage.

The patient also underwent an endoscopy for the evaluation of anemia, dyspnea, and possible gastrointestinal fistulae. This procedure showed evidence of reflux and gastritis, but no evidence of fistulae or malignancy was detected. Thereafter, abdominal MDCT with oral and intravenous contrast was performed, but no evidence of malignancy or fistulae was reported.

The patient underwent close pericardiocentesis, and 400 mL of fluid with a turbid appearance was removed. A pigtail was embedded. After the removal of almost 600 mL of fluid, the procedure was terminated due to the high concentration of the fluid. Cardiac surgery consultation was performed because no liquid was removed via the pigtail. The pericardial window was installed associated with a pericardial biopsy, and 100 mL of purulent fluid was removed (Fig. 2).

Figure 2: The image shows the purulent fluid removed from the patient's pericardium.

The pericardial fluid was sent for analysis, cytology, Gram staining, and smearing. The results were positive for adenosine deaminase (ADA=91). The results also showed an exudative fluid, no blood, and no evidence of malignancy. As a result, a Koch bacillus (BK) culture of the pericardial fluid was performed, and the pericardial tissue was sent for pathologic evaluation. The results of the pericardial biopsy showed fibrosis and myxoid changes, with no evidence of malignancy or tuberculosis. The result of the BK culture was negative. A chest tube was then installed, and 500 mL of liquid with a chylothorax appearance was removed. At the time of hospitalization, the patient received intravenous antibiotics, but they were discontinued within 2 days due to negative blood culture results. Moreover, low doses of diuretics (20 mg of furosemide every 12 h) and colchicine (1 mg daily) were initiated due to clinically prominent edema in the lower limbs. The patient did not receive antiviral drugs.

Outpatient oncology consultation was also requested owing to the observation of prominent perihilar lymph nodes in the thoracic MDCT with contrast. The possibility of lymphoma was also ruled out using peripheral blood smears, laboratory data, and physical examinations. No lymph node biopsy was performed

In follow-up, within 2 weeks and also 2 months after discharge from the hospital, the patient had no lower limb edema, no dyspnea, and no other symptoms. Physical examinations were also normal. Follow-up echocardiography showed a left ventricular ejection fraction of 50%–55% and no evidence of pericardial effusions or constrictive pericarditis. Also 2 months after discharge, thoracic MDCT with contrast showed no evidence of lymphadenopathy.

DISCUSSION

Pneumopericardium is almost a rare condition primarily reported to occur following traumatic or iatrogenic events. To date, some cases of spontaneous pneumopericardium have been reported. The current paper described a 51-year-old man with a history of COVID-19 pneumonia presenting with orthopnea, pleuritic chest pain, and no productive coughs. The patient was later diagnosed with spontaneous pneumopericardium. The present case had pericardial effusions associated with

pneumopericardium. He was evaluated for possible etiologies, and all laboratory tests were negative except the COVID-19 PCR test. Therefore, we decided that the patient was a case of pneumopericardium following COVID-19 pneumonia.

The coronavirus is a large family of viruses that includes the common cold virus and the agent of severe acute respiratory syndrome (SARS). [3] The disease was called "COVID-19" by the World Health Organization (WHO) after the death toll exceeded 1000. [4] The most common clinical manifestations of COVID-19 pneumonia are fever, fatigue, dry coughs, myalgia, shortness of breath, and gastrointestinal complications such as diarrhea and vomiting. [5] There have been reports of pericardial involvement following COVID-19 infection. These involvements include pericardial inflammation, [6] symptomatic pericardial effusions, [7] and acute pericarditis. [8] The most frequently reported cardiac complications of COVID-19 are pericardial effusions and cardiac tamponade.

Recently, some cases of spontaneous pneumopericardium following COVID-19 have been reported. Nonetheless, to the best of our knowledge, this is the first case in Iran. Based on the evidence, COVID-19 infection could cause severe pulmonary damage and lead to acute respiratory distress syndrome in a sizable percentage of infected patients. Diffuse alveolar damage is also another common pulmonary injury among infected patients. We believe that these conditions are associated with pulmonary barotrauma from mechanical ventilation, especially with high positive end-expiratory end-pressure (PEEP). Coughs also constitute another significant recognized risk factor for pneumopericardium and are a common finding in COVID-19-infected patients. Our case also experienced severe coughs both during and after COVID-19 infection and also at the time of hospitalization. A salient point regarding our case was the late initiation of spontaneous pneumopericardium following COVID-19 infection (almost 14 days).

Juárez-Lloclla et al [9] reported 12 cases of spontaneous pneumopericardium and pneumomediastinum following COVID-19 infection in Peru. Based on their report, 10 patients had both pneumopericardium and pneumomediastinum. They showed that fever, coughs, dyspnea, and tachypnea were the most common initial findings, and the diagnostic technique was the chest X-ray associated with the thoracic CT scan. It was also stated that pneumopericardium was an emergency condition due to the chances of pulmonary embolism. Their study emphasized that the COVID-19 pandemic increased the probability of spontaneous pneumopericardium due to severe pulmonary damage following coughs and intubation in patients. Two other cases were reported by Hazariwala et al [10] in 2020. They described 2 patients with COVID-19 pneumonia complicated by spontaneous pneumomediastinum, pneumopericardium, pneumothorax, and subcutaneous emphysema. They also reported that the symptoms of their patients started within 27 and 18 days.

Zhu and Lieber [11] described a 39-year-old man presenting with progressive shortness of breath, fever, and coughs that also had positive test results for COVID-19. They stated that the chest CT on the day of admission showed extensive pneumopericardium, pneumomediastinum with severe subcutaneous emphysema, small bilateral pneumothoraces, and bilateral diffuse ground-glass opacities and consolidation in the lower lobes. Their case did not require chest tubes or any interventional strategies and had complete symptom resolution within 13 days after hospitalization. Another case of pneumopericardium following COVID-19

was reported by Singh et al, [12] who described a 33-year-old Hispanic man presenting with fever, body aches, worsening shortness of breath, coughs, and headaches. He was then diagnosed with COVID-19 infection, with pneumopericardium noted on the chest CT. The signs and symptoms were resolved within 3 days by medical treatments, and a repeated chest CT showed decreasing pneumopericardium and persistent bilateral consolidation. In contrast to our case, the presented case of pneumopericardium was diagnosed by the time of COVID-19 infection and was also self-limited requiring no interventions. Scacciavillani et al [13] described a 61-year-old man diagnosed with COVID-19 pneumonia that also underwent mechanical ventilation. Within a few hours, neck and chest subcutaneous emphysema was noted and pneumopericardium, pneumomediastinum, and left pneumothorax were reported in the chest CT. He required no cardiac procedures and was self-resolved. The main cause of pneumopericardium, in this case, was pulmonary barotrauma from mechanical ventilation, which we discussed earlier.

The administration of pericardial windows and chest tubes to remove the liquid was the main treatment strategy in the mentioned cases. Nevertheless, some cases might be self-limited. Our presented case had pneumopericardium and massive pericardial effusions, necessitating surgical strategies. This issue is highly dependent upon the patient's clinical conditions and risk factors. Older age, past medical history, and obesity are the most significant risks of severe COVID-19 infection that could also lead to complications such as spontaneous pneumopericardium. Within the previously reported cases of spontaneous pneumopericardium following COVID-19, most reported cases had worse outcomes than other causes of pneumopericardium. The reported cases required intubation and mechanical ventilation. Additionally, 50% of the cases reported by Juárez-Lloclla et al [9] died. Hamad et al [14] believed that pneumopericardium might occur in patients with COVID-19 infection and could have no clinical manifestations. Nonspecific ST-segment or T-wave changes and low-voltage electrocardiograms could be detected in patients. They claimed that air leakage was the most common cause of pneumopericardium, which could require interventions if it was massive.

The salient point of the current case was that not only did he have no fever at hospital admission, but also his pneumopericardium manifested itself almost 14 days after COVID-19 pneumonia. Our case also had pericardial effusions and was evaluated for possible etiologies. This case highlights the potential severity of COVID-19. While the prognostic significance of such a rare finding is not known, pneumopericardium in COVID-19 infection should be considered in the appropriate clinical context, and it remains an area to be further elucidated. We also suggest that cardiologists pay attention to the possibility of pneumopericardium in cases with previous COVID-19 infection.

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