CASE REPORT

Seyma Tunc¹
Pinar Yıldız¹
Mertay Boran²

¹ Düzce University, Faculty of Medicine, Department of Chest Diseases, Düzce, Turkey
² Düzce University Faculty of Medicine, Department of Thoracic Surgery, Düzce, Turkey

Corresponding Author:
Seyma Tunc
Düzce University, Faculty of Medicine, Department of Chest Diseases, Düzce, Turkey
Phone: +90 3805421390/5591
mail: seyma1100@gmail.com

Received: 11.04.2021
Acceptance: 12.10.2021
DOI: 10.18521/ktd.912819

Rare Complications of COVID-19 Pneumonia: Pneumomediastinum and Atrial Fibrillation

ABSTRACT
Objective: Coronavirus disease 2019 (COVID-19) is considered as systemic disease involving many vital organs in addition to the lungs, including the heart, liver, and kidneys. Pneumomediastinum associated with COVID-19 pneumonia is a rare condition usually seen in patients with underlying lung pathology, infections, or mechanical intubation. We presented a case of late diagnosis of pneumomediastinum and atrial fibrillation in COVID-19 patient undergoing Noninvasive mechanical ventilation (NIMV).

Case: A 66-years-old male patient with a history of COVID-19 pneumonia and 19 days hospital follow up and a NIMV treatment was admitted to our emergency department with palpitations and dyspnea. Thoracic CT and electrocardiography revealed, scattered ground glass areas, pneumomediastinum and high-velocity atrial fibrillation, respectively.

Conclusion: We recommend regular checks with imaging methods and ECG during follow-up in patients with COVID-19. Clinicians should remember that ventilator-related lung damage may be seen in COVID-19 patients undergoing NIMV.

Keywords: COVID-19, Pneumomediastinum, Positive Pressure Ventilation, Atrial Fibrillation

COVID-19 Pnömonisinin Nadir Komplikasyonları: Pnönomediastinum ve Atriyal Fibrilasyon

ÖZET


INTRODUCTION

Coronavirus disease 2019 (COVID-19), which was reported for the first time at the end of 2019, is considered as systemic disease involving many vital organs in addition to the lungs, including the heart, liver, and kidneys (1,2). Complications such as hypoxemic respiratory failure is the most common conditions leading to intensive care unit (ICU) admission among hospitalized COVID-19 patients with pneumonia. A comprehensive literature review of studies involving patients with COVID-19 pneumonia revealed 15 cases of spontaneous pneumomediastinum, a rare condition usually seen in patients with underlying lung pathology, infections, or mechanical intubation (3, 4, 5). Here we presented a case of late diagnosis of pneumomediastinum and atrial fibrillation in COVID-19 patient undergoing noninvasive mechanical ventilation (NIMV).

CASE

The 66-year-old male patient had a known diagnosis of benign prostatic hyperplasia. He was a 20 pack years former smoker who had not smoked for 30 years. He had had no regular medication or substance use. Forty days prior to admission to the emergency department of our hospital, the patient tested positive for SARS-CoV-2 by real-time reverse transcription polymerase chain reaction and he had been hospitalized in the ICU for 6 days due to severe lung involvement observed via thoracic computed tomography (CT) (Figure 1) and the need for a noninvasive mechanical ventilation (NIMV). He had been discharged home with methylprednisolone treatment after 19 days of follow-up. Fifteen days after first discharge, he was readmitted to our emergency department (ED) with palpitations and dyspnea.

Figure 1. Thoracic CT images taken during the first hospitalization due to COVID-19 pneumonia, showed bilateral disseminated ground glass areas and normal mediastinal pathology.

Vital signs at the time of emergency admission were as follows: fever, 36.5°C; blood pressure, 138/68 mmHg; and oxygen saturation (sO2) 92% without external oxygen supplementation. Laboratory results were as follows: C-reactive protein, 6.5 mg/dL; white blood cell count, 10.5×103/μL; lymphocyte count, 1.43×103/μL; neutrophil count, 8.77×103/μL; d dimer, 0.43 g/mL; procalcitonin, 0.15 ng/mL; ferritin, >2000 ng/mL; troponin I, 0.41 ng/mL; aspartate aminotransferase, >891 IU/L; alanine aminotransferase, >1533 IU/L; and lactate dehydrogenase (LDH), 655 U/mL. Electrocardiography (ECG) revealed high-velocity atrial fibrillation (AF). Chest X ray (Figure 2) and Thoracic CT (Figure 3) were performed: The air collection was seen in the mediastinum and was diagnosed as pneumomediastinum. The scattered ground glass areas were observed in both lungs and were interpreted as compatible with pulmonary edema with COVID-19 sequelae. Echocardiography revealed moderate aortic regurgitation and minimal mitral insufficiency.

Figure 2. Chest X ray revealed a slight left deviation in the cervical trachea, increased mediastinal width and heart size, air around trachea and heart and bilateral disseminated ground glass areas.
Retrospective examination of X-ray findings indicated that no other radiological evaluations had been made during ICU follow-ups or at discharge, except for thoracic CT, which was performed 40 days before for a diagnosis of COVID-19.

Liver function disorder was considered to have occurred secondary to hypoxemia. Bilateral minimal pleural fluid observed on CT was interpreted as compatible with AF and volume overload.

The patient was prescribed bed rest, oxygen therapy, and rhythm-regulating amiodarone infusion followed by amiodarone maintenance therapy, and diuretic and anticoagulant therapy. Conservative follow-up, avoidance of NIMV applications, and X-ray follow-up were performed for pneumomediastinum. He was discharged after 9 days of follow up with sO2 of 96% in room with no cough or shortness of breath. The patient outpatient clinic control at 10th day after discharge was uneventful and Chest X ray (Figure 4) was in normal limits.

**DISCUSSION**

Pneumomediastinum, defined as the presence of free air in the mediastinum, is a rare condition most commonly observed in young, male patients (6,7). Pneumomediastinum can be spontaneous, iatrogenic, or traumatic. Mediastinal structures, such as the trachea and esophagus, in the mediastinum are covered with the mediastinal pleurae, which separate them from the pleural space. Although the pathophysiology of pneumomediastinum is unclear, air within the esophagus and trachea and air within the lung alveoli are potential sources of air for pneumomediastinum (7). In an experimental study in cats regarding the pathogenesis of pneumomediastinum performed in 1944, it was suggested that with the increase in alveolar interstitial pressure gradient, the air in the alveoli passes into the mediastinum through the perivascular and peribronchial sheaths and causes pneumomediastinum (8). In cases with only pneumomediastinum without passage of air into the pleural area, the lack of air collection in the bronchial walls or peribronchial area does not support the suggestion regarding air leakage secondary to alveolar rupture caused by alveolar pressure changes. It is thought that pneumomediastinum may occur when air is transported to a lower pressure environment between the soft tissues surrounding the mediastinal organs after an abnormal increase in pressure in the mediastinal region (7, 9). The development of pneumomediastinum has also been reported in excessive respiratory effort, such as coughing, vomiting, sneezing, defecation, and use of the voice at high volumes (7). During high NIMV or recurrent intense coughing attacks with high intratracheal pressure, air may pass through the separations in the pars membranacea fibers of the trachea and move toward the lower pressure mediastinal area, causing pneumomediastinum. Air in the mediastinum may develop as diffuse massive pneumomediastinum with air formation on all mediastinal surfaces or minimal air accumulation of 1 to 2 cm air. The development of massive pneumomediastinum is mostly seen in cases with long-term high NIMV treatment (7). Clinically, the
most common symptoms in pneumomediastinum are dyspnea and chest pain. Other symptoms include cough, hoarseness, swelling in the neck, nausea, swallowing difficulties, odynophagia, and rhinorrhea (6,7). Chest X ray after clinical suspicion is helpful in the diagnosis of pneumomediastinum, while a definite diagnosis is made with thoracic CT (6,7). Our patient was male and 66 years old, and had no additional symptoms other than shortness of breath.

Alveolar damage caused by the inflammatory response in COVID-19 has emerged as the reason for symptoms of respiratory distress, or the need for a NIMV treatment, or pneumomediastinum as a potential complication of the disease (10). The 22% of hospitalized patients diagnosed with COVID-19 may require invasive mechanical ventilation (IMV) and in 15% of these patients who required IMV, may develop barotrauma related pneumothorax, pneumomediastinum, and pneumopericardium in multiple time periods (11).

High peak serum levels of LDH are thought to be associated with alveolar damage (10). It has been hypothesized that severe disseminated alveolar damage causes interstitial emphysema and alveolar rupture resulting in the leakage of air through the bronchoalveolar sheath (5). In our patient, the LDH level was 655 U/L at the time of ED, but thoracic CT indicated no air collection in the bronchoalveolar sheath or peribronchial area. In our case, mediastinal emphysema invading around the cervical trachea, and its extension to the lower pericardial area suggested the development of massive pneumomediastinum caused by damage at the tracheal level.

Our case had no history of blunt or penetrating thoracic trauma to the chest, or underlying chronic respiratory disease. Among causes of pneumomediastinum, there is a history of barotrauma or volutrauma. In addition, it was understood that radiological imaging was not performed during the history of noninvasive mechanical ventilation NIMV use or until admission to the ED of our hospital, and it was thought that pneumomediastinum had been left undiagnosed for a long time.

Arrhythmia in patients with COVID-19 infection is complex and multifactorial, and has been suggested to be caused by metabolic disorders, hypoxia, acidosis, intravascular volume imbalances, and neurohormonal and catecholaminergic stress (12). AF has been reported as the most common type of arrhythmia in COVID-19 patients (13). Indeed, 27.5% of COVID-19 patients admitted to ICU in the USA develop atrial tachyarrhythmia, and 63% of these have AF (14).

In some cases of pneumomediastinum, low voltage values in ECG, nonspecific acute coronary syndrome changes, and ST-T changes in lateral precordial leads have been observed (14). AF was thought to have developed secondary to COVID-19 infection in our patient, who was hospitalized in the ICU without known heart disease, and the pneumomediastinum secondary to NIMV may have been a facilitating factor for AF.

**CONCLUSION**

Respiratory disturbances in patients with COVID-19 may not always be due to pulmonary embolism or worsening infection. We recommend regular checks with X-ray and intermittent ECG during follow-up in patients with COVID-19. Clinicians should take in mind that ventilator-related lung damage usually may be seen in COVID-19 patients undergoing NIMV.

**REFERENCES**