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Anorexia nervosa accompanied by bilateral pneumothoraces

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Abstract

Anorexia nervosa is an eating disorder that can be accompanied by various complications. An 18-year-old female patient, who was treated for 2 years due to anorexia nervosa, was admitted to our hospital because of bilateral pneumothoraces. A bilateral tube thoracostomy was performed, and she was diagnosed with idiopathic spontaneous pneumothorax and discharged. The patient was admitted to the hospital 9 months later due to respiratory complications. No increase was found in the patient's pneumothorax, but hypercarbic respiratory failure was discovered. During her treartment in our intensive care unit none of her cultures were positive. The patient was under non-invasive mechanical ventilation and monitored. She was intubated following an increase in parenchymal infiltrates and in the severity of her concurrent respiratory failure. The patient died due to ARDS on the 17th day of her hospitalization.

Keywords

acute respiratory distress syndrome; anorexia nervosa; bilateral pneumothoraces

Introduction

Eating disorders are becoming increasingly prevalent in outpatient clinics. These disorders may affect every organ and system in the body; however, the lungs are the less-commonly affected organs. In these cases, spontaneous pneumothorax, pneumomediastinum and emphysema are reported to develop frequently [1]. In this particular case study, the patient to be discussed was treated for 2 years for the diagnosis of anorexia nervosa, a tube thoracostomy was performed following the development of bilateral pneumothoraces, but the patient was transferred to the intensive care unit due to the deterioration of her general medical condition.

Case Presentation

The 18-year-old female patient was admitted to emergency room complaining of shortness of breath, respiratory disorder, and cough accompanied by expectoration. A postero-anterior chest X-ray showed bilateral pneumothoraces accompanied by infiltrations concordant to pneumonia. The patient was hospitalized in the chest diseases clinic, but then referred to the intensive care unit when her general medical condition deteriorated and her hypoxemic respiratory failure worsened in arterial blood gas. According to the patient's history, she had been treated for 2 years by the psychiatry department due to

her diagnosis of anorexia nervosa. She was a nonsmoker.

The patient first admitted to emergency room approximately 9 months ago, complaining of shortness of breath and cough. She was hospitalized due to pneumothorax observed in both of her lungs during examinations. The case was discussed with the chest surgery department and a tube thoracostomy was performed (Fig 1). Drainage was terminated when no increase was observed in the current pneumothorax in both lungs of the patient (Fig 2). Thoracic computed tomography showed bilateral pneumothoraces, which started at the upper lobe apical segment level in both hemithoraxes and continued to the superior lingula segment level on the left, and which scattered to the diaphragmatic region in the right middle lobe. (Fig 3,4), In the transbronchial biopsy obtained through fiberoptic bronchoscopy (FOB), minimal fibrotic thickening, edema and bronchial mucosa fragments were observed in the normal alveolar septa. Eating disorder-related pneumothorax was considered; the patient was discharged and received outpatient treatment. During follow-up treatments, no progression was observed in the pneumothorax, which was observed in the upper zones of both lungs.

9 months later when she readmitted to our emergency room and accepted to our respiratory intensive care unit, her general medical condition was poor and she was confused, dyspneic, tachypneic and tachycardic. Her appearance was quite cachectic and body mass index was 13 kg/m2. During the physical examination, it was observed that respiratory sounds were reduced in the upper areas of both lungs, and her expiration was longer. Bilateral and common coarse crackles were present during inspiration. From the cardiovascular system examination S1(+), S2(+), the following results were obtained: pulse: 142/min, arterial blood pressure: 100/60 mmHg, arterial oxygen saturation in room air: 87%, arterial blood gas values in room air: Ph: 7.29, PCO2:86 mmHg, PO2:60 mmHg, HCO3:37 mmol/L, PaO2/FiO2: 285. The patient was given non-invasive mechanical ventilation under BIPAP vision. IPAP 26 cmH2O, EPAP 6 cmH2O and FiO2 were started with 35%. Biochemistry analyses were as follows: Glucose 68 mg/dl; Urea 22 mg/dl; Creatinine 0.3 mg/dl; Uric acid 1.6 mg/dl; Total protein 6.6 g/dl, 2.8 g/dl; Calcium 9.2 mg/dl; Sodium 134 mmol/L; Potassium 4.5 mmol/L; Chlorine 98 mmol/L; Total bilirubin 0.7 mg/dl; AST 16 U/L; ALT 11 U/L; GGT 56 U/L; LDH 190 U/L; ALP 141 U/L; total cholesterol 71 mg/dl; triglyceride 78 mg/dl; HDL 16 mg/dl; LDL 39 mg/dl; VLDL 16 mg/dl; Iron 4 mmg/dl; UIBC 156 mmg/dl; CRP: 33 mg/L; WBC:14.9 103/mm3; neutrophile 10.5 103/mm3; haemoglobin 13.7 g/dl; haematocrit 41.3%. In the postero-anterior chest X-ray, it was observed that the pneumothorax line which was limited to the bilateral apical region had extended, and that common infiltrations and consolidation had developed in the lower right zone when compared to the X-ray from 9 months ago (Fig 5). Thorax computed tomography showed pneumothorax and common peribronchial infiltrations with bilateral ground-glass density were present in both lungs, and non-homogeneous consolidation including air bronchograms were present in the laterobasal and porterobasal segments of the right lung's lower lobe (Fig 6,7). The patient's clinical condition deteriorated and confusion increased, and the following results were obtained from arterial blood analysis: Ph: 7.02, PCO2: 129 mmHg, PO2: 84 mmHg, HCO3: 33.5 mmol/L, sat O2: 87.5% FiO2: 100%, PaO2/FiO2: 84. She was then intubated based on these results. In the pressure control mode, she was supported with PEEP: 8 cmH2O, Above PEEP: 24 cmH2O FiO2 : 40%, respiratory rate 14. In her echocardiography, a pericardial effusion of 9.5 mm was detected in the left ventricle posterior, which surrounded the heart entirely. Her ejection fraction was measured as 60–62%. In the etiological analyses, immune globulin levels were normal, P-ANCA and C-ANCA were negative, and

negative, and rheumatoid level was normal. Blood, urine or deep tracheal aspirates cultures were negative. Sputum and lavage were negative in terms of acid fast bacilli. The patient's clinical condition deteriorated rapidly, bilateral infiltrations increased (İmage 8), ARDS and shock developed and respiratory failure worsened; as a result, multi-organ failure developed. The patient didn't respond to interventions and died on the 17th day of her hospitalization.

Discussion

Anorexia nervosa is an eating disorder that can be accompanied by various complications. It has been reported to affect gastrointestinal, cardiovascular, hematologic, endocrine, renal, neurological and dermatological systems [1]. The respiratory system may be affected as well, but its pathophysiology is not yet definite. In animals, it has been determined that long-term fasting leads to a decrease in total lung protein, connective tissue, hydroxyproline and elastin levels [2]. Emphysema is also believed develop in this manner, but this damage is defined as emphysema-like rather than emphysema.

After administering tube thoracostomy to the patient who developed bilateral spontaneous pneumothorax, clinical recovery was achieved but total expansion wasn't obtained radiologically. The patient was consulted by the chest surgery team, tube thoracostomy was ended and the patient received outpatient follow-up treatment. Following discharge, the patient was admitted for two follow-ups treatments at one-month intervals. It was observed that she had no complaints and that the pneumothorax line hadn't extended. The patient didn't attend the subsequent follow-ups, but was admitted to the intensive care unit in the 9th month due to deterioration in her general medical condition and development of respiratory failure even though the pneumothorax line hadn't extended. Thorax computed tomography showed no increase in bilateral pneumothorax, whereas bronchiectasis and fibrosis had developed as well as consolidation. No pathology was observed in the patient that would lead to apical bulla or pneumothorax. The patient's condition was considered as secondary to malnutrition, and associated with her recovery's delay and prevention. Even though a nutrition program (intravenous liquid, blood products, parenteral and enteral nutrition) was initiated for the patient, no improvement was observed in the general condition and oxygenation parameters. Biffl et al. stated that treatment with VATS was successful following the long-term leak in the patient with anorexia nervosa and pneumothorax; as mentioned, structural changes occur in the lung of patients with anorexia nervosa and thus lead to a decrease in surfactant growth and a limited recovery of the lungs [1]. Corless et al. stated that bilateral tube thoracostomy was performed in patients with anorexia nervosa and bilateral spontaneous pneumothorax, and that a bilateral thoracoscopic pleurectomy was performed to prevent recurrence. In patients with anorexia nervosa, alveolar ruptures are thought to emerge from any condition (such as coughing, vomiting) in which the alveolar structure weakens and intrathoracic pressure increases [2]. In a case of anorexia nervosa reported by Danzer et al., and also Lee et al., pneumomediastinum, pneumothorax and subcutaneous emphysema were observed. Pneumomediastinum, pneumothorax and subcutaneous emphysema are rarely seen together. It is thought that the patient's vomiting anamnesis caused the intraabdominal and intrathoracic pressures to increase and the condition to occur even if the patient is not vomitting [3,4]. Hochlehnert et al. reported a patient with anorexia nervosa who developed spontaneous pneumothorax, and highlighted that anorexia nervosa caused pneumomediastinum more commonly than pneumothorax. This is a rare

condition that is generally self limited; its treatment lies in providing analgesia in addition to conservative treatment [5]. Malnutrition is reported to be associated with emphysema, bulla and bronchiectasis development, and there is a correlation between BMI and tomography measurement of emphysema [6]. In patients with anorexia nervosa, it is believed that peripheral intrapulmonary air leaks progress in a retrograde fashion: the air is dissected in the interstitial area along the bronchial tree, and eventually leads to wide mediastinal, retroperitoneal and subcutaneous emphysema even without smoking , because of this diffision capacity for oxygen decreases [7]. Generally, supplemental treatment and weight gain are expected to lead to spontaneous recovery in most cases.

Limited and little recovery of our patient led to deterioration of her general medical condition, and deterioration of her general medical condition led to decreased recovery. The patient, whose pulmonary functions decreased over time and whose respiratory muscles appeared to have weakened, was intubated. In the control chest X-ray, consolidation from hilus to periphery was detected in both lungs, and it was believed that surfactant deficiency-related ARDS had developed in the patient. The patient didn't respond to treatment and died.

Conclusion

Anorexia nervosa can affect all systems in the body, including the respiratory system and lungs. Spontaneous pneumothorax is one of the complications associated with this disease. Long-term and severe malnutrition may limit recovery and even make it irreversible. Emphysema-like destruction and surfactant deficiency were reported in these patients. As in our case, the general medical condition may deteriorate, the damage may be irreversible, and the patient may die. Such complications must be considered in patients with anorexia nervosa, and early nutrition and treatment programs must be initiated.

Figures

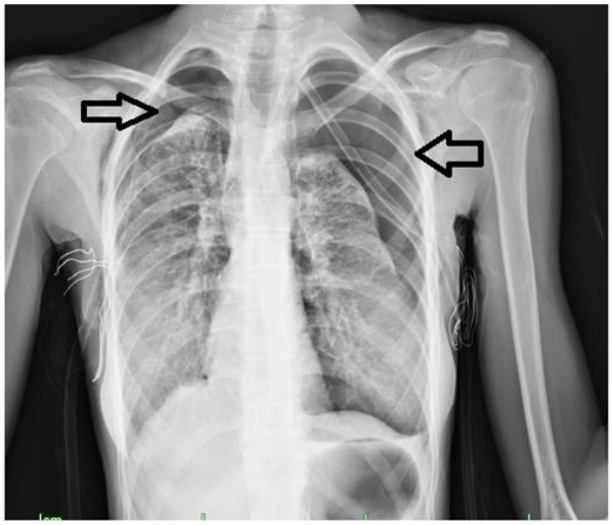


Figure 1: Chest X-ray of the patient taken during first hospitalization showing bilateral pneumothoraces and performed bilateral tube thoracostomy. Pneumothoraces are shown by bold arrows.

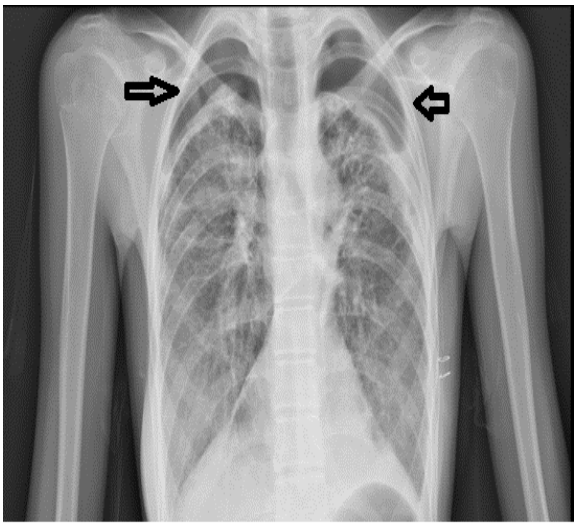


Figure 2: Chest X-ray of the patient at the time of drain removal, pneumothoraces in both lungs didn't resolve completely but there was no increase in pneumothoraces. Pneumothoraces are shown by bold arrows.

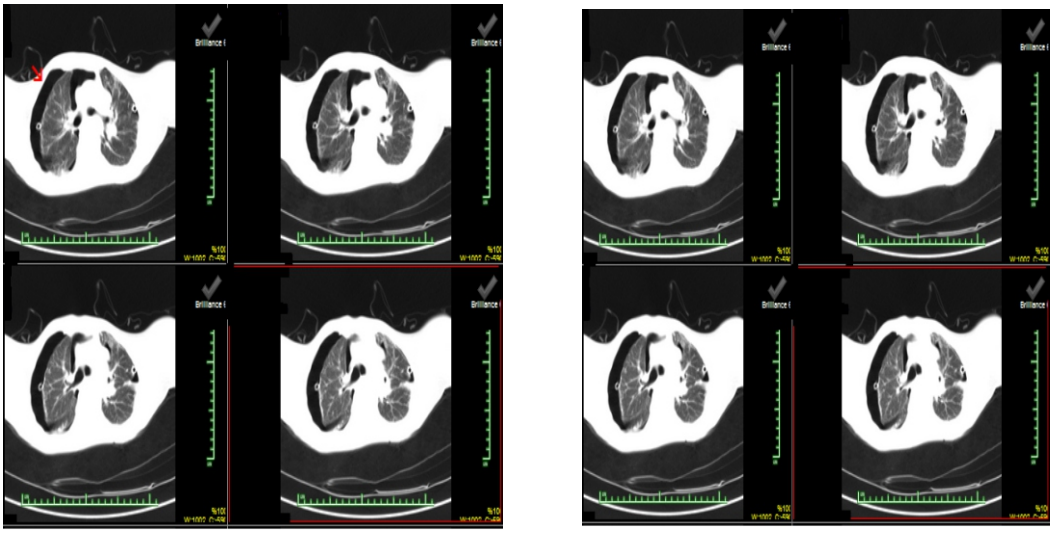
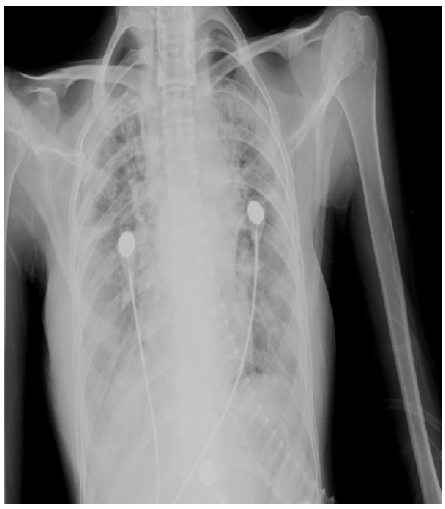


Figure 3,4: Chest computed tomography taken during the patient's first hospitalization showing bilateral pneumothoraces. Pneumothoraces are shown by arrows.



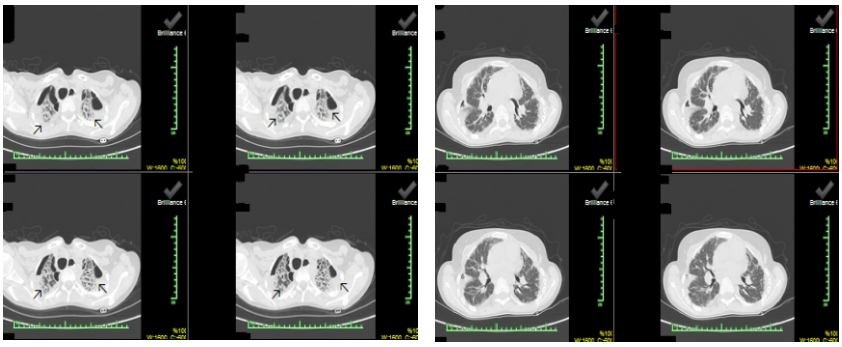


Figure 6,7: Chest computed tomography of the patient 15th day in the respiratory intensive care showing pneumothorax and infiltrations with bilateral ground-glass density with non-homogeneous consolidation in the right lung's lower lobe

Figure 5: Chest X-ray of the patient taken third day in the respiratory intensive care unit in second hospitalization after 9 months showing bilateral pneumothoraces progression and new infiltrations.

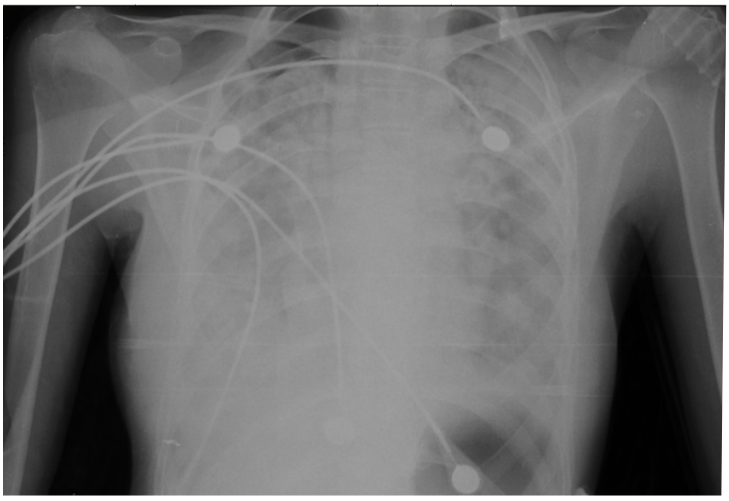


Figure 8: Chest X-ray of the patient taken 15th day in the respiratory intensive care unit showing bilateral infiltrations resulted in ARDS 2 days before patient was deceased.