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Case Report

Large Pneumothorax Associated with Mechanical Ventilation in Patients with Chronic Respiratory Disease

Alireza Rahat Dahmardeh[1], Aliakbar Keykha[2,3]*

[1]Department of Anesthesiology and Critical Care, School of Medicine, Zahedan University of Medical Sciences, Zahedan, Iran.

[2]Community Nursing Research Center, Zahedan University of Medical Sciences, Zahedan, Iran.

[3]Department of Medical-Surgical Nursing, School of Nursing and Midwifery, Zahedan University of Medical Sciences, Zahedan, Iran.

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ABSTRACT

Difficult intubation and mechanical ventilation are common causes of pneumothorax in critically ill patients. Patients with chronic respiratory tract disease are at high risk for pneumothorax caused by positive-pressure mechanical ventilation. A timely diagnosis of pneumothorax is critical as it may evolve into tension pneumothorax. In this case report, we report a patient who has a decreased level of consciousness due to opioid overdose. In the emergency room, she underwent mechanical ventilation and became conscious after a naloxone injection. After extubation, she has extensive subcutaneous emphysema. However, her hemodynamic and respiratory status is normal. The chest CT scan showed a large pneumothorax caused by mechanical ventilation.

Introduction

Pneumothorax related to mechanical ventilation (PRMV) has a high mortality rate. PRMV often occurs in the early stage of mechanical ventilation. Underlying pulmonary disorders are associated with a high incidence of PRMV [1]. Chronic obstructive pulmonary disease, asthma, pneumonia, and acute respiratory distress syndrome (ARDS) are common disease that increases the risk of PRMV [2]. The mechanical ventilation positive pressure leads to the rupture of alveoli and air leakage into the pleural space [3]. In patients under mechanical ventilation, a decrease in tidal volume, worsening hypoxia, tachycardia, hypotension, decreased breath sounds on the lung involved, tracheal deviation, subcutaneous emphysema, and an increase in airway pressures (peak and plateau) may be a sign of pneumothorax. However, these are not specific symptoms of pneumothorax. Each patient's symptoms may differ according to comorbidity, diseases, and underlying problems [4]. Early diagnosis and treatment of PRMV is essential. Continuous air leakage caused by the ventilator's positive pressure leads to the development of tension pneumothorax. Tension pneumothorax is a dangerous disorder that causes cardiovascular collapse and increases the mortality rate [5].

Case Report

The patient was a 51-year-old woman whose husband brought her to the emergency room due to a decreased level of consciousness. The patient's husband stated that his wife had been using opium for many years. She also has a history of chronic respiratory disease for the past five years and is being treated with salbutamol spray, Seretide spray, and montelukast tablets. The patient's level of consciousness was 8 on the Glasgow Coma Scale (GCS). In the examination, the pupils were miotic, BP

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*Corresponding author.
E-mail address: aliakbar.keykha@gmail.com

was 90/50 mmHg, HR was 45 beats/min, RR was 6 breath/min, Spo_2 was 60%, and T was 36/5 °C. Due to the decreased level of consciousness and reduced Spo_2, the patient was immediately transferred to the resuscitation room. First, she was hyperoxygenated with a Mapleson breathing system. Endotracheal intubation was performed with an ETT size 7, which was difficult due to the anterior position of the trachea and the short neck of the patient (Mallampati score 3). Mechanical ventilation of the patient was started with AC/PRVC mode with tidal volume (TV): 450 CC, respiratory rate (RR): 12/min, inspiratory time (IT): 1.2 sec, positive end-expiratory pressure (PEEP): 5 cm H_2O, and FIO_2: 100%. The patient's blood glucose level was 80 mg/dL with a glucometer. Due to the history of opium addiction and overuse of opium, naloxone injection was done with caution for the patient (An ampoule of 0.4 mg of naloxone was diluted with 4 cc of sterile water and 1 cc of the solution injected each time). By injecting 0.8 mg of naloxone, the patient became conscious. Therefore, the infusion of 0.4 mg of naloxone per hour with N/S serum was continued for the patient. After two hours, the patient became fully conscious, and the hemodynamic and respiratory status was stable. The arterial blood gas analysis also showed that the patient's breathing was acceptable. ABG (PH= 7/39, $PaCO_2$= 36/8, HCO_3= 23/5, BE= 1, PaO_2= 88, O_2Sat= 97). Therefore, the patient was extubated. Due to the history of chronic respiratory disease and decreased level of consciousness, chest CT was performed for the patient to check the pulmonary condition and the possibility of aspiration pneumonia. Chest CT showed ground glass opacities with atelectasis foci in both lungs' middle and upper regions, which suggested chronic bronchiolitis (Figure 1).

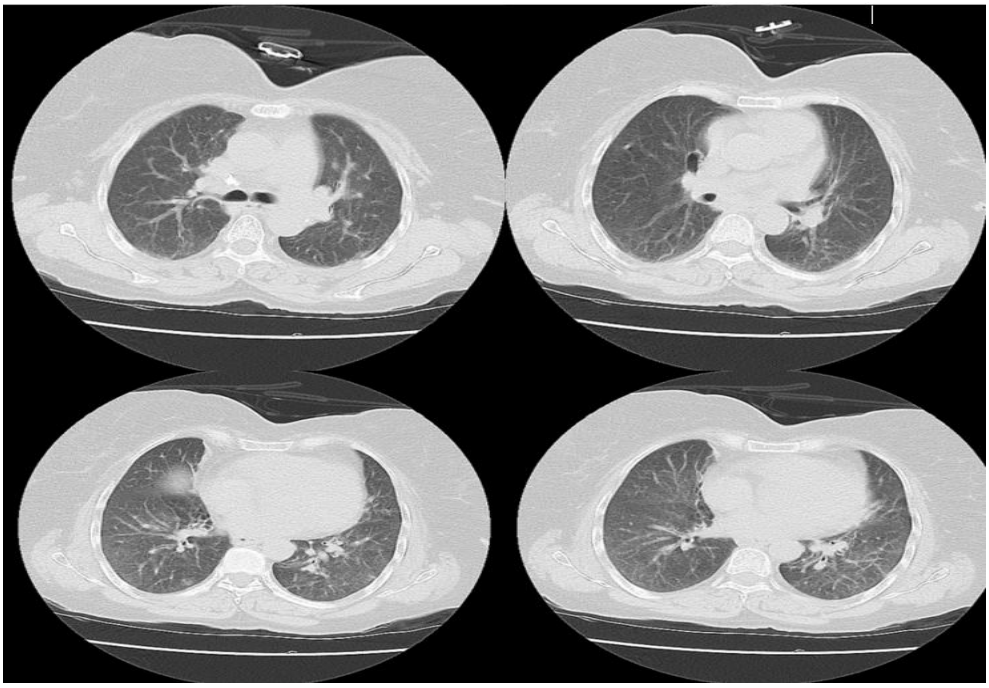

Figure 1- A chest CT showed ground glass opacities with consolidation spots in the middle and upper regions of both lungs

Therefore, the patient was observed in the emergency room to make sure that she did not lose consciousness again under the influence of opium.

After 4 hours, the patient's lab results were reported, which showed only leukocytosis, and the other results were normal.

WBC=16/9×10^3 /mm, RBC=4/4 mil/mm, HB=13/9g/dl, HCT=43/3%, PLT=217×1000, PT=13/sec, PTT= 40/sec, INR= 1/3, BS= 86 mg/dl, BUN= 16 mg/dl, CR=1 mg/dl, K=4/5 meq/l, Na=141 meq/l, AST=29 U/L, ALT= 17 U/L, ALKP= 251 U/L, BIL total= 0/46 mg/dl

Based on the history of respiratory disease, opacities in the chest CT, and leukocytosis, the patient's drug treatment was started as follows: Cap Azithromycin 500 mg the first day and then 250 mg daily for 4 days, Amp Ceftriaxone 1g BID, Spray Salbutamol 4 puff QID, Spray Atrovent 4 puff QID, N-acetylcysteine 1g BID, and Pantoprazole 40mg daily.

In the patient's re-examination, we found that extensive subcutaneous emphysema had developed in the patient's head, neck, and chest. However, the patient's respiratory and hemodynamic status was utterly stable (BP= 110/70 mmHg, HR=85/min, RR=14/min, Spo_2=95%, and T=37/5 °C).

Brain CT and chest CT were performed for the patient, which showed extensive bilateral pneumothorax and extensive subcutaneous emphysema from the patient's head to abdomen (Figure 2,3).

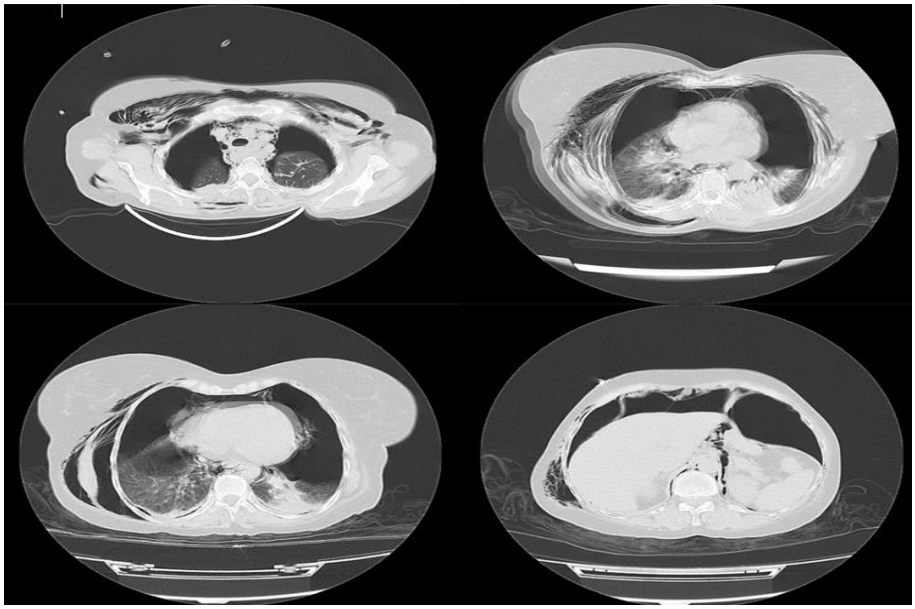

Figure 2- A chest CT of the patient showed severe bilateral collapse of the lung, severe bilateral pneumothorax, pneumomediastinum, pneumoperitoneum, extensive subcutaneous emphysema, and consolidation of the base of the lungs in the context of lung infection or aspiration.

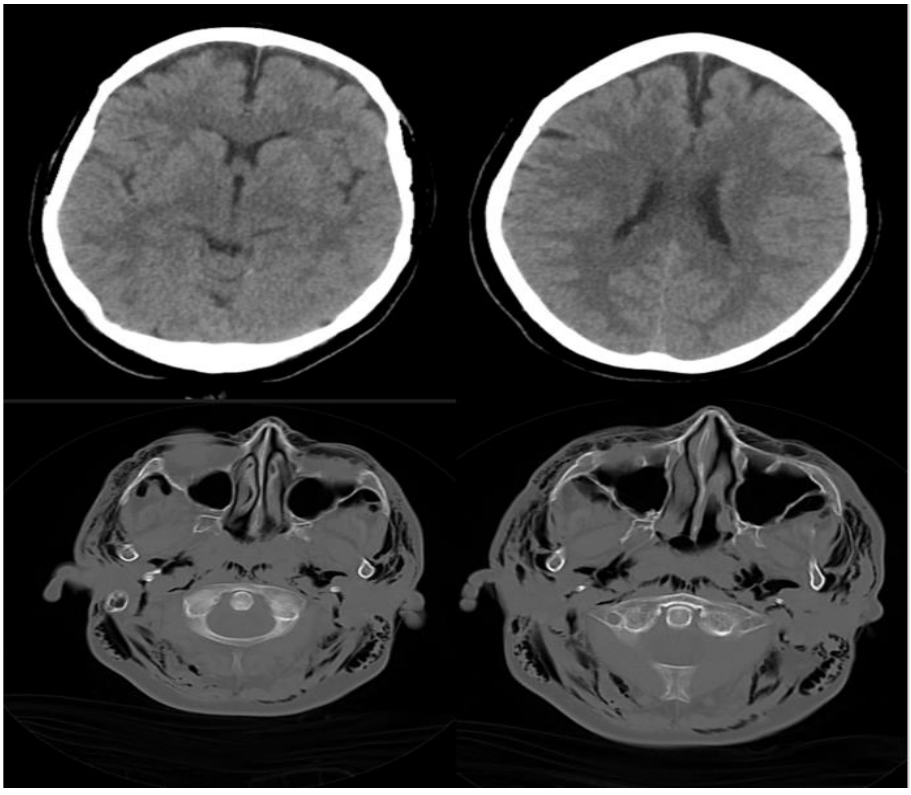

Figure 3- A brain CT scan of the patient showed the normality of the parenchyma and bone structures and extensive subcutaneous emphysema of the soft tissue of the head.

Due to the stability of the patient's respiratory and hemodynamic status, arterial blood gas analysis was performed again to check for hypoxemia, which was as follows: PH=7/30, $PaCO_2$=49/3, HCO_3=20/5, BE=1, PaO_2=75, O_2Sat=93/7. Therefore, the patient was transferred to the ICU to be monitored. ABG was performed every 6 hours. In the first 6 hours after admission, the patient did not have any symptoms of respiratory distress, and ABG was also acceptable (PH=7/31, $PaCO_2$=45/8, HCO_3=22/5, BE=-3/8,

PaO_2=80/4, O_2Sat=95/7). However, the ABG performed 12 hours after hospitalization showed severe hypoxemia and mixed acidosis (PH= 7/1, $PaCO_2$= 85, HCO_3=18/6, BE=-6/2, PaO_2=40/4, O_2Sat=55). Therefore, chest CT was performed again for the patient. Chest CT showed an increase in the amount of pneumothorax (Figure 4).

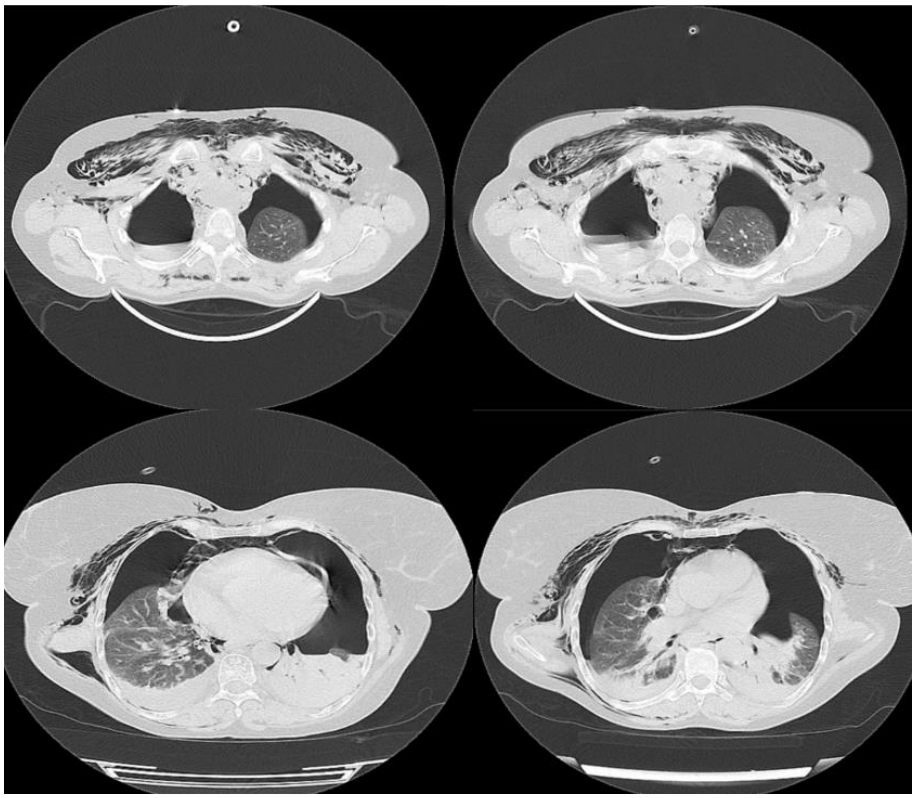

Figure 4- A chest CT of the patient showed severe bilateral collapse of the lung and bilateral hydrothorax, pneumothorax, Pneumomediastinum, extensive subcutaneous emphysema, and consolidation of the base of the lungs.

After being transferred from the radiology department to the ICU, the patient experienced severe respiratory distress. According to the chest CT evidence and respiratory distress, the surgeon placed a double-sided chest tube for the patient (Figure 5). However, the patient's respiratory distress did not improve. Therefore, after injecting 200 mcg of fentanyl, 5 mg of midazolam, and 40 mg of propofol, the patient was intubated with a 7.5 tracheal tube and was placed under mechanical ventilation with AC/PRVC mode with tidal volume (TV): 400 CC, respiratory rate (RR): 16/min, inspiratory time (IT): 0.9 sec, positive end-expiratory pressure (PEEP): 3 cm H_2O, and FIO_2: 100-40% based on the spo_2 level.

After insertion of the chest tube, different amounts (between 300 and 600 cc) of purulent secretions were removed from the patient's right chest tube daily. From purulent secretions, the sample was sent for culture. In the secretion culture, Escherichia coli was reported to be sensitive to trimethoprim/sulfamethoxazole and resistant to cephalosporins, aminoglycosides, quinolones, ampicillin/sulbactam, carbapenems and piperacillin/tazobactam. Therefore, this drug was also added to the patient's antibiotics. Due to the need for long-term hospitalization in the intensive care unit, a nasogastric tube was also inserted for the patient. From the second day after hospitalization, the gavage was started with the minimum required amount (50 cc every 3 hours), and based on the patient's tolerance, it was increased to 120 cc per hour. We also gavage the patient's opium according to the previous schedule to prevent withdrawal syndrome.

Fourth days after admission, the patient was fully conscious and had an acceptable respiratory condition. Also, the respiratory variables measured by the ventilator were suitable. Therefore, the patient was placed in spontaneous breathing mode. Then, we tried to wean the patient from the ventilator by spontaneous breathing trial with a T-piece. However, every time, the patient had tachypnea and decreased spo_2 and could not tolerate weaning. Therefore, we performed a chest CT for the patient again to investigate the cause of the weaning intolerance, which showed increased pleural effusion on the right side of the chest. However, the amount of pneumothorax, pneumomediastinum, pneumoperitoneum, and subcutaneous emphysema decreased compared to the previous CT (Figure 6).

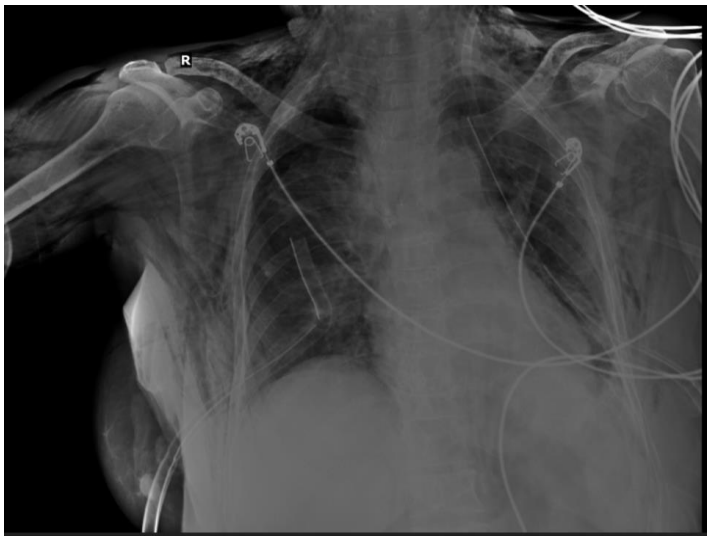

Figure 5- A CXR of the patient showed the expansion of the lung parenchyma after bilateral chest tube insertion and extensive subcutaneous emphysema.

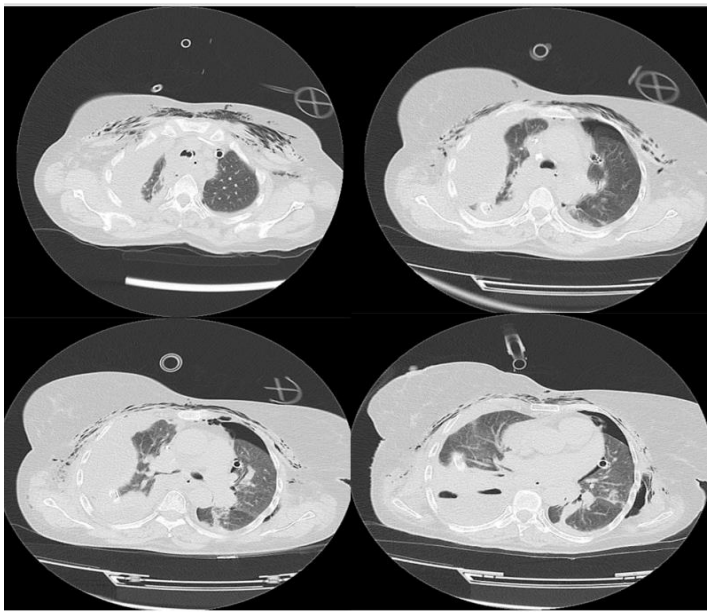

Figure 6- A chest CT of the patient showed mild hydrothorax and pneumothorax on the left side with evidence of contusion in the path of the chest tube tip, severe pleural effusion on the right side in the form of loculated with internal air lucency (pneumothorax), collapse consolidation in the lower lobe (RLL) of the right lung, pericardial effusion, pneumoperitoneum, pneumomediastinum, pneumothorax and subcutaneous emphysema (their value has decreased compared to the previous CT scan (Figure 2,4).

Therefore, surgical consultation was done again. Due to the increase in pleural effusion in the right lung and the bent tip of the right chest tube, the surgeon decided to insert another chest tube on the right side of the chest to facilitate secretion drainage (Figure 7). The drainage of purulent secretions from the two chest tubes on the right side of the chest continued, and the chest X-rays did not show any improvement in the patient's lung condition. However, the patient did not have a fever or leukocytosis. So, on the seventh day after hospitalization, the secretions were removed from the chest tube, pulmonary secretions, blood, and urine samples were sent for culture, and we empirically changed the patient's antibiotic treatment to piperacillin-tazobactam 3.375g QID, vancomycin 1gr BID and levofloxacin 750mg daily. The laboratory delivered the results of the cultures of the patient after 48 hours. The results of the patient's blood and urine cultures were negative. However, the results of lung secretions and chest tube secretions cultures were reported of Pseudomonas aeruginosa sensitive to piperacillin/tazobactam. The patient's antibiotic regimen continued for 7 days. Then, to re-examine the lung

problems, a chest CT was performed again so that if the pulmonary condition was better than before, the patient could be weaned from the ventilator. Due to full consciousness, the patient was ventilated from the fourth day after hospitalization in a spontaneous mode with PEEP=5 cmH_2O, PS=5 cmH_2O, and FIO_2=40%. However, the conditions of the lungs were not suitable for weaning from the ventilator (Figure 8).

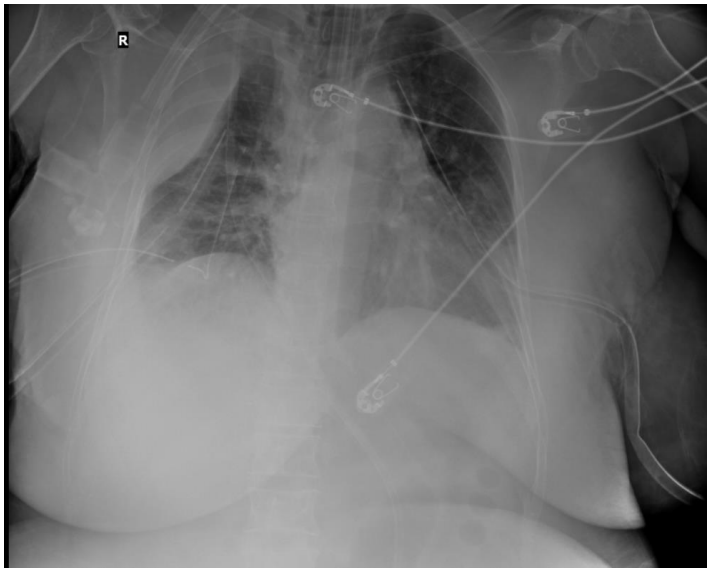

Figure 7- The patient's chest X-ray showed pleural effusion, bilateral consolidation at the base of both lung and placement of two chest tubes on the right side of the chest.

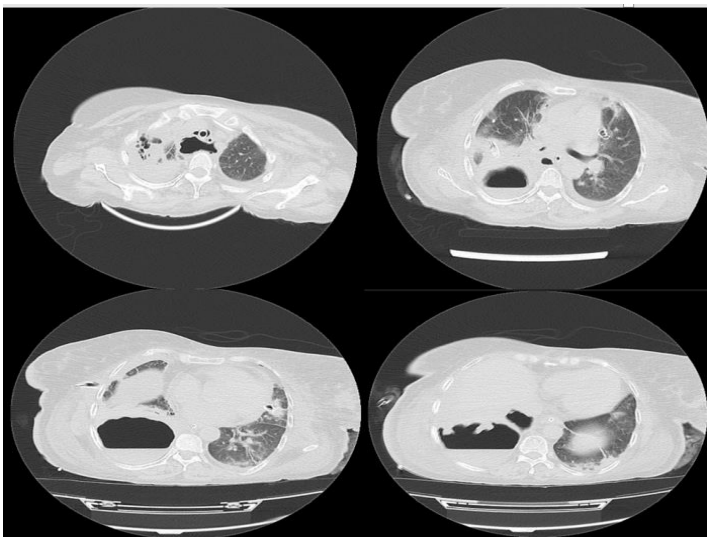

Figure 8- A chest CT of the patient showed pneumomediastinum in the upper part of the mediastinum, loculated pleural effusion with several foci of air in the upper part of the right lung, severe hydrothorax and pneumothorax in the right lower lobe (RLL), and evidence of consolidation in the lower part of the left lung in the context of aspiration pneumonia.

Therefore, the patient was tracheostomy by percutaneous dilatational tracheostomy (PDT) method with local anesthesia and injection of 200 mcg of fentanyl, and mechanical ventilation of the patient continued with the spontaneous mode. Due to the non-removal of the consolidation and the non-reduction of the chest tube secretions, another sample was sent for culture of the lung secretions and chest tube. Samples were also sent from lung and chest tube secretions for tuberculosis diagnosis. AFB smear was negative for TB. However, a lung secretions culture was reported of Klebsiella pneumoniae sensitive to colistin, and a culture of chest tube secretions was reported of Klebsiella pneumoniae and Pseudomonas aeruginosa sensitive to colistin. Therefore, colistin 3mil units TDS was added to the patient's antibiotic combination. After the start of colistin, the amount of lung secretions and purulent secretions coming out of the chest tube decreased a lot. Therefore,

the surgeon removed one of the right chest tubes and the left chest tube. In the following days of hospitalization, we noticed that the color of right chest secretions changed and was the same color as the patient's gavage solution. Therefore, we NPO the patient and asked for a gastroenterologists and Pulmonologist consultation for further investigation and to diagnose the cause. Because of the patient's NPO, it was not possible to gavage the patient's opium, so we started the morphine infusion at 1-3 mg/h. However, the patient became restless due to the withdrawal syndrome and pulled the nasogastric tube and removed it. With the gastroenterologist's opinion, we again inserted an NG to decompress the stomach and prevent more digestive secretions into the lungs. But the tube was not placed easily and we could not hear the sound of air blowing on the stomach region with the stethoscope. Therefore, to confirm the placement of the tube, we performed a chest X-ray, which showed that the tube was twisted in the patient's esophagus (Figure 9).

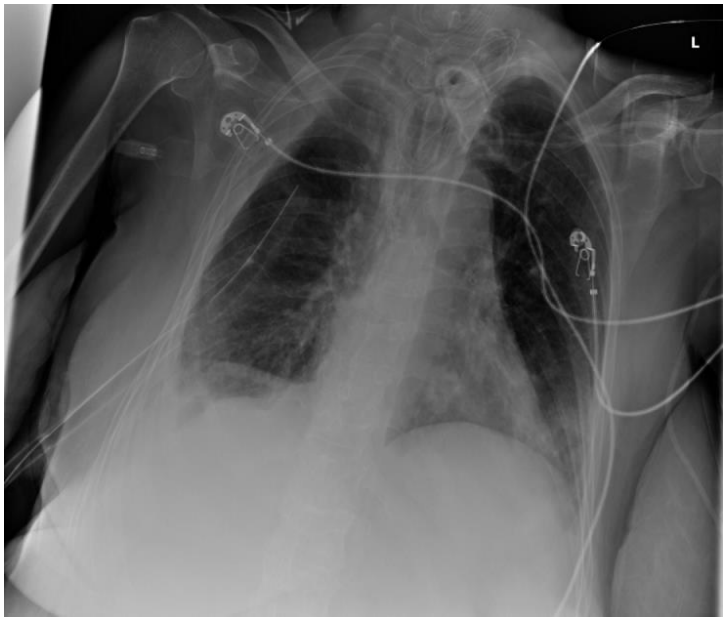

Figure 9- The patient's chest X-ray showed the twisting of the nasogastric tube inside the esophagus.

Therefore, the nasogastric tube was removed and scheduled for emergency endoscopy. In the endoscopy, a perforated ulcer was seen in the lower third of the esophagus. The esophagus was obstructed after the ulcer, and passing the endoscope into the stomach was impossible. Therefore, a biopsy was taken from the wound. In the pathological examination, no malignancy was observed, and only an inflammatory and necrotic tissue caused by the esophageal ulcer was reported (Figure 10).

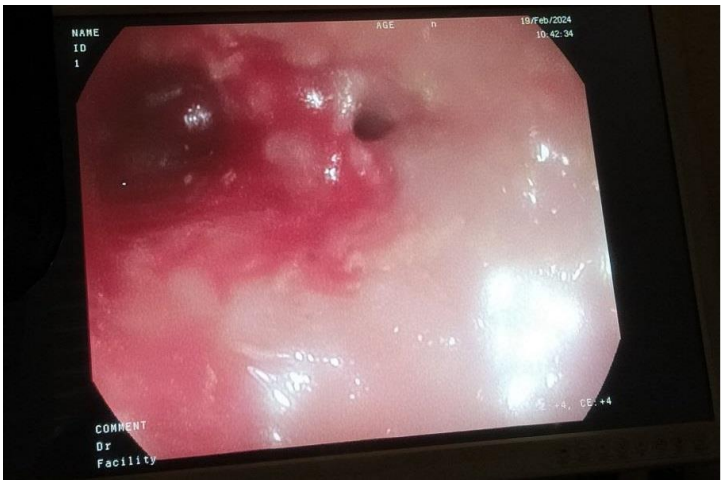

Figure 10- Endoscopic image of the esophagus showed perforation of the esophagus and obstruction of the lower part of the esophagus

In the following days of hospitalization, the patient developed hemodynamic instability; we could not stabilize the patient's hemodynamic status with norepinephrine and dopamine infusion. Due to severe hemodynamic instability, it was impossible to perform a bronchoscopy. Then, the patient's consciousness decreased, and the patient suffered from sepsis and multiple organ dysfunction syndrome. Therefore, we had to switch the patient ventilation mode to AC/PRVC. Cardiologist, infectiologist, and nephrologist consultation were done to adjust the dosage of drugs. However, none of the treatment measures were adequate for the patient's recovery. Finally, the patient suffered cardiac and respiratory arrest. Cardiopulmonary resuscitation was ineffective, and the patient died.

Discussion

The treatment process of this case showed that the rupture of alveoli due to mechanical ventilation in the context of chronic lung disease causes the spread of organisms and infections inside the lung into the thorax space. Also, alveolar rupture causes permanent air leakage, and pneumothorax does not resolve despite using a chest tube. These complications lead to poor outcomes or patient death. The alveolar rupture caused by mechanical ventilation occurs along the vascular sheath, and the dissection of air passes through the mediastinum, subcutaneous tissue, and retroperitoneum. Therefore, it may manifest as pulmonary interstitial emphysema, pneumomediastinum, pneumoperitoneum, or subcutaneous emphysema. Other manifestations of pneumothorax related to mechanical ventilation (PRMV) include tachycardia, chest pain, tachypnea, agitation, hypotension, cyanosis, and mental status change. Tachycardia is the most common symptom [1]. Therefore, a patient with a history of underlying lung disease must be evaluated for any symptoms after undergoing mechanical ventilation [3]. If the amount of air leakage is small in the early stages of pneumothorax, the physical examination and vital signs may be normal [6]. In another study, it was reported that five patients with COVID-19 developed pneumothorax after mechanical ventilation. Of these 5 patients, 2 were mechanically ventilated with PC-AC, 2 with VC-AC, and one with PRVC-AC. Therefore, it is possible to create pneumothorax related to mechanical ventilation with all pressure, volume, and dual modes. Similar to the patient examined in the present study, in this study, 3 of the five patients examined died, which showed that the outcome of pneumothorax related to mechanical ventilation is poor when it is accompanied by an underlying lung disease [7]. In another case report, pneumothorax related to mechanical ventilation was reported in a 54-year-old patient with a hemorrhagic stroke. The remarkable point of this case was that the symptoms caused by pneumothorax (decreased TV and dropped Spo_2 level) were visible only if the patient was turned to the right lateral decubitus. The mode of mechanical ventilation in the reported patient was PCV, with a peak pressure of 15 cmH_2O, a respiratory rate of 14 breaths per minute, and positive end-expiratory pressure (PEEP) of 9 cmH_2O [8]. This case is contrary to the traditional belief that the risk of barotrauma is higher in volume ventilation modes than pressure ventilation modes. Because despite the use of pressure control mode with low airway pressure and the absence of underlying lung disease, pneumothorax related to mechanical ventilation has occurred. We used the assist control/pressure-regulated volume-controlled ventilation (AC-PRVC) mode in the present case. In addition to assuring that the patient receives a sufficient volume, this mode of mechanical ventilation also reduces the risk of barotrauma. This mode of mechanical ventilation is better than volume modes for preventing barotrauma in patients with underlying lung disease [9]. Tidal volume (VT) and intrinsic positive end-expiratory pressure (PEEPi) are two critical factors of lung hyperventilation and increase the risk of barotrauma [10]. In the present patient, there was no high risk for pneumothorax related to mechanical ventilation due to the use of PRVC mode, low PEEP, tidal volume 7 mL/kg of IBW, and very short mechanical ventilation time. We tried to reduce the risk of barotrauma and, thus, pneumothorax by using a lung-protective mechanical ventilation strategy. However, this strategy was ineffective, and the patient developed pneumothorax related to mechanical ventilation.

Other studies have mentioned that the incidence of barotrauma was not lower when lung-protective ventilator strategies were used; however, there is a higher incidence of barotrauma when the plateau pressure rises above 35 cmH_2O [11-12]. Low tidal volume ventilation is essential in patients at higher risk for barotrauma, such as patients with ARDS, COPD, asthma, Pneumocystis jiroveci pneumonia (PJP), and chronic interstitial lung disease (ILD) [11,13].

Conclusion

Lung Protective strategy in mechanical ventilation cannot prevent barotrauma in patients with underlying lung disease. Therefore, if the patient is under mechanical ventilation, they should be examined for pneumothorax symptoms.

Ethical Considerations

The Zahedan University of Medical Sciences research ethics committee approved the present case report by the ethics code of IR.ZAUMS.REC.1403.124.

Consent

We received informed consent from the patient's wife to publish this case report, provided that we do not mention the patient's demographic characteristics in any part of the case report.

Abbreviations

PRMV: Pneumothorax Related to Mechanical Ventilation
ARDS: Acute Respiratory Distress Syndrome
°C: Degree Celsius
ETT: Endotracheal Tube
AC/PRVC: Assist-Control/Pressure Regulated Volume Control
ICU: Intensive Care Unit
IBW: Estimated Ideal Body Weight in (kg)
TV or VT: Tidal Volume
PCV: Pressure Control Ventilation
PC-AC: Pressure Control – Assist Control
VC-AC: Volume Control- Assist Control
COPD: Chronic Obstructive Pulmonary Disease
TB: Tuberculosis
AFB: Acid- Fast Bacilli

References

[1] Hsu CW, Sun SF. Iatrogenic pneumothorax related to mechanical ventilation. World J Crit Care Med. 2014; 3(1):8-14.

[2] de Lassence A, Timsit J-F, Tafflet M, Azoulay É, Jamali S, Vincent F, et al. Pneumothorax in the Intensive Care Unit: Incidence, Risk Factors, and Outcome. Anesthesiology. 2006; 104(1):5-13.

[3] Thachuthara-George J. Pneumothorax in patients with respiratory failure in ICU. J Thorac Dis. 2021; 13(8):5195-204.

[4] Rankine JJ, Thomas AN, Fluechter D. Diagnosis of pneumothorax in critically ill adults. Postgrad Med J. 2000; 76(897):399-404.

[5] Roberts DJ, Leigh-Smith S, Faris PD, Blackmore C, Ball CG, Robertson HL, et al. Clinical Presentation of Patients With Tension Pneumothorax: A Systematic Review. Annals of Surgery. 2015; 261(6):1068-78.

[6] Rastegari H, Heidari M, Keykha A. Traumatic Tension Pneumothorax with Extensive Subcutaneous Emphysema. Arch Anesthesiol Crit Care. 2024;

[7] Akdogan RE, Mohammed T, Syeda A, Jiwa N, Ibrahim O, Mutneja R. Pneumothorax in Mechanically Ventilated Patients with COVID-19 Infection. Case Rep Crit Care. 2021; 2021(1) 6657533.

[8] Mayahara T, Katayama T, Higashi Y, Asano J, Sugimoto T. Position-Dependent Symptoms of Pneumothorax During Mechanical Ventilation: A Case Report. Cureus. 2023; 15(12):e50820.

[9] Ali AA, El Wahsh RA, Agha MA, Tawadroos BB. Pressure regulated volume controlled ventilation versus synchronized intermittent mandatory ventilation in COPD patients suffering from acute respiratory failure. Egypt J Chest Dis Tuberc. 2016; 65(1):121-5.

[10] Ioannidis G, Lazaridis G, Baka S, Mpoukovinas I, Karavasilis V, Lampaki S, et al. Barotrauma and pneumothorax. J Thorac Dis. 2015; 7(Suppl 1):S38-43.

[11] Diaz R, Heller D. Barotrauma and Mechanical Ventilation: StatPearls Publishing, Treasure Island (FL); 2023.

[12] Boussarsar M, Thierry G, Jaber S, Roudot-Thoraval F, Lemaire F, Brochard L. Relationship between ventilatory settings and barotrauma in the acute respiratory distress syndrome. Intensive Care Med. 2002; 28(4):406-13.

[13] Anzueto A, Frutos-Vivar F, Esteban A, Alía I, Brochard L, Stewart T, et al. Incidence, risk factors and outcome of barotrauma in mechanically ventilated patients. Intensive Care Med. 2004; 30(4):612-9.