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Ventilator Weaning Difficulties in ICU: A Study on VAP Patients with Post-Tracheostomy, Thoracic Trauma, and Thoracic Spinal Cord Injury

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ABSTRACT

Ventilator weaning failure is an inability to adapt to reduced mechanical ventilator assistance, which can slow down and prolong the weaning process. Ventilator weaning failure is characterized by increased respiratory rate, use of respiratory muscles, gasping breath, out-of-synchronization efforts to assist breathing and ventilator support, shallow breathing, agitation, and abnormal blood gas arteries. Ventilator weaning failure is an inability to spontaneous breathing trial (SBT), re-intubation, and assisted ventilation after extubation or death within 48 hours after extubation. Weaning process failure factors are divided into non-ventilator and ventilator factors. Ventilator factors include poor patient conditions, long-term use of ventilators, and abnormal blood gas analysis (BGA). Meanwhile, the ventilator factor is related to the condition of the patient's lungs that have suffered severe damage or barotrauma and excessive tidal volume pressure. This study analyzes ventilator weaning failure in patients with Ventilator-Associated Pneumonia (VAP), Post Thoracic Trauma, and Post Spinal Cord Injury in Thoracic 10-12 at the intensive care unit. This research method is based on case reports by compiling comprehensive nursing care. The results of the analysis of ventilator weaning failure in this patient are related to the worsening of the patient's condition, complex thoracic trauma, prolonged use of mechanical ventilators causing ventilator-associated pneumonia (VAP), and respiratory acidosis.

Keywords: Ventilator Weaning Difficulties, Post-Tracheostomy, Thoracic Trauma, Spinal Cord Injury, Ventilator-associated Pneumonia

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1. INTRODUCTION

Thoracic trauma can cause various serious complications with high morbidity and mortality. Thorax trauma is said to be the second leading cause of death among existing types of traumas. Rib fractures are the most common injuries encountered after thoracic trauma. Rib fractures carry significant morbidity and mortality because most are accompanied by severe

injury to the lungs or other organs, which can lead to respiratory distress and progressive respiratory failure (Peek et al., 2021).

Thoracic trauma results in a mortality rate of 20% of all deaths due to trauma. For patients with mild or severe thoracic trauma, the mortality rate reached 18.72%. The other highest clinical condition is the occurrence of rib fractures or rib fractures, both single and multiple, with a prevalence of 33.3%, lung contusions at 15.5%, and pneumothorax at 10%. The heaviest coastal fracture is the flail chest, with a prevalence reaching 60.8%, and this condition requires mechanical ventilation assistance (Octavia et al., 2018).

Rib fractures are the most serious injury received, covering 55% of patients aged over 60 years who die after experiencing road traffic accidents, and 90% of patients show additional injuries (Coary et al., 2020). Based on research by Peek et.al (2022) in the Netherlands, research from January 2015 to December 2017 showed that a total of 245,548 acute patients were treated in hospital through the emergency department (IGD). Of this number, 14,850 patients had rib fractures or rib fractures (6%), 573 (3.9%) patients with flail chest, 4438 (29.9%), 9,273 patients (62.4%) with primary thoracic trauma, and 6,663 (44.9%) of the patients were elderly.

Patients with critical conditions accompanied by instability or failure of organ systems require the assistance of special technological equipment in the intensive care unit (ICU) which aims to support the patient's life. The World Health Organization (2020) states that around 50 million people every year are treated in the ICU, with the main causes being trauma and infection, 40% of whom have to use mechanical ventilators. Based on data obtained from the Indonesian Ministry of Health in 2020, in Indonesia, there were 3 million patients treated in the ICU in 2020, 40 - 45% of whom used mechanical ventilation machines, with a mortality rate for patients with or without a ventilator of 5-10% (Cecep et al., 2023).

The reported prevalence of VAP varies from 5 to 40% of ventilated patients depending on the country, ICU type, and criteria used to diagnose VAP (Dongol et al., 2021). Ventilator-associated Pneumonia (VAP) occurring 48 hours after endotracheal intubation is the second most common type of HAI in the ICU. The cause of death of Intensive Care Unit (ICU) patients apart from their critical illness is related to nosocomial infections. Pneumonia is an infection of one or both lungs. Ventilator-associated pneumonia

(VAP) is a nosocomial infection associated with mechanical ventilation affecting 86% of all nosocomial pneumonia within hospitals. This condition develops mostly after 48 hours after initiation of mechanical ventilation and endotracheal intubation.

Ventilator-associated Pneumonia (VAP) occurs due to micro-aspiration from the patient's oropharyngeal cavity and other factors. Pathogenic microorganisms are often found in the mouth of patients, usually in patients who experience decreased consciousness because they cannot expel secretions. This occurs due to two mechanisms, namely stagnation in the mouth, which results in pathogens in the mouth being unable to enter the stomach. Next, microorganisms remaining in the mouth will migrate into the lungs, which is called aspiration. The host or patient's immunity will find it difficult to fight the aspirated microorganisms, resulting in a pulmonary infection process. In addition, pathogenic microorganisms in VAP cases also originate from nosocomial transmission during treatment in the ICU. This situation is also exacerbated by the mucociliary system, which is not optimal in eliminating secretions in the respiratory tract, and the environment is contaminated with pathogenic microorganisms (Febyan & Soroy Lardo, 2018).

In patients with decreased consciousness, it will result in the inability to cough adequately due to expiratory muscle weakness, causing accumulation of mucus secretions; some patients experience excessive and thick bronchial secretion production. This is due to uninhibited vagal activity, which may be related to the disappearance of peripheral sympathetic tone. This factor can cause atelectasis, pneumonia and has the potential to cause respiratory failure, so this is what causes ventilator associated pneumonia (VAP), so this factor also causes patients to fail to wean the ventilator. Ventilator weaning disorder is an inability to adapt to reduced mechanical ventilator assistance, which can hinder and prolong the weaning process. The implementation carried out was mechanical ventilation weaning and mechanical ventilation management (Amri et al., 2022). There are many factors that cause patients to experience weaning failure or ventilator weaning problems depending on the patient's condition. So, this study aims to analyze ventilator weaning disorders in patients with Ventilator Associated Pneumonia (VAP), Post Trauma Thorax, and Post Spinal Cord Injury Thoracic 10-12.

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2. CASE REPORT

A 56-year-old male patient came conscious through the emergency room on January 30, 2023, at around 22.14 WITA. While repairing the ceiling, the patient complained of right chest pain after falling from the 2nd floor (± 6 meters). When the patient fell, he was unconscious for ± 5 minutes and did not remember what happened. There were no other complaints such as nausea, vomiting and headaches. The patient had a chest tube installed from another private hospital before being referred with the production of an 830cc serous hemorrhagic drain. In the initial physical assessment, it was found that there was damage to the right iliac region of the abdomen, asymmetrical breathing movements (the right lung was left behind), tenderness in the right thorax, and crepitation of the anterior-posterior right hemithorax.

Meanwhile, a physical examination of the thoracolumbar showed tenderness (+) as high as Th 10, hypoesthesia as high as Th 10, and anesthesia As 12. Examination of the right upper extremity in the right wrist region showed edema, deformity, and tenderness in the distal 1/3 of the radius. On January 31, 2023, at 07.01 WITA, the patient was transferred to the Mawar Treatment Room; the patient is planned to undergo surgery on February 6, 2023, namely the coste clipping operation. Decompression, stabilization, fusion (DFS) operations, and close reduction + SAC (Distal Radius) Immobilization Operation Plans. On February 6, 2023, the patient was evaluated as unable to move his legs from the waist down and felt tingling. On February 6, 2023, the patient experienced decreased consciousness while breathing assistance. Preoperative desaturation occurred, so the operation was postponed. On 6/2/2023 at 15.15 WITA, the patient was transferred to the ICU with vital signs examination results of 176/67 mmHg, GCS E1VxM1, and SpO2 = 64%, an evaluation of the duration of surgery was carried out. The supine position was repeated after being positioned on the right lateral side, and the left lung did not expand. The patient underwent ETT intubation in the ICU, and a BiPAP mechanical ventilator was installed. In February 2023, a new diagnosis was developed, namely moderate adult respiratory distress syndrome (ARDS) with a PF ratio of 133. Then, on February 11, due to prolonged use of a ventilator, the patient underwent a complete blood count and procalcitonin examination, resulting in a diagnosis of ventilator-associated pneumonia (VAP) et causa Acinetobacter baumannii. On February 17, 2023, the patient had the WSD removed, but the chest tube in the right

hemithorax was still in place. On February 20, 2023, the patient underwent a tracheotomy. On February 23, 2023, 10.05 WIB – 14.30 WIB, surgery was performed to decompress canal stenosis, TB spondylitis debridement, stabilization and fusion, laminectomy + SSI (pedicle screw), ORIF old fracture/dislocation, bone grafting and costal plating with thoracotomy. After the operation, the patient's consciousness improved with GCS E4VxM6.

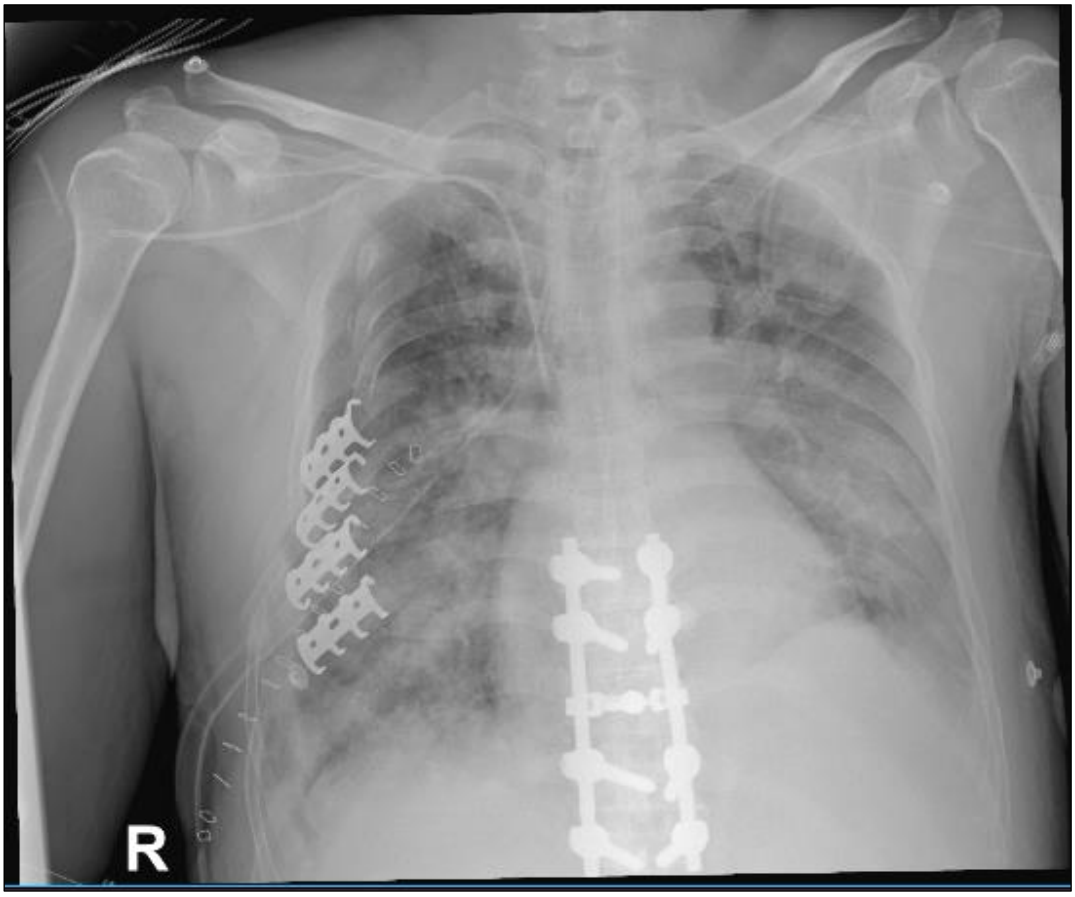

Figure 1. AP Thorax on February 23, 2023

On a thoracic examination, AP was visible. The inter-pedicle internal fixation was installed at the CV Th9 - L1 level with good position and apposition. At 17.04 WITA, the patient was transferred back to the ICU. Patients undergo airway management, pain management, nutritional management, and post-operative wound care.

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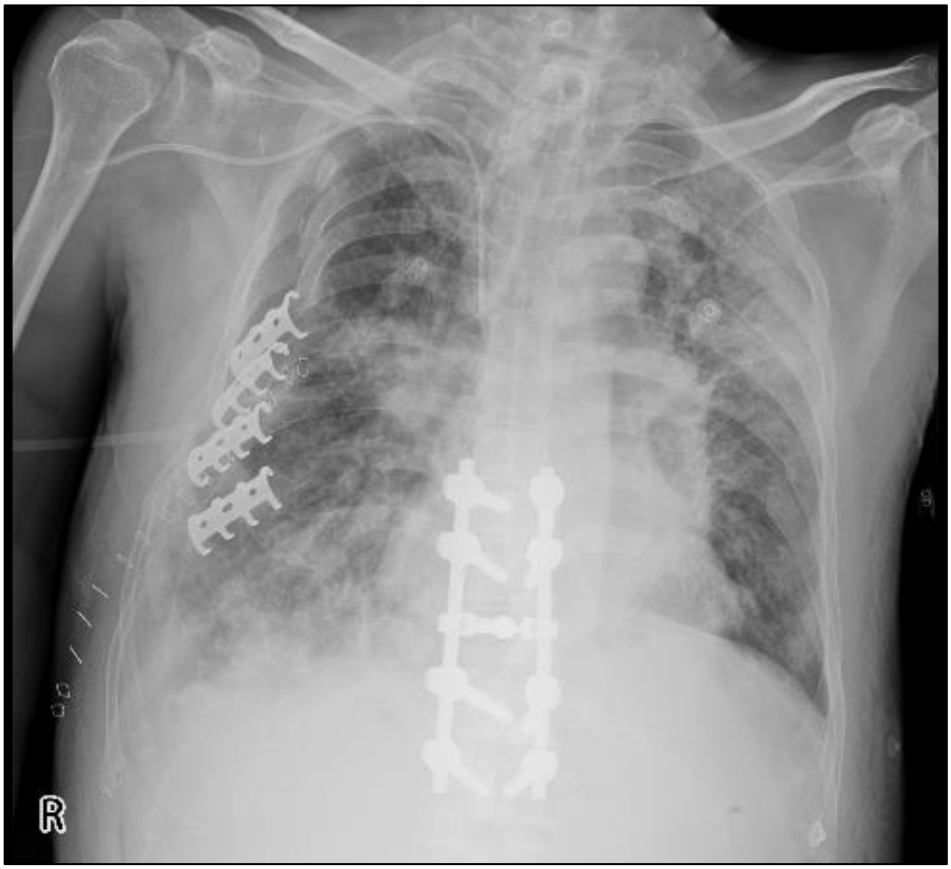

Figure 2. AP Thorax on March 7, 2023

Interpretation results are compared to chest photos dated 02/23/2023:

1) Cor did not appear abnormal.
2) Pneumonia consolidation effect is reduced
3) Right pleural effusion, reduced impression
4) Complete fracture displaced posterior os costae 2, 3, 4.5 right and right lateral os costae 6, 7
5) Deformity of posterior left ribs 7 and 8, suspect old fracture

The weaning or ventilator weaning process lasted 6 days, from March 13, 2023-March 18, 2020. The assessment results showed that the patient had met the criteria for the ventilator weaning process, namely as follows:

a. The patient is conscious of GCS E4VxM6, has normal body temperature, and has an adequate cough
b. FiO2 ≤ 50 %
c. PEEP < 8
d. Blood gas analysis is within normal limits, or there is no respiratory acidosis

e. Stable heart function, namely heart rate ‹ 140 x / minute, blood pressure within normal limits, no myocardial ischemia

On March 11, 2023, the patient was tried for weaning, where at 07.04 WITA, the patient was installed on a BiPAP PV mode ventilator with FiO2 50% and PEEP 6. Then, at 07.11, WITA weaning was tried by reducing the FiO2 to 45%, PEEP 5, and hemodynamic results were obtained. And SpO2 within normal limits. At 09.12, WITA weaning was carried out again, where the FiO2 was gradually reduced to 40%, Pins 18, Freq 20, Psupp 14, and PEEP 5 showed that hemodynamic results and SpO2 were relatively stable. However, after the weaning trial, the patient's breathing was inadequate, so2 was maintained at 50% and PEEP 6. Meanwhile, on March 12, 2023, at 12.00 WITA, the patient's breathing effort was inadequate, and there was an increase in RR, the FiO2 increased to 70%, and PEEP 6. At 18.00, the hemodynamic status was within normal limits, and RR was within normal limits, namely 18-23 x / minute, FiO2 was reduced again to 50%, and PEEP 6.

On March 13, 2023, at 06.11, WITA Increased again to 60%, and PEEP 6 had good hemodynamic status, namely BP = 119/74 mmHg, pulse = 88 x / minute, and RR = 16 x / minute. However, laboratory results on March 12, 2023, showed that the patient experienced partially compensated respiratory acidosis. At 07.49 WITA, FiO2 was increased again to 60% and PEEP 6, usually because the patient would be helped with self-care, bathing, and defecating, where the patient had to be mobilized on the right and left sides. Sometimes, the patient's breathing was inadequate during this mobilization, and diaphoresis occurred.

On March 14, 2023, at 07.00 WITA, the patient's hemodynamic condition improved with BP = 122 / 73 mmHg, Pulse = 92 x / minute, Pulse = 92 x / minute, RR within normal limits, namely 18 x / minute SpO2In 95%, FiO2 was reduced to 50% and PEEP was reduced to 4. Meanwhile, AGD results showed that the patient had fully compensated metabolic alkalosis. At 12.00 WIB, FiO2 was 50%, and PEEP was 6 because hemodynamic status was unstable, tachycardia occurred, and SpO2 was below 93. 5, FiO2 was increased to 70%, and PEEP was 6.

On March 15, 2023, at 06.27, WITA hemodynamic status was unstable with BP = 150 /85 mmHg, HR = 107 x / minute, RR = 26 x / minute, SpO2= 93%, and at dawn at 05.00 WITA shortness of breath worsened until oxygen saturation decreased to 84% so that FiO2 was increased to 80%, PEEP 6, Pins 20. Freq 20 and PuSupp 12. The blood

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gas analysis results on that date showed fully compensated respiratory acidosis. At 08.13 WITA, the FiO2 was again lowered to 50% and PEEP 4. At 16.00 WITA after mobilization on the right side and left side, the patient's breathing was slightly worse, so the FiO2 was increased to 55%, and PEEP 4, where the hemodynamic status improved and SpO2 was 99%.

3. DISCUSSION

From the evaluation of weaning trials with the criteria of FiO2 ≤ 50%, patients still experienced failure due to unstable hemodynamic response, increased RR, oxygen saturation below normal, and blood gas analysis results where PCO2 increased. Additionally, objective responses found in patients included shortness of breath, gasping breath, diaphoresis, and cold acral.

From day 1 to day 3, the patient's response during weaning was quite good, even though it ended in failure and FiO2re-improved. However, on the 4th day, the patient's hemodynamic status began to become unstable, experiencing tachycardia and a gradual decrease in SpO2. Until the 5th day, the patient's condition began to worsen, where the patient experienced decreased consciousness with GCS E3VxM5 and experienced desaturation and hypotension. This situation worsened on the 6th day until FiO2 was fully increased to 100% with PEEP 5, but the condition worsened. The patient's GCS decreased to E1VxM1, desaturation 75%, hypotension 87/54 mmHg (with Vascon therapy 0.4 mcg/kg body weight) until, In the end, the patient experienced arrest and died.

In this condition, many things that influence failure in the weaning process are due to the complexity of the patient's condition and the decline or worsening of the patient's condition. Research conducted by Cecep et al. (2023) stated that the factors for failure of the weaning process are divided into 2, namely non-ventilator factors and ventilator factors. Ventilator factors include poor patient condition, long-term ventilator use, and abnormal blood gas analysis (AGD) results. Meanwhile, the ventilator factor is related to the condition of the patient's lungs, which have suffered severe damage or barotrauma and excessive tidal volume pressure.

Thoracic trauma causing rib fractures and flail chest pain is caused by three main problems, namely hypoventilation due to pain, impaired gas exchange in lungs damaged due to fractures, and changes in respiratory mechanisms. Pain is related to rib movement, which can reduce tidal volume and increase the occurrence of significant

atelectasis. These two things can then cause retention of pulmonary secretions and pneumonia (May et al., 2016) in patients with thoracic trauma accompanied by accompanying complications, namely rib fractures, flail chest, and pulmonary contusions resulting in respiratory distress or Adult Respiratory Distress Syndrome (ARDS). ARDS occurs through several mechanisms, namely (1) Suprapontine input, which includes (the central cortex, limbic system, and hypothalamus). Pain, anxiety, and discomfort stimuli enter the supra pontine structures, which are then transmitted to the central respiratory center and central chemoreceptors (medulla oblongata). (2) Inflammatory input related to thoracic trauma will stimulate inflammatory mediators (histamine, bradykinin, prostaglandin), conveying this response to the respiratory center in the brain. This stimulus is conveyed to the chemoreceptors in the lungs. (3) mechanical input is related to atelectasis, where the lungs cannot expand properly. Mechanical receptors in the lungs convey this signal to the respiratory center in the brain. (4) Biochemical Input is related to the response to lung damage. It will disrupt the perfusion-ventilation process, characterized by an increase in PaCO2, a decrease in pH, and a decrease in PaO2. Peripheral chemoreceptors respond to this situation through the carotid body located in the arteries. In these 4 mechanisms, the respiratory center will activate the function of the respiratory muscles so that the lungs will make breathing efforts due to the stimulation of the neuromechanical coupling respiratory mechanism so that they will experience respiratory distress (Spinelli et al., 2020).

Patients with respiratory failure or respiratory distress require respiratory assistance using a mechanical ventilator. Inadequate breathing results in the patient being dependent on a mechanical ventilator for a long time, which will lead to the development of a new diagnosis, namely ventilator-associated pneumonia (VAP). The mechanism of VAP is due to the aspiration of pharyngeal bacteria, where the distal airways should be sterile and contaminated by the micro-aspiration of digestive pathogens. Endotracheal intubation makes the patient unable to cough effectively, thereby disrupting the mucociliary clearance function, reducing the protective ability of the airway, thereby increasing the risk of VAP (Vallecoccia et al., 2020). Based on research by Hui Kao et al. (2023) with the title "Mortality and Ventilator Dependence in Critically ill Patients with Ventilator-Associated Pneumonia Caused by Carbapenem-

resistant Acinetobacter baumannii states that out of a total sample of 402 patients, 25.2% or 101 patients had VAP experienced death on the 21st day, this was related to the risk factors of decreased body mass index (BMI), high APACHE II score, increased SOFA score, use of vasopressors, PF ratio ‹ 200, hypo albumin, extended stay in the ICU and antibiotic resistance. This research also states that the level of ventilator dependence for VAP patients when they leave the hospital is 60%. Meanwhile, the level of ventilator dependence in patients with VAP on day 21 was 48.8%. This figure is related to the difficulty of weaning in patients on mechanical ventilators. In addition, the condition of sepsis in patients who depend on mechanical ventilation for a longer time has a higher rate of difficulty weaning than patients without sepsis.

Based on research by Kyriakoudi et al. (2022) in a study entitled "Weaning Failure in Critically Ill Patients is Related to The Persistence of Sepsis Inflammation," out of 65 intubated patients (42 sepsis patients and 23 non-sepsis patients) stated that sepsis patients had a percentage of failure in spontaneous breathing trial (SBT) than non-sepsis patients with a ratio of 85% and 15%, of which 51% were patients diagnosed with ventilator-associated pneumonia (VAP). Systemic inflammatory conditions are related to cardiopulmonary stress during the ventilator weaning process.

4. CONCLUSION

Based on the analysis of ventilator weaning disorders in the cases raised, it was related to several things, namely related to severe lung damage due to thoracic trauma, which resulted in complications of rib fractures and flail chest, causing the lungs not to expand optimally and gas exchange problems. Severe injury conditions cause patients to be unable to breathe adequately and dependent on a mechanical ventilator. On the other hand, using mechanical ventilators for a long time has resulted in developing a new diagnosis, namely ventilator-associated pneumonia (VAP), due to microaspiration invasion of pathogens into the respiratory tract. The infection experienced by the patient will develop systemically, causing sepsis, where the release of inflammatory mediators will send signals to the brain, and the brain will activate breathing efforts through nerves connected to the central respiratory system, which will impact respiratory distress. This complexity is a factor in weaning failure in this case.

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AUTHOR CONTRIBUTIONS

Substantial contribution to conception, data collection, analysis, writing manuscript and revision: Virgiana Piesesha.

CONFLICT OF INTEREST

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

DATA AVAILABILITY STATEMENT

The data are not publicly available due to privacy or ethical restrictions.

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