Mini Review
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Ventilator-induced lung injury in ARDS

Abstract

Acute respiratory distress syndrome (ARDS) develops in nearly 2 to 19 patients in every 100 critically ill patients, and the incidence of ARDS demands the implementation of mechanical ventilation to support the respiratory distress in the patients. However, mechanical ventilation is the primary cause that leads to ventilator- induced lung injury. A sequence of pathophysiological mechanisms involving volutrauma/barotrauma results in ventilator-induced injury in the later stages. In other words, ventilator-induced lung injury is an outcome experienced as a result of physiological and morphological alterations of the lungs due to mechanical ventilation. Among all factors, VILI primarily occurs as a result of improper ventilation, and further continuation of improper ventilation can even result in a secondary ventilator-induced lung injury. Furthermore, ventilator-induced lung injury can result in hypoxia, pulmonary edema, and multi-organ dysfunction and can even risk the life of the patient. This makes it essential to identify some effective strategies that can act as a measure to support protective ventilation to prevent ventilation-induced lung injuries. This review explores the clinical aspects of barotrauma to gather proper information about the aspects that contribute to ventilator- induced lung injury so that the recommendations can be suggested to prevent the increasing incidences of these injuries during ventilation. To conduct this review, an extensive search of multiple databases, including PubMed, ScienceDirect, Medline, etc., was conducted with the mentioned keywords, and 15 articles were shortlisted to be reviewed within this article. The findings of this review have indicated that protective ventilation is the most effective strategy that can support the survival of patients suffering from ventilator- induced lung injury. Protective ventilation not only helps in saving lives, but was also found to be a useful measure in preventing lung injuries experienced by the patients due to mismatch between actual and required optimum ventilator settings for the patient. Further findings of the review also indicate that ventilator-induced lung injuries could be prevented by ensuring the transpulmonary pressure is within the physiological range and the position of the patient is maintained supine for the majority of the time to support homogeneity in the distribution of the transpulmonary pressure.

Keywords: barotrauma, lung injury, mechanical ventilation, volutrauma

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Introduction

Acute respiratory distress syndrome is observed as a comorbidity in multiple illnesses and occurs in 2.5–19% of critically ill patients admitted in ICUs.[1–4] It has been observed that the patients likely to be the first hit by acute respiratory distress syndrome show different symptoms managed by mechanical ventilation, which is associated with volutrauma and barotrauma.[5,6] In most cases, mechanical ventilation is observed to be a factor that induces lung damage, and it has been identified that implementation of mechanical ventilation can aggravate the adverse effects after the initial trauma experienced by the lungs. Such aggravated response results in different scars within the lungs known as ventilator-induced lung injuries (VILI).[7] Leakage of the blood/gas barrier and scar formation are some of the alarming changes observed in VILI. However, several studies have highlighted that the use of positive end-expiratory pressure (PEEP) can prevent VILI.

Pathophysiology of VILI

Considering the pathophysiology of VILI, it is evident that these injuries involve complex overlapping interactions. It usually occurs as a result of two primary phenomena. The first is found to be associated with an abnormal increase in the inspiratory pressure that occurs at the end of the inspiratory cycle and results in over- distention of alveoli.[6,8] This over-distension creates a risk of rupture of alveolar walls and airways, primarily in the non-dependent lung regions.[7] The second phenomenon that is observed in the pathophysiology of ventilator-induced lung injury is evident in the dependent lung region. This phenomenon occurs due to excessive stress experienced by alveoli because of the force exerted at the opening during inspiration and at the time of closing during expiration. This is found to be associated with the utilization of insufficient positive-end expiratory pressure (PEEP) to prevent collapse.[7,8] However, there is always a possibility that the two phenomena overlap. Similarly, an inappropriately high PEEP can amplify the consequences of the cyclic alveolar collapse and its reopening, leading to alveolar injuries in the form of atelectrauma, since it may generate over-distension and an excessive dropping of tidal volume within the lungs. Furthermore, experimental research has supported the notion that the occurrence of VILI depends upon the collapse of the airways and the cyclical recruitment in lungs that might be damaged by a prior inflammatory state.[7]

Ventilator related factors that increase the risk of ventilator-induced lung injuries in ARDS

The primary factor that leads to ventilator-induced lung injuries is that, in most cases, mechanical ventilation uses high tidal volume and high plateau pressure to ensure that the patient is protected from hypoxemia.[8,9] This attempt to ensure a proper supply of oxygen to the patient suffering from acute respiratory distress syndrome to prevent collapse indirectly causes excessive stress on the lungs and also damages the alveolar walls due to over-distension that results in ventilator-induced lung injuries.[9,10] Another factor is the non-homogeneous distribution of transpulmonary pressure in the lungs. Transpulmonary pressure in the lungs is defined as the distending force experienced by the lungs.[11] In general, this force is shared

homogenously by every fiber of the lung, but when this distribution is uneven, the regions that collapse do not take any strain, and the entire stress is then taken up by the neighboring fibers resulting in excessivestress on them, which, in prolonged duration can result in rupture or scars that become evident in the form of ventilator-induced lung injuries.[12] This excessive strain also activates the macrophages and the epithelial cells to release interleukin-8, which is aggravated by the recruitment of neutrophils by the cytokines in the affected location resulting in full-blown inflammation, further contributing to the worsening of the situation.[13] In addition to these factors, capillary endothelial stress failure is also likely to contribute to ventilator-induced lung injury. This is because of the enhanced regional blood flow that occurs as a result of attempted ventilation- perfusion matching usually leads to enhancement in the stress of capillary epithelium, and because of this reason, it is often observed that instead of supporting the lung injury, the mechanical ventilation further worsens it.[5]

Possible protective strategies to reduce the incidences of VILI in ARDS

Based on the risk factors that have been identified, some of the strategies that can be implemented to reduce the incidences of ventilator-induced lung injury in acute respiratory distress syndrome are stated in this section.

Lung protective ventilation: Protective ventilation refers to the implementation of ventilation such that it makes the simultaneous use of the concept of low tidal volume and limited plateau pressure to ensure that the over-distension of alveoli, which might lead to VILI, does not occur.[7–9] Alongside minimizing the chances for over-distension, protective ventilation also ensures an appropriate positive end-expiratory pressure to prevent the chances of collapse at the time of expiration.[6,14] This will lead to reduced experience of stress by the lungs, thereby protecting the lungs and minimizing the risk of lung injuries.[9,10] The implementation of lung protective ventilation in patients with acute respiratory distress syndrome has been found to improve the ultimate patient outcomes causing minimum damage to the patient and ensuring a better recovery.[9,15]

Maintaining the transpulmonary pressure and placing the patient in the supine position: Maintaining the transpulmonary pressure in the normal physiological range and placing the patient in the supine position ensures its effective and homogeneous distribution. This also reduces the regional stress that is experienced by some of the elastin and collagen fibers of the lungs due to the improper distribution of transpulmonary pressure which further prevents the associated occurrence of the development of ventilator- induced lung injury and inflammation in the lungs.[5,7,8,11–13,15]

Ensuring low tidal volume: It has been evaluated that the patients of acute respiratory distress syndrome present themselves with two regions of the lungs: aerated non-dependent lung region and a non-aerated dependent lung region. The dependent regions are more likely to be collapsed because they are relatively lower down in comparison to the other area of the lungs from a gravitational perspective. Because of the lower amount of air that is accessible for ventilation, this condition is referred to as having "baby lungs[5] & quot; Decreased tidal volume thus becomes a remedy to ventilate this "baby lung" and avoid overinflation of the relatively small, regularly aerated portions.[7,16] This strategy was evaluated to be effective in controlling the death rates occurring due to ventilation-induced lung injuries in the patients of ARDS.[3,5,8]

Management of capillary and arterial pressure: Making use of vasopressors in addition to the administration of IV fluids can significantly help in achieving high mean arterial pressure and cerebral perfusion pressure. When capillary pressure is high by keeping the arterial pressure high, the chances of acquiring ventilator-induced lung injury are significantly minimized and, therefore, can act as a strategy to prevent ventilator-induced lung injuries.[5]

Quick review table: Intervention Number of studies that discussed this intervention. Lung protective ventilation 7. Maintaining the transpulmonary pressure and placing the patient in the supine position 7. Ensuring low tidal volume 5. Management of capillary and arterial pressure 1.

Conclusion

Acute respiratory distress syndrome develops as a comorbidity in every 2–19 out of 100 critically sick individuals, and this prevalence of ARDS necessitates the use of mechanical ventilation to sustain the patients respiration. However, due to a series of pathophysiological pathways that include barotrauma, volutrauma, and atelectrauma as common components that eventually results in ventilator-induced injury, mechanical ventilation is the main factor that causes ventilator-induced lung injury. Or, to put it another way, ventilator-induced lung damage is a consequence of the physiological and morphological changes to the lungs brought upon by mechanical breathing. These injuries result from inappropriate ventilation, and if the problem persists, it may potentially cause secondary ventilator-induced lung damage.[2] Ventilator-induced lung injury can also lead to hypoxia, pulmonary edema, multi-organ dysfunction, and can even put the patients life in danger. According to the current reviews findings, protective ventilation is the most efficient method for ensuring the survival of patients with ventilator-induced lung injury. Protective ventilation was proven to be helpful not only in sustaining the patients lives but also as a safeguard against the lung injuries that the patients can sustain from improper handling during ventilation. Additional findings also showed that patients should be kept in supine position for a majority of the time to support homogeneity in the distribution of transpulmonary pressure, transpulmonary pressure must be kept within the physiological range, and other such steps must be taken to prevent ventilator-induced lung injuries.

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Conflicts of interest

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