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(REVIEW ARTICLE)



COVID-19 and mechanical ventilation

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Abstract

Mechanical ventilation (MV) is used to treat patients with severe coronavirus disease 2019 (COVID-19). This severe respiratory illness, typically develops 8 days after symptom onset and when it does not respond to non-invasive respiratory support, it requires advanced respiratory support, including high concentrations of inspired oxygen and mechanical ventilation. Such therapies are also required for the acute respiratory distress syndrome (ARDS), which has been widely studied over several decade. Obesity as a disease causes a restrictive lung disease and is a sufficient predisposing factor for difficult ventilation. Higher BMI patients are more likely to be young, with single organ failure, less chronic comorbidity but with increased severity of hypoxemia at presentation. Severe respiratory failure from coronavirus disease 2019 pneumonia not responding to non-invasive respiratory support requires mechanical ventilation. Although ventilation can be a life-saving therapy, it can cause further lung injury if airway pressure and flow and their timing are not tailored to the respiratory system mechanics of the individual patient. The phenomenon of "hard lung" is observed as the ventilation of the intubated patient is very arduous and recruitment requires a lot of effort. Coronavirus disease 2019-induced acute respiratory distress syndrome is more severe in morbidly obese patients. This relationship between BMI and mortality was investigated by several observational studies, but the relationship was not universally observed. Some studies found increased BMI was associated with an increased risk of requiring intubation and ventilation, but with no clear relationship with mortality. The combination is quite difficult as these patients oppose the ventilator. Our objective was to determine the association between MV for treatment of COVID-19.

Keywords: COVID-19; Ventilation; Obesity; Pneumonia; ARDS; MV

1. Introduction

The prevalence of adult obesity and severe obesity in 2017 to 2018 has increased since 2009 to 2010 and is now 42% and 9%, respectively [1]. Obesity is a global disease with at least 2.8 million people dying each year as a result of being overweight or obese according to the world health organization figures. Obesity is affecting most of the physiological processes and modifying the functions of the system including the immune system [2]. It is crucial to understand the effect of obesity on the course of infection to prevent or mitigate the morbidities and mortalit [3,4]. In the current COVID-19 era, bariatric teams are aware of the potential risks and thus stressing the extra caution and appropriate management of these patients [5]. Knowing the scale of the obesity problem in the world, we anticipate difficult times for this group of patients in Europe, America, Middle East and rest of the world with a high rate of obesity [6]. In 2009, a significant percentage of admissions to the hospitals and mortality because of H1N1 Influenza a virus infection was due to obesity, an estimated 151,700–575,400 total deaths was reported U [7,8]. Patients with coronavirus disease 2019 often suffer severe symptoms, from viral pneumonia to respiratory distress. This respiratory distress can lead to alveolar damage and fibrosis in the lungs, which reduces oxygen saturation in the blood. COVID-19–related respiratory distress has been associated with higher rates of mortality and intensive care unit (ICU) admission than respiratory distress associated with other illnesses. The recommended treatment for patients suffering from severe respiratory

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distress due to COVID-19 is mechanical ventilation (MV), which provides oxygen to these critically ill patients and removes carbon dioxide from the blood. The use of MV treatment for severe COVID-19 is common [9-15].

During inspiration, the lung expands owing to a positive transpulmonary pressure. In spontaneous ventilation, this gradient is produced by a negative pleural pressure created by the inspiratory muscles, mainly the diaphragm. In contrast, controlled mechanical ventilation relies upon a positive airway pressure driving gas into the lungs, with the positive transpulmonary gradient dependent upon an increased alveolar pressure, and passive movement of the chest wall. The fundamental ventilator parameters that can be set are airway pressure and flow and their timing, which need to match the patient's respiratory system resistance and elastance (inverse of compliance). At each time point during inspiration, airway pressure is determined by the equation of motion and equals the sum of end-expiratory alveolar pressure, the product of flow and resistance to flow, and the product of tidal volume and elastance of the respiratory system. Mechanical ventilation can be delivered in mandatory mode or in assisted mode to support spontaneous breathing. In the latter modality, the patient's inspiratory effort triggers the delivery of breaths, the work of breathing is shared in various proportions between the respiratory muscles and the mechanical ventilator, and the transpulmonary pressure is generated by a combination of a negative pleural pressure and a positive alveolar pressure. This ventilatory support can be necessary to sustain life in the acute phase of the disease while the immune system fights the viral infection, yet can cause harm to the patient if the levels of positive pressure are not tailored to the associated lung mechanics (ventilator-induced lung injury). Ventilator-induced lung injury is well studied in classical ARDS, a syndrome associated with a distinct histopathological entity termed diffuse alveolar damage (DAD). Diffuse alveolar damage is a widespread, heterogeneous inflammatory reaction comprising alveolar infiltrates with leucocytes and proteinaceous deposits, damage to alveolar pneumocytes, the basement membrane and endothelium, and patchy areas of haemorrhage [16-17].

2. Case Report

A 55-year-old man (was admitted to the intensive care unit due to respiratory failure), with a body weight of 130 kg and coronavirus disease 2019 pneumonia. The patient was intubated and put into mechanical ventilation with model ACV, FiO2 70%, which gradually dropped to 50%, PEEP 18 which gradually dropped to 14, Tidal Volume 500 ml which gradually dropped to 480 ml and respiratory rate 30/min which gradually dropped to 25/min, and I: E $\frac{1}{2}$ to 1/3 and finally to 1/4. Suppression of the patient was achieved by drip co-administration of Ultiva, Diprivan, Esmeron, Dormicum, and Levophed in titrated doses for the best possible ventilation of the patient. Obesity in combination with COVID-19 leads to increased peak, up to 60 and high airway resistance. Satisfactory dosing of Esmeron 60 mg/h and placement of low tidal volumes <500 ml, as well as respiratory rate < 30/min, PEEP < 16, appears to improve ventilator ventilation when co-administered: Ultiva, Diprivan, Dormicum at satisfactory levels in combination with Levophed to maintain hemodynamic stability.

2.1. Management and Outcomes

The pathophysiology of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection can lead to a pattern of lung injury in patients with severe COVID-19 pneumonia typically associated with two distinct phenotypes, along a temporal and pathophysiological continuum, characterized by different levels of elastance, ventilation-to-perfusion ratio, right-to-left shunt, lung weight and recruitability. Understanding the underlying pathophysiology, duration of symptoms, radiological characteristics and lung mechanics at the individual patient level is crucial for the appropriate choice of mechanical ventilation settings to optimize gas exchange and prevent further lung injury. The choice of tidal volume should be based on obtaining a driving pressure < 14 cmH2O, ensuring the avoidance of hypoventilation in patients with preserved compliance and of excessive strain in patients with smaller lung volumes and lower lung compliance. The level of positive end-expiratory pressure (PEEP) should be informed by the measurement of the potential for lung recruitability, where patients with greater recruitability potential may benefit from higher PEEP levels. Prone positioning is often beneficial and should be considered early.

3. Discussion

Severe respiratory failure from coronavirus disease 2019 pneumonia not responding to non-invasive respiratory support requires mechanical ventilation. Although ventilation can be a life-saving therapy, it can cause further lung injury if airway pressure and flow and their timing are not tailored to the respiratory system mechanics of the individual patient. Studies suggest that MV patients have a greater hazard of inpatient readmission and all-cause mortality compared to non-MV patients. Whether this is due to a difference in severity of illness, for which MV may be a proxy or a consequence of MV itself, patients who are ventilated appear to be at greater risk for adverse outcomes following discharge.

4. Conclusion

This severe respiratory illness (COVID-19), when it does not respond to non-invasive respiratory support, it requires advanced respiratory support, including high concentrations of inspired oxygen and mechanical ventilation. COVID-19 pneumonia is a pathophysiological entity distinct from classical ARDS and requires different ventilatory management. The hypoxaemia is predominantly underpinned by an increased dead space and V'/Q' mismatch and is not correlated with the healthy lung volume. Tidal volumes should not be limited stringently, but should be more liberal as long as the driving pressure is limited. PEEP levels should not be titrated in relationship to hypoxaemia but on the potential for recruitability of the lung, which is not necessarily high if there is minimal air-space disease. Thus it appeared that low respiratory rate < 30/min and low tidal volume < 500 ml with a desired PEEP less than or equal to 14, has a beneficial effect on obese patients.

Compliance with ethical standards

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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