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Respiratory Failure in Acute Medical Unit

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Abstract

The huge rise in patients presenting to acute medical units with respiratory failure during covid-19 pandemic has exposed the acute physician to different types of assisted ventilation, which was not implemented frequently before the pandemic. High-flow nasal oxygen and non-invasive ventilation have reduced the need for invasive mechanical ventilation.

Shortage in level 3 beds is still an issue, and even after the pandemic, the idea of introducing level 1.5 or enhanced care in acute medical units is evolving, which means that acute physicians are more likely to deal with respiratory failure cases and should be aware of the indications and benefits of these two modalities. The authors discussed in this review the basics of using high-flow and non-invasive ventilation in a straightforward approach.

Keywords: High flow nasal oxygen non-invasive ventilation; Acute respiratory failure; Bilevel non-invasive ventilation; BiPAP; CPAP.

Introduction

Respiratory failure is defined as hypoxia (reduced arterial partial pressure of oxygen (PaO₂) below 8 kPa on room air at sea level) with or without hypercapnia. It occurs due to impairment of gas exchange between the lungs and the blood. (T1RF) is the presence of hypoxia without hypercapnia and commonly occurs in conditions leading to ventilation-perfusion mismatch, such as pneumonia, pulmonary embolism, or pulmonary oedema, impairment in diffusion like in interstitial lung diseases, and presence of right to left shunt which is common in congenital heart diseases.

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Copyright[®] 2023 by Ibrahim MW, et al. All rights reserved. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited. While type 2 respiratory failure (T2RF) is hypoxia with hypercapnia (arterial partial pressure of carbon dioxide (PaCO₂) more than 6.5 kPa on room air at sea level), the main pathology in T₂RF is hypoventilation which is common in acute exacerbation of chronic obstructive pulmonary disease (AECOPD), hypoventilation secondary to coma from drug overdose, underlying disorders neuromuscular or obesity hypoventilation syndrome (OHS), worth mentioning that any cause of T1RF can lead to T2RF if not treated adequately [1].

Early recognition of symptoms and signs of respiratory failure (RF) is essential. Patients usually present with respiratory distress, hypoxia, increased respiratory rate, use of accessory muscles, anxiety with central cyanosis due to hypoxia, headache, blurred vision, and coma with hypercapnia. Initially, applying oxygen can save a life in severe hypoxia, even if the patient is known to have chronic T₂RF. Researchers can titrate oxygen later once oxygen levels are adequate to avoid hypercapnia, but severe hypoxia has to be dealt with urgently as it kills within minutes. Urgent arterial blood gas (ABG), portable chest X-ray and initiation of treatment are recommended with a very low threshold to start non-invasive ventilation (NIV) if the patient deteriorates or does not respond to initial treatment.

Discussion

Nasal cannula provides low-flow oxygen (1-6 L/min) and is usually started in any patient with hypoxia targeting a peripheral oxygen saturation (SpO₂) of 94-98% in T1RF or 88-92% in T2RF, when nasal cannula fails to achieve this target and the patient is still

hypoxic with T1RF, The next step is usually high-flow oxygen via a non-rebreather mask which provides roughly 80 plus % of FIO₂ or humidified high-flow oxygen, which is delivered by a high-flow nasal cannula (HFNC) and can provide FiO₂ up to 100% with flows up to 60 L/min or higher delivered through a nasal cannula and this can lead to a positive end-expiratory pressure by less than 5 CM H₂O. HFNC may not reduce mortality but has been shown to improve dyspnoea and comfort and reduce the rate of intubation in patients with T1RF [2,3].

Current recommendations for patients with a high risk of T2RF are to use venturi masks which provide a fixed percentage of fractional inspired oxygen (FiO₂) to minimize the risk of CO₂ retention leading to established T2RF, and early use of NIV if no response to treatment. Researchers proceed with urgent intubation and mechanical ventilation in extreme cases where respiratory distress is or the significant, patient presents hemodynamic instability and is likely to be arrested [4].

NIV delivers a positive pressure ventilation through a non-invasive interface (mask) and delayed initiation of NIV in RF can risk clinical deterioration [5]. Patients with failure should be respiratory closely monitored in level 1.5 (enhanced care) or level 2 (high dependency unit) at the least, nurse patients at more than a 30-degree angle and prepare the ventilator after connecting the interface-which should fit well to patient's face without excessive strap tension by headgear-to ventilator tube. Common types of NIV are bilevel positive airway pressure (BiPAP or Bilevel NIV), used mainly in patients with T2RF, and continuous positive

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airway pressure (CPAP), used for patients with T1RF [5].

Bilevel NIV ventilation provides inspiratory and expiratory positive airway pressure, IPAP and EPAP, respectively, at two different levels. The difference between both pressures is called delta positive airway pressure (delta PAP), which correlates with the tidal volume delivered. The larger the delta PAP, the larger the tidal volume and hence alveolar ventilation, assuming the respiratory rate is constant. Patients should be able to breathe spontaneously; each breath is supported to the same level during Bilevel NIV ventilation. There are two modes in Bilevel NIV, the spontaneous mode, where the patient is supported during spontaneous breaths, and the timed mode, where the patient is supported during timed breaths. Commonly a spontaneous-timed mode is selected to ensure every breath is supported. A minimum respiratory rate is present as a backup if the patient does not breathe spontaneously; the backup rate is usually between 8-12 breaths per minute. The initial IPAP setting is usually 10 cm H_2O and then up titrated by 2 cm H_2O increments to a maximum of 20 cm H₂O (maybe 25 cm H₂O if morbid obesity). The rationale for starting low and going slow is to ensure compliance, allow patient-ventilator synchrony, provide the IPAP required to reduce the respiratory rate and improve dyspnoea while increasing the tidal volume and minute ventilation to treat the hypercapnia. Initial EPAP is usually 4 cm H₂O and can be increased gradually, especially in morbid obesity, to a maximum of 10 cm H₂O if oxygenation remains poor. However, increasing the EPAP will reduce the delta PAP and hence the delivered tidal volume; oxygenation also can improve by increasing the FiO₂ instead of EPAP aiming a target SpO₂ of 88 to 92% in patients with T2RF [6].

CPAP is commonly used for T1RF as it delivers a continuous level of PAP, leading to improvements in oxygenation rather than ventilation. Tidal volumes cannot be titrated as in Bilevel NIV, and patients using CPAP must initiate all breaths; this means only one mode is available. The initial level is usually 5 cm H₂O and can be up titrated to a maximum 20 cm H₂O (higher levels are not well tolerated) with FiO₂ to keep SpO₂ 94 or above as the initial respiratory failure treated here is T1RF and should have no risk for hypercapnia. Titration aims to reduce respiratory rate and improve dyspnoea and oxygenation [7].

The authors have explained the indications for NIV and settings for Bilevel NIV and CPAP individually. The author advises close monitoring of vital signs, oxygenation, and mental status while up titrating the pressure for the first hour, then further evaluation clinically and by a repeat arterial blood gas (ABG) to evaluate the response, if there is a response and the patient shows improvement in oxygenation with a reduction in the respiratory rate researchers then continue with the NIV. However, suppose there is no clinical improvement, and the ABG does not show signs of resolution of respiratory failure after one hour of optimal pressures and treatment. In that case, mechanical ventilation should be prepared with the intensivist unless the ceiling of treatment was decided to be NIV-only [8].

Ventilator desynchrony is a common scenario, especially in patients who are NIV naïve; desynchrony means a mismatch between the ventilator phases and the patient's breathing, which could be due to

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interface leak, anxiety, or auto-PEEP. Nebulizer with a reduction in the respiratory rate and tidal volumes can help with auto-PEEP; changing the mask, tightening the straps, or even changing the ventilator mode can help with leaks, and for anxiety accompanied with NIV use reassurance and change the interface and if this is not enough considering gentle sedation with low doses of morphine (with close monitoring to avoid worsening respiratory failure) and antiemetic is recommended [9].

Other modes of NIV which are not commonly used are pressure support ventilation (PSV), which allows the patient to take some control to trigger each breath and regulate the depth and pattern of breathing, and proportional assist ventilation (PAV), which delivers inspiratory flow and volume proportional to the patient's effort. Comparing both modes as a solution for desynchrony found that PAV was more tolerable and comfortable than PSV [10].

So far, the authors have discussed the indication for NIV and the practicality of applying NIV in the acute medical unit (AMU) with a few tips on common problems. Below the authors will discuss signs of success or failure of NIV, weaning, and possible complications of NIV. Clinical improvement (reduction in dyspnoea and respiratory rate with improvement in oxygenation) and gas exchange (reduction in PaCO₂ and increase in PH on ABG) should be expected within the first two hours of NIV use, assuming an optimal level of pressure is applied, and the treatment for the initial condition leading to RF was initiated early [8]. Worsening of gas exchange, inability to clear secretions, hemodynamic instability with increasing respiratory rate, and anxiety or encephalopathy after two hours of NIV are all signs of NIV failure, which can occur in up to one-third of patients. Consequently, it is a good practice to decide the ceiling of treatment and communicate this clearly to patients and families before initiating NIV [11, 12].

Successful NIV is continued for at least 24 hours with minimal interruption for the intake of small amounts of food and liquids due to the risk of aspiration and for patient comfort. Following the initial 24 hours, consideration for weaning should be done progressively, and clinical indicators for weaning are a respiratory rate below 22 bpm, SpO2 over 90% on less than 60% of FiO2, and hemodynamic stability with normal pH on minimal NIV settings (10/5 cm H₂O on Bilevel NIV or 10 cm H₂O on CPAP or less). Weaning can then be achieved by reducing the pressure support for 12 or 24 hours before disconnecting NIV, disconnecting from the NIV for 2 hours twice a day or more, or a combination of both [13].

Complications of NIV are mainly local, affecting the face skin due to tight masks or eye irritation with epistaxis due to mucosal dryness. Changing the mask, cushioning the bridge of the nose, and applying heated humidification may all help [14]. Gastric distention can occur, but vomiting and aspiration pneumonia are less likely. Nasogastric tube placement is not recommended for all patients on NIV; with however, caution patients with percutaneous endoscopic gastrostomy as pneumoperitoneum and tube displacement occur [15]. Serious NIV-related can complications, such as barotrauma, are rare compared to invasive mechanical ventilation [16]. Suctioning of secretions is routine during

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NIV; however, excess secretions, vomiting, facial injuries, bowel obstruction, and unconsciousness are the main contraindications of NIV. Severe bronchoconstriction can be dealt with by nebulizers delivered through a specific port which is universal in ventilator circuits [17].

Conclusion

Acute physicians are required to be confident in dealing with RF cases and initiating assisted ventilation, HFNC has shown to improve symptoms of dyspnoea and reduce rate of mechanical ventilation in patients with T1RF.

Treatment of the cause of RF should not delay early initiation of NIV as delayed introduction of NIV can lead to increase rate of NIV failure and requirement for mechanical ventilation when indicated and finally, close monitoring is essential when assisted ventilation is started and escalation plans or ceiling of treatment should be initiated early and communicated well with patients and the next of kin.

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