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Editorial

Failure of Non-Invasive Ventilation (NIV) in Acutely Hypercapnic COPD Patients: Importance of the Timely Use of Invasive Ventilation

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Worldwide, acute exacerbations of chronic obstructive pulmonary disease (COPD) is one of the leading causes of hospital admissions in the general medical ward, emergency department and the intensive care unit (ICU).

Since the mid-1990's, non-invasive ventilation (NIV) has significantly helped in the management of acute hypercapnic respiratory failure in COPD patients by significantly reducing the need for invasive ventilation thereby reducing the overall stay in the hospital, reducing morbidity and mortality and helping to improve the quality of life [1-4]. Consequently, non-invasive ventilation (NIV) is now the cornerstone in the treatment of a significant number of patients with an acute exacerbation of COPD.

While NIV is of significant importance in the treatment of these patients, a significant subset consisting of 15 - 30% of COPD patients ultimately fail to respond to NIV and require invasive mechanical ventilation. This is more apparent in patients with severe CO_2 retention, superadded bacterial infection such as lung parenchymal consolidation, multiple organ failure, altered mentation and severe respiratory fatigue [1,5]. Hence, it is of utmost importance for the discerning pulmonologist to identify the severity of respiratory failure at the earliest possible and decide on further appropriate management.

When necessary, the physician must not delay the decision to intubate the patient as prolonged NIV in such severe cases can result in prolonged ICU stay with increased duration on a ventilator resulting in possible ventilator-associated infections and increased mortality [6,7]. Hence, time is of the essence in such cases.

Hence, it is of paramount importance that a physician is able to determine with clinical confidence when NIV has been rendered ineffective and when invasive ventilation is necessary before further clinical deterioration.

Acute CO_2 retention with consequent hypercapnic respiratory failure is predominantly the result of increased airway resistance combined with air-trapping, ventilation-perfusion mismatch and respiratory muscle fatigue. Non-invasive ventilation helps combat this pathophysiology by improving the tidal volume (TV) and reducing the work of breathing. This is indeed beneficial because, if successful, NIV bypasses all potential complications associated with invasive ventilation [3].

Multiple randomized, controlled, multicentre, clinical trials have demonstrated a significant reduction in morbidity and mortality following the timely use of NIV in acutely hypercapnic COPD patients. Mortality reduced by nearly 40 - 50% [2,3]. As a result, both the American Thoracic Society (ATS) and the European Respiratory Society (ERS) recommend the use of BiPAP (bi-level NIV) in these patients [5].

Since, non-invasive ventilatory management is in reality a supportive therapy and not a therapeutic cure in COPD patients with severe hypercapnic respiratory acidosis, if the work of breathing continues to deteriorate despite the use of NIV, delaying intubation and invasive ventilatory therapy can significantly increase morbidity and mortality in these patients.

While oxygen saturation is an important marker of the patient's clinical condition, it cannot be considered the sole indicator of recovery. Altered mentation, respiratory muscle fatigue carbon dioxide retention (increased $PaCO_2$ levels), haemodynamic instability (altered vital parameters) and increased work of breathing with the active use of accessory muscles of respiration, often provide earlier and more meaningful indicators of impending respiratory failure.

Various studies have identified the clinical and laboratory predictors of failure of non-invasive ventilation. They include, severe respiratory acidosis with an arterial pH below 7.25 [8], tachypnoea with a respiratory rate above 30 - 35 breaths per minute after one to two hours of non-invasive ventilation which suggests failure of respiratory muscles and rising $PaCO_2$ levels despite bilevel pressure support, which indicates worsening alveolar ventilation.

Another alarming clinical finding is deterioration in the neurological status of these patients. Increasing confusion, agitation, refusal to wear the oxygen mask, and a declining Glasgow Coma Scale (GCS) serve as serious cause for concern since non-invasive ventilation essentially requires patient cooperation for effective use. Once patient cooperation disappears so does the effectiveness of NIV therapy.

Excessive respiratory effort, especially strong inspiratory effort during NIV use makes the patient prone to patient self-inflicted lung injury (P-SILI) [4]. In this condition, large negative intrathoracic pressure swings increase transpulmonary pressure thereby increasing lung injury even before intubation. As a result, continued use of NIV in these patients may contribute to more severe pulmonary damage.

Hence it is important to reassess patients within the first one or two hours following the commencement of NIV. Early physiological improvement is a strong clinical predictor of improvement in these patients. Indicators of physiological stabilisation include an improvement in arterial pH levels, reduction in tachycardia and tachypnoea, improvement in the blood pressure and improved mentation [5].

However, the absence of improvement in these clinical markers within a reasonable timeline is indicative of the need for invasive ventilation without further delay.

Physicians have adopted the concept of a "golden window" for invasive ventilation since failure of NIV is not usually precipitous but more gradual. While respiratory muscle fatigue may worsen gradually, compensatory mechanisms may still mask deterioration. During this period clinicians may be misguided by stable oxygen saturation levels despite rising $PaCO_2$ levels and increasing respiratory effort leading to loss of valuable time. Hence, by the time respiratory collapse occurs, the patient's condition is usually precarious and emergency intubation and invasive ventilation are accompanied by higher morbidity and mortality.

When emergency intubation is performed following severe respiratory fatigue there is usually a higher rate of hypotension, cardiac arrest, severe hypoxia and aspiration pneumonia. More so, these complications are compounded by the fact that in COPD patients there is already pre-existing hyperinflation which compromises venous return and cardiovascular stability.

Also, if intubation is delayed, patients are exposed to prolonged periods of inadequate ventilation. As a result, severe respiratory acidosis occurs which reduces myocardial contractility and predisposes to cardiac arrhythmias. Consequently, there is also reduced catecholamine responsiveness which may contribute to multi-organ dysfunction.

A constant dilemma which clinicians face is to determine when is the "right time" for intubation and invasive ventilation. In such instances, parameters over a period of time are more important than a single laboratory parameter. For example, if a patients pH improves from 7.23 to 7.30 over a couple of hours it is likely that he is responding despite residual acidosis. However, if the pH remains static or further deteriorates despite NIV management it is an indicator of treatment failure.

Clinical acumen integrating multiple parameters simultaneously should supersede reliance on any one single parameter. Heart rate, respiratory rate, haemodynamic parameters, signs of respiratory muscle fatigue, level of mentation, patient tolerance, serial arterial blood gas reports, secretion levels in the respiratory tract and the underlying disease process should all influence decision-making.

Situations where invasive ventilation is required more urgently include increased respiratory tract secretions which cannot be effectively cleared, increased risk of aspiration pneumonia and compromised airway protection.

Severe community-acquired pneumonia exacerbating the COPD significantly increases NIV failure rates compared with isolated exacerbations [6]. Also, severe life-threatening conditions such as cardiogenic shock, multi-organ failure and septic shock often require urgent invasive ventilation irrespective of blood gas findings.

Sedation should best be avoided in patients on NIV as far as possible as it frequently complicates the treatment. Administering sedation in order to maintain mask tolerance may result in suppression of the airway reflexes and may obscure neurological deterioration. Moreover, sedation may mask failing NIV therapy.

In patients on NIV it must be remembered that increasing inspiratory positive airway pressure may transiently improve tidal volume but progressively increasing ventilatory requirements often indicate worsening of the disease process. Further increase in inspiratory pressure may unnecessarily delay intubation while also causing discomfort, gastric insufflation, and mask-related complications.

Hence, invasive mechanical ventilation should be promptly used when needed without considering it as a therapeutic failure. Modern lung-protective ventilation strategies, careful management of dynamic hyperinflation, permissive hypercapnia where appropriate, and structured liberation protocols have significantly improved morbidity and reduced mortality in mechanically ventilated COPD patients.

Clinicians may rightly worry about prolonged ventilator dependence in COPD patients as it is easy to put a COPD patient on invasive ventilation but difficult to wean him off. Although this is true, the need to stabilize life-threatening respiratory failure outweighs the risks involved in an acute situation. Delayed intubation in the hope of avoiding mechanical ventilation frequently results in poor outcomes and higher morbidity and subsequent mortality.

Communication with patients and families is of vital importance. It should be explained that NIV is the next stage in ventilatory support when nasal oxygen fails to improve the clinical condition. However, invasive ventilation may become necessary if NIV fails to improve the patient clinically, and the patient continues to deteriorate.

Consequently, patient and relatives must be given the option of advanced decision-making. All patients with advanced COPD do not want to be subjected to invasive mechanical ventilation for various reasons. The patients wish should be respected and hence discussions should occur much before acute deterioration, whenever possible.

Therefore, if the patient decides against invasive ventilation, the physician must adopt a symptomatic and palliative line of management without subjecting the patient to invasive ventilation.

Standardized NIV monitoring protocols must be employed to assess the patient during hospitalisation. These include, heart rate, respiratory rate, arterial blood gas analysis, work of breathing, consciousness level, and haemodynamic status. A standardized protocol

helps to reduce variability between physicians especially regarding recognition of NIV failure with consequent early involvement of intensive care specialists.

A high-dependency unit (HDU) can be particularly useful in this situation as it can provide continuous monitoring, regular blood gas measurements, 24-hour experienced nursing care, and immediate availability of intubation and invasive ventilation, if needed. Such systems reduce the risk of patients remaining on NIV when it is no longer effective.

In summary, COPD management requires dynamic thinking wherein it is accepted that while NIV is useful in most patients, some patients may need invasive ventilation. It is important to know when the balance has shifted in favour of invasive ventilation and to adopt it without delay. Invasive ventilatory management must not be viewed as failure in management but rather as the next step in the treatment of a difficult case of COPD.

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