Section: Respiratory Medicine

Original Research Article

EARLY IDENTIFICATION OF NONINVASIVE VENTILATION FAILURE IN COPD PATIENTS USING THE HACOR SCORE: A PROGNOSTIC INDICATOR FOR IMPROVED OUTCOMES

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ABSTRACT

Background: Noninvasive ventilation is an important involvement for managing acute-on-chronic respiratory failure in non-COPD patients, reducing the need for invasive mechanical ventilation. However, a significant proportion of patients' involvement in NIV failure, leading to dishonoured consequences. Early identification of failure risk factors is critical to optimising patient management and improving survival rates.

Materials and Methods: This observational study was shown in the respiratory intensive care unit of a medical college hospital in India. The study was conducted from April, 2024 to March, 2025. This observational study analysed 60 non-COPD patients receiving NIV. Clinical and physiological parameters, as well as heart rate, respiratory rate, Glasgow Coma Scale score, arterial blood gases, and ventilator situations, were recorded at baseline and 1–2, 12, and 24 hours after NIV beginning. Odds ratios for NIV failure was calculated, and 1000 bootstrap samples were used to validate results.

Results: The NIV failure rate was 13.3%, with a hospital mortality rate of 10.0%. Patients who failed NIV had significantly higher heart rates at all time points ($P < 0.01$), lower GCS scores at 12h and 24h ($P = 0.02$), insistently lower pH levels ($P < 0.01$), and significantly reduced PaO_2/FiO_2 ratios at 12h and 24h ($P < 0.03$). The highest risk of NIV failure was observed at 12 hours (OR: 2.14, 95% CI: 1.52–3.02), with bootstrap analysis confirmative these results.

Conclusion: The study has concluded that that early monitoring of non-invasive ventilation (NIV) is crucial for predicting failure in patients with acute-on-chronic respiratory failure.

Keywords: Noninvasive ventilation, NIV failure, Acute-on-chronic respiratory failure, PaO_2/FiO_2 ratio, Glasgow Coma Scale, Arterial blood gases, Respiratory monitoring.

INTRODUCTION

Chronic obstructive pulmonary disease is an advanced respiratory condition characterised by airflow limitation and determined respiratory symptoms.[1] Acute exacerbations of COPD suggestively contribute to morbidity and mortality, often principal to hospital admissions and an enlarged healthcare problem.[2] Noninvasive ventilation has occurred as a foundation in the management of acute respiratory failure due to COPD exacerbations, reducing the need for endotracheal intubation and refining patient consequences.[3] However, an important subset of patients involves NIV failure, requiring timely identification and intervention to prevent further clinical deterioration.[4]

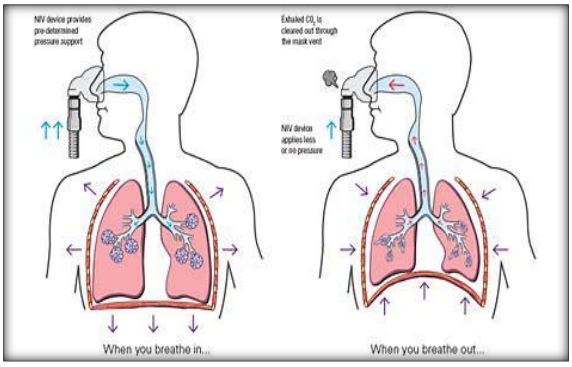

Figure 1: Noninvasive ventilation

NIV failure is well-defined as the incapability of the therapy to accomplish adequate gas exchange, resulting in the need for intubation or leading to adverse consequences, as well as mortality.[5] The reported failure rates differ between 20% and 50%, dependent on the severity of disease and patient-specific factors. The efficacy of NIV was assessed during the interference by monitoring important physiological limits, as well as arterial blood pH, PaO2, PaCO2, PaO2/FiO2 ratio, Glasgow Coma Scale, respiratory rate, and heart rate.[6] If respiratory failure presented signs of development, an effort was made to release the patient from NIV. The respiratory failure degenerated and met the criteria for invasive mechanical ventilation, intubation was achieved.[7]

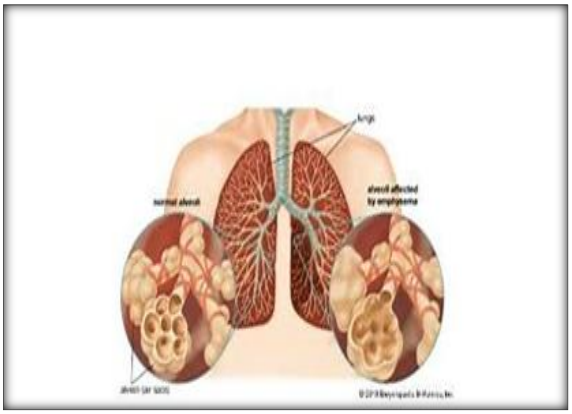

Figure 2: NIV in COPD

Classifying early predictors of NIV failure is serious for optimising management approaches and educating on enduring prediction.[8] Several physiological, clinical, and biochemical parameters have been examined for their role in forecasting NIV success or failure. These comprise arterial blood gas values, respiratory rate, hemodynamic parameters, and patient acceptance.[9]

Table 1: COPD patients common causes of NIV failure,[10]

Cause	Explanation
Severe Respiratory Acidosis	pH < 7.25 in spite of optimum NIV situations
High APACHE II Score	Higher severity of illness related to NIV failure.
Insistent Hypoxemia	Insufficient oxygenation despite FiO2 optimisation
Extreme Secretions	Incapability to clear secretions leading to mucus plugging.
Hemodynamic Instability	Hypotension or arrhythmias interfering with ventilation.
Neurological Impairment	Summary consciousness or agitation affecting NIV acceptance
Mask Embarrassment	Poor observance due to air leaks or pressure sores

The early documentation of patients at risk of NIV failure allows clinicians to make informed choices regarding the appreciation of care, including the need for intubation or alternative interferences.[11] Delayed acknowledgement of failure can lead to deteriorated outcomes, including augmented mortality, continued intensive care unit stays, and higher healthcare costs. Therefore, an evidence-based method to expect NIV failure is important in optimising patient care and resource distribution.[12]

Table 2: NIV Failure in COPD of early predictors,[13]

Predictor	Clinical Significance
High Respiratory Rate (>30 bpm)	Recommends inadequate ventilatory provision
pH < 7.25 on ABG	Designates severe acidosis and possible NIV failure
PaCO2 > 60 mmHg	Indicates deteriorating hypercapnia
PaO2/FiO2 < 200	Replicates poor oxygenation position
Use of Accessory Muscles	Recommends increased work of breathing
Glasgow Coma Scale < 12	Designates potential neurological concession
High Lactate Levels	Related to tissue hypoxia and poor prognosis

These important predictors of NIV failure in COPD patients with acute respiratory failure. We will travel numerous clinical and physiological markers, discuss risk stratification models, and survey the role of developing skills such as artificial intelligence and machine learning in early documentation. By sympathetic these factors, healthcare professionals can improve decision-making processes, reduce complications, and improve overall patient consequences.[14]

Table 3: Approaches to Reduce NIV Failure Risk,[15]

Approach	Justification
Early ABG Monitoring	Helps in appropriate adjustments of ventilator surroundings

Proper Mask Fit	Reduces air leaks and recovers patient comfort
Secretion Organisation	Prevents mucus plugging and enhances airway clearance
Hemodynamic Optimisation	Ensures stable blood pressure and cardiac function
Close Clinical Monitoring	Allows early detection of signs of decline
Adjusting NIV Situations	Individualised surroundings to match patient needs
Deliberation of High-Flow Nasal Cannula	Another in uncertain cases with mild ARF

By participating in these analyses and approaches in clinical practice, the success of NIV in COPD patients can be improved, refining overall patient consequences and reducing the need for invasive mechanical ventilation.[16]

MATERIALS AND METHODS

Research Design

This observational study was shown in the respiratory intensive care unit of a medical college hospital in India. The study was conducted from April, 2024 to March, 2025. The study protocol was accepted by the local ethics committee of the First Affiliated Hospital of Chongqing Medical University. Due to the observational landscape of the study, the necessity for informed consent was ignored. Non-invasive ventilation was managed using either the BiPAP Vision or V60 ventilators. Ventilator situations followed before recognised protocols, and modifications were made based on patients' biological responses, as well as PaCO2 levels and respiratory distress severity, and all the patients have COPD. Demographic and clinical information, with age, sex, Acute Physiology and Chronic Health Evaluation II scores, diagnosis, and comorbidities, were collected before NIV beginning. Functional limitations such as respiratory rate, heart rate, blood pressure, consciousness level, and arterial blood gas values were documented at baseline and 1–2, 12, and 24 hours following NIV beginning. Ventilator situations, as well as support pressure and positive end-expiratory pressure, were also documented at these times, opinions. Patients were followed up until discharge or death.

Inclusion Criteria

- Analysis of acute-on-chronic respiratory failure with respiratory acidosis.
- Beginning of NIV as a first-line treatment.
- PaCO2 level < 45 mmHg.
- pH > 7.35.

Exclusion Criteria

- Respiratory failure due to an aggravation of COPD
- Prophylactic use of NIV ensuing extubation.
- Release use of NIV for respiratory failure post-extubating.
- Unintentional extubating followed by NIV submission.
- Use of a high-flow nasal cannula before or after NIV beginning.

Statistical Analysis

The HACOR score was considered at baseline and 1–2, 12, and 24 hours after NIV beginning. This score, ranging from 0 to 27, measured five important variables: heart rate, acidosis, consciousness, oxygenation, and respiratory rate. Higher HACOR scores specified an increased risk of NIV failure. Continuous variables were stated as means with standard deviations or medians with interquartile ranges as suitable. Comparisons between groups were conducted using independent sample t-tests or Mann–Whitney U tests for continuous variables and chi-square or Fisher's exact tests for categorical variables. The prognostic ability of the HACOR score for NIV failure was assessed using the area under the receiver operating characteristic curve. The optimal cutoff value was resolved based on the maximal Youden index. To the inside validate results, 1000 bootstrap samples were used to approximate the odds ratio and 95% confidence interval per 1-point increase in the HACOR score. A two-sided p-value of <0.05 was measured significant differences.

RESULTS

In 60 non-COPD patients with acute-on-chronic respiratory failure, the condition was sleeping apnoea-hypopnea syndrome 21 (35%), followed by chronic thoracic sequelae 14 (23.3%) and bronchiectasis 13 (21.7%). Less common conditions included chest wall deformity 6 (10%), obesity-hypoventilation syndrome 2 (3.3%), and other respiratory disorders 4 (6.7%). The complete non-invasive ventilation failure rate was 13.3%, with one patient (1.7%) acceding during NIV. There were no significant differences in age, sex, diagnosis, diseases, or prevalence of chronic respiratory conditions between those who qualified successfully for NIV and those who had NIV failure (Table 4).

Table 4: Condition of non-COPD patients with Acute on Chronic Respiratory Failure

Condition	Cases	Percentage (%)
Sleep apnoea-hypopnea syndrome	21	35.00%
Chronic thoracic sequelae	14	23.30%
Bronchiectasis	13	21.70%
Chest wall deformity	6	10.00%
Obesity-hypoventilation syndrome	2	3.30%
Other conditions	4	6.70%
Total	60	100%

Median NIV duration	96 hours	-
NIV Failure	8	13.3%
Hospital Mortality	6	10%

In 60 patients, important biological differences between successful and failed NIV cases emerged within the first 24 hours. Patients who qualified for the NIV failure had significantly higher heart rates at all time points ($P < 0.01$), lower GCS scores at 12h and 24h ($P = 0.02$), and persistently lower pH levels ($P < 0.01$), suggesting greater respiratory distress. In addition, the PaO_2/FiO_2 ratio was significantly lower in NIV failure patients at 12h and 24h ($P < 0.03$), indicating impaired oxygenation, while insistently elevated $PaCO_2$ levels at 24h ($P = 0.01$) suggested inadequate CO_2 clearance (Table 5).

Table 5: Variations in Vital Signs and Ventilator Parameters

Parameter	Successful NIV (Mean ± SD)	NIV Failure (Mean ± SD)	P-value
Before NIV			
Heart rate (bpm)	107 ± 22	122 ± 23	<0.01
Respiratory rate (bpm)	29 ± 6	28 ± 5	0.68
Mean arterial blood pressure (mmHg)	101 ± 16	103 ± 22	0.76
GCS score	14.5 ± 1.2	14.2 ± 1.2	0.38
pH	7.26 ± 0.07	7.22 ± 0.07	0.01
$PaCO_2$ (mmHg)	81 ± 18	77 ± 17	0.28
PaO_2/FiO_2 (mmHg)	199 ± 99	173 ± 79	0.28
12 Hours of NIV			
Heart rate (bpm)	89 ± 16	113 ± 31	<0.01
Respiratory rate (bpm)	22 ± 4	22 ± 3	0.85
Mean arterial blood pressure (mmHg)	88 ± 11	91 ± 14	0.48
GCS score	14.8 ± 0.5	14.5 ± 0.7	0.02
pH	7.38 ± 0.05	7.27 ± 0.12	<0.01
$PaCO_2$ (mmHg)	65 ± 15	71 ± 22	0.19
PaO_2/FiO_2 (mmHg)	241 ± 86	182 ± 64	0.03
Support pressure (cmH_2O)	18 ± 4	18 ± 3	0.89
PEEP (cmH_2O)	7 ± 3	6 ± 2	0.53
24 Hours of NIV			
Heart rate (bpm)	87 ± 17	105 ± 30	<0.01
Respiratory rate (bpm)	23 ± 4	25 ± 6	0.11
Mean arterial blood pressure (mmHg)	90 ± 12	92 ± 23	0.61
GCS score	14.9 ± 0.9	14.2 ± 0.8	0.02
pH	7.40 ± 0.07	7.29 ± 0.14	<0.01
$PaCO_2$ (mmHg)	59 ± 15	73 ± 33	0.01
PaO_2/FiO_2 (mmHg)	256 ± 80	171 ± 68	<0.01
Support pressure (cmH_2O)	19 ± 4	20 ± 3	0.78
PEEP (cmH_2O)	7 ± 3	6 ± 2	0.32

The odds of NIV failure gradually increased over time, with the highest risk observed at 12 hours (OR: 2.14, 95% CI: 1.52–3.02), signifying that early biological corrosion is a strong predictor of NIV failure. After applying 1000 bootstraps, the confidence intervals widened, mainly at 12 hours (1.60–6.19) and 24 hours (1.15–3.85), reflecting enlarged variability but maintaining the same complete. The constancy of the OR estimates before and after bootstrapping supports the robustness of these findings. These results, critical importance of monitoring patients closely within the first 12–24 hours of NIV to identify those at a higher risk of failure and consider timely escalation of care (Table 6).

Table 6: Odds Ratios (OR) for NIV Failure at Different Time Points

Time Point	OR (95% CI)	OR (95% CI) Under 1000 Bootstraps
Before NIV	1.15 (1.04–1.28)	1.15 (1.04–1.31)
1-2h of NIV	1.99 (1.50–2.64)	1.99 (1.59–3.28)
12h of NIV	2.14 (1.52–3.02)	2.14 (1.60–6.19)
24h of NIV	1.53 (1.18–1.98)	1.53 (1.15–3.85)

DISCUSSION

Early identification of non-invasive ventilation failure in chronic obstructive pulmonary disease patients is important to improving clinical consequences and reducing morbidity and mortality. In spite of its efficacy, a significant proportion of patients with acute hypercapnic respiratory failure secondary to COPD aggravation do not answer adequately to NIV, requiring quick recognition of failure indicators to facilitate timely interference.[17] One of the important causes of NIV failure in COPD is the severity of respiratory acidosis at initiation. Studies have shown that patients with a pH lower than 7.25 at presentation are at a higher risk of NIV failure. This is probably due to the severity of

underlying respiratory muscle fatigue and the inability of the patient to generate an adequate compensatory response to NIV.[18] Monitoring arterial blood gases within the first 1–2 hours after initiating NIV is critical, as persistent or worsening hypercapnia and academia propose an inadequate response and the need for escalation to invasive mechanical ventilation.[19]

Additional dangerous factor is the level of awareness. The Glasgow Coma Scale has been extensively used to measure neurological status in patients on NIV. A low GCS score (< 8) often indicates severe hypercapnic encephalopathy, which is a forecaster of NIV failure. Patients who remain drowsy or unresponsive despite adequate ventilation settings may require endotracheal intubation to prevent further deterioration.[20] The presence of severe dyspnoea and tachypnoea also serves as an important early indicator of NIV failure. Determined respiratory rate above 30 breaths per minute after 1–2 hours of NIV, propose ongoing respiratory distress and poor variation to the ventilator. This frequently indicates excessive work of breathing, despite ventilatory support, and may require invasive intervention. In difference, a significant reduction in RR post-NIV initiation is related with improved outcomes.[21]

Hypoxemia that does not recover with NIV is an added strong predictor of failure. While NIV efficiently corrects hypercapnia, patients who endure to exhibit profound hypoxemia despite optimal situations may have important ventilation-perfusion mismatch, pulmonary embolism, or pneumonia. Continuous monitoring of oxygen saturation and assessment of the need for increased inspired oxygen concentration are essential in these cases. In addition, hemodynamic instability is a concerning sign of impending NIV failure.[22] Hypotension, arrhythmias, or signs of shock propose an inadequate physiological response to NIV or a fundamental difficulty such as sepsis or myocardial infarction. These conditions may require additional interventions, including vasopressor support and invasive ventilation.[23]

The patient's acceptance and cooperation with NIV play a critical role in its success. Factors such as extreme air leaks, discomfort due to mask intolerance, or incapability to coordinate with the ventilator may conciliation its effectiveness. Poor observance due to anxiety, confusion, or severe anxiety can lead to early withdrawal, reducing the chances of success.[24] Providing sedation when necessary, optimising mask fit, and ensuring adequate patient education can enhance adherence. Radiological and clinical results, including deteriorating infiltrates on chest X-ray or signs of aspiration pneumonia, can also predict NIV failure. The presence of new lung infiltrates often suggests an underlying infectious process or fluid overload, both of which can impair gas exchange and necessitate intubation.[25]

Biomarkers such as raised serum lactate, C-reactive protein, and procalcitonin levels have been discovered as potential predictors of NIV failure. High lactate levels specify tissue hypoxia and poor systemic perfusion, whereas raised inflammatory markers may propose an ongoing infection or exacerbation-related inflammation, both of which can worsen respiratory function. Eventually, a combination of clinical, biochemical, and physiological parameters should be used to identify NIV failure early.[26] The addition of these gauges into clinical practice can help guide appropriate decision-making, stopping unnecessary suspensions in starting invasive ventilation when required. Future research should focus on emerging authorised predictive models, joining machine learning and artificial intelligence to enhance early identification and individualised patient organisation.[27]

The primary signs of NIV failure in COPD patients are critical for optimising treatment approaches and refining patient consequences. Systematic monitoring of ABG, respiratory rate, consciousness level, hemodynamic parameters, and obedience to NIV can help clinicians make timely interferences. A structured method to identifying high-risk patients can aid in dropping difficulties, hospital length of stay, and overall mortality in this susceptible inhabitant.[28]

CONCLUSION

The study has concluded that that early monitoring of non-invasive ventilation (NIV) is crucial for predicting failure in patients with acute-on-chronic respiratory failure. Key indicators such as elevated heart rate, lower GCS scores, and abnormal pH and PaO_2/FiO_2 ratios within the first 24 hours are strong predictors of NIV failure. The odds of NIV failure increase significantly over time, especially at 12 hours, highlighting the importance of early intervention and timely escalation of care for at-risk patients. These findings emphasize the need for close monitoring during the initial stages of NIV to improve patient outcomes. The significance of early biological changes in predicting NIV failure in non-COPD patients with acute-on-chronic respiratory failure. Important indicators such as determined tachycardia, deteriorating pH, decreased PaO_2/FiO_2 ratio, and lower GCS scores within the first 12–24 hours provide valuable perceptions for clinical decision-making. The maximum risk of NIV failure was detected at 12 hours, underscoring the need for early and frequent patient monitoring. Realising an active method based on these predictive markers may help optimise NIV success, reduce the need for invasive ventilation, and improve overall patient consequences.

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