Section: Miscellaneous

Original Research Article

UTILITY OF NON INVASIVE POSITIVE PRESSURE VENTILATION IN PATIENTS OF ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH RESPIRATORY FAILURE

Shaik Umar Pasha[1], Shaik Mohammed Ansari[2], K. Prabhakar[3]

[1]Assistant Professor, Department of Respiratory Medicine, Government Medical College & General hospital, Narsampet, Warangal, Telangana, India.
[2]Assistant Professor, Department of Pulmonology Medicine, VRK Women's Medical College & Research Institute, Hyderabad, Telangana, India.
[3]Assistant Professor, Department of Pulmonary Medicine, Osmania Medical College Government General and Chest Hospital, Hyderabad, Telangana, India.

Received *: 17/03/2026*
Received in revised form *: 27/04/2026*
Accepted *: 13/05/2026*

Corresponding Author:
Dr. Shaik Umar Pasha,
Assistant Professor, Department of Respiratory Medicine, Government Medical College & General hospital, Narsampet, Warangal, Telangana, India.
Email: shanu.thenur@gmail.com

DOI: 10.70034/ijmedph.2026.2.655

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (2); 3986-3993

ABSTRACT

Background: The aim is to study the predictors of noninvasive ventilation in patients with COPD acute exacerbation.

Materials and Methods: It was a prospective observational study, 'Patients of Acute Exacerbation of COPD with Respiratory Failure "admitted in NRI General Hospital in the department of' Respiratory medicine'. The study group consisted of 50 patients of all age groups. Patients with decreased oxygen, severe respiratory acidosis, acid-base balance and with respiratory failure were kept on NIV according to the ICU protocol with criteria and guidelines.

Results: The mean PR was 95.40 ± 20.33 BPM, mean SBP was 138.16 ± 21.11 mm Hg, mean DBP was 93.72 ± 16.20 mm of Hg, RR was 32.28 ± 7.10 BPM, temperature was 97.40 ± 1.84 F, SPO2 on RA was 85.86 ± 7.68%. 68% had BIPAP, 32% had CPAP. The mean PH at 0 hr was 7.36 0.10, and at 24 hr it was 7.39 0.06, with no statistically significant difference in PH with time. Mean PCO2 at 0 hour was 48.03 ± 14.88 and the mean PCO2 at 24th hour was 40.82 ± 6.02. a statistically significant difference was observed in case of PCO2 across time. At 0 hour, the mean P02 was 60.06+_ 6.31, and at 24 hours, the mean PO2 was 106.34 +_28.58. Over time, there was a considerable variation in the mean value of Po2. After 0 hour, the mean HCO3 was 24.37+_6.15, and at 24 hours, it was 24.66 5.56. Over time, there was no statistically significant difference in mean HCO3 levels.. In this current study, the mean lactate at 0 hour was 2.28 ± 1.26 and the mean lactate at 24 hours was 2.08 ± 1.41. In the current study' the mean SPO2 on room air was 85.86 ± 7.68 and the mean SPO2 on ventilation was 95.12 ± 3.27. A highly significant difference was observed in the mean SPO2 values with and without ventilation. In this current study 28% of the patients stayed for <5 days, 52% stayed for a duration of 6-10 days, 20% stayed for a duration of 11-15 days. In the present study, 88% had a successful ventilation, whereas 12% had a failed ventilation and needed an intubation.

Conclusion: The present study concluded that, patients on NIPPV with respiratory failure should be monitored for changes in HR, RR, pH, and PaO2 at regular intervals, so that patients who require invasive ventilation should be intubated as soon as possible to avoid an unnecessary increase in morbidity and death.

Keywords: COPD, NIPPV, SPO2, acute exacerbation, Intubation, Non invasive ventilation.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common and treatable disease characterised by "persistent respiratory symptoms and airflow limitations that is due to airway and or alveolar abnormalities usually caused by significant exposure to noxious particles or gases and influenced by host factors including abnormal lung development". Significant comorbidities may have an impact on morbidity and mortality. Patients Symptoms may range from being asymptomatic to respiratory failure.[1]

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) launched by World Health Organization (WHO) and National Heart, Lung and Blood Institute (NHLBI).[1] The GOLD report for 2019 outlines a simplified method of evaluating and choosing initial treatment for COPD patients. The refined ABCD evaluation method directs healthcare professionals to define the seriousness of the disorder and the GOLD category classification. Once the diagnosis of COPD has been confirmed by spirometry (FEV1 / FVC < 0.7), the FEV1 is used to assess the severity (GOLD rating 1-4). The GOLD group (A-D) is then defined by the severity of the symptoms and the history of exacerbation.

The principal cause of hypoxemia in patients with COPD is ventilation/perfusion (V/Q) abnormality caused due to "progressive airflow limitation and emphysematous destruction of the pulmonary capillary bed".[2] Development of pulmonary hypertension in patients with COPD3 is mainly influenced by "alveolar hypoxia".

"Physiological balance between vasodilation and vasoconstriction is lost in patients with severe COPD". hypoxia leads to development of endothelial dysfunction.

"Non-invasive ventilation (NIV) is themethod of ventilation support through the upper airway of the patient using masks or other similar devices".[3] NIV is different from those which bypass the upper airway with a tracheal tube, laryngeal mask, or tracheostomy which are invasive.[4] It is often used for copd acute exacerbation because these exacerbations can be reversed quickly and because the hypercapnic ventilation failure that occurs in patients with this disorder appears to respond well to non-invasive ventilation.[4-8]

The desire to avoid complications of invasive ventilation has been a major driving force behind the increasing use of non-invasive ventilation. Although "invasive mechanical ventilation is" highly effective and reliable to support alveolar ventilation, endotracheal intubation carries well-known risks of complications. These fall into three main categories: 'complications caused due to the intubation and mechanical ventilation process, those caused by the loss of airway defence mechanisms and complications which occur following the removal of the endotracheal tube.[9]

This technique has been shown to effectively 'Improve acute respiratory failure (ARF), in particular ventilator-associated pneumonia.[10]

"The main modality is non-invasive pressuresupport (NIPSV) ventilation". Other Older modalities, such as (CPAP) and those recently came into the market, have been successful in the setting up of ARF.

"Recent US survey showed that the use of NIV to treat patients of acute exacerbations of chronic obstructive pulmonary disease (COPD) increased by more than 400 per cent in 10 years '(from 1 per cent in 1998 to 4.5 per cent in 2008) 'was associated with a 42 per cent reduction in IMV'.

Potential causes of NIV failure may include lack of trained personnel, over-reliance on NIV effectiveness, comorbidity, and lack of specific recommendations for optimal duration and NIV settings.

The setting and duration of the NIV shall be determined by the physician in charge and personal or group experience.

Aim: To study the predictors of noninvasive ventilation in patients with COPD acute exacerbation.

Objectives:

1. Hours of NIV (1st 72 Hours), Day 4, Day 7.
2. Ph, Pco2, p02, Hco3
3. NIV settings (IPAP /EPAP)
4. Previous exacerbation / previous NIV
5. BMI/ other co morbidities
6. Pack years of smoking.

MATERIALS AND METHODS

Study population: ''Patients of Acute Exacerbation of COPD with Respiratory Failure "admitted in NRI General Hospital in the department of' RESPIRATORY MEDICINE.

Study design: prospective observational study

Inculusion criteria

Exclusion Criteria:

1. Patients Age <20 years.
2. Extreme old age patients (>80 years of age)
3. Patients requiring invasive ventilation.
4. Moribund patients.
5. Non cooperative patients

Method of data collection

The study group consisted of 50 patients of all age groups. Collected demographic details including age, ABG analysis, blood investigations, GCS, socio-economic status; occupation, personal history and other comorbidities. Patients with decreased oxygen, severe respiratory acidosis, acid-base balance and with respiratory failure were kept on NIV according to the ICU protocol with criteria and guidelines.

Patients vitals such as oxygen saturation, heart rate, respiratory rate, blood pressure were monitored and the settings were adjusted according to the patients comfortability and improvement. Patients who improved were shifted to ward and advised the homebased non-invasive ventilation. Patients with

poor gcs and worsened clinical conditions and shown no improvement with NIV were intubated.

Statistical Analysis: The data was entered in excel sheet and analyzed using SPSS (Version 16). Descriptive statistics with mean, standard deviation and proportions (%) were calculated for continuous variables. Totest the hypothesis Chi Square test,40 independent sample t test and paired t tests were used appropriately.p value <0.05 was considered as statistically significant.

RESULTS

Table 1: Age distribution

Age distribution(years)	Frequency (n)	Percentage (%)
20 – 30	4	8%
31 – 40	6	12%
41 – 50	6	12%
51 – 60	13	26%
61 – 70	13	26%
71 – 80	8	16%
Total	50	100%
Mean ± SD	56.06 ± 15.32	

The participants' average age was 56.06 15.32 years, with 8% belonging to the age groups of 20-30 years, 12 percent to the age group of 31-40 years, 12 percent to the age group of 41-50 years, 26 percent to the age group of 51-60 years, 26 percent to the age group of 61-70 years, and 16 percent to the age group of 71-80 years.

Table 2: Gender distribution

Gender distribution	Frequency (n)	Percentage (%)
Male	34	68%
Female	16	32%
Total	50	100%

In the present study, 68% of the participants were male and 32% of the participants were female

Table 3: Presenting complaints

Presenting complaints	Frequency (n)	Percentage (%)
Dyspnea	50	100%
Chest pain	50	100%
Cough with expectoration	50	100%
Fever	21	42%
Vomiting	7	14%
Fatigue	9	18%
Loss of Appetite	2	4%
Loss of weight	3	6%
Total	50	100%

In the present study, all the cases had dyspnoea, chest pain and cough with expectoration. 42% of the cases had fever, 14% of the cases had vomiting, 18% of the cases had fatigue, 4% of the cases had loss of appetite, 6% of the cases had loss of weight.

Table 4: Past history

Past History	Frequency (n)	Percentage (%)
Smoker	25	50%
Biomass exposure	19	38%

In the present study, 50% of the cases had previous history of smoking and 38% had history of biomass exposure

Table 5: Type of respiratory failure

Type of respiratory failure	Frequency (n)	Percentage (%)
Type 1	27	54%
Type 2	23	46%
Total	50	100%

In the present study 54% of the participants had type 1 respiratory failure and 46% had type 2 respiratory failure.

Table 6: Co-morbidities

Co morbidities	Frequency	Percentage
Diabetes	9	18%
Systemic hypertension	15	30%
CAD	6	12%
CKD	2	4%

In the present study, 18% had diabetes, 30% had systemic HTN, 12% had CAD and 4% of the subjects had CKD.

Table 7: Body mass index (kg/m2)

BMI	Frequency	Percentage
<18.5	3	6%
18.6 – 24.9	24	48%
25 – 29.9	16	32%
>30	7	14%
Total	50	100%

In the present study, 6% were having a BMI <18.5, 48% had a BMI of 18.6-24.9, 32% had BMI between 25-29.9 and 14% had BMI >30.

Table 8: Baseline vitals in the study population on admission

Vitals	Mean ± SD
Pulse Rate / min	95.40 ± 20.33
SBP	138.16 ± 21.11
DBP	93.72 ± 16.20
RR	32.28 ± 7.10
Temp	97.40 ± 1.84
SPO2 on Room Air	85.86 ± 7.68

The mean PR was 95.40 ± 20.33 BPM, mean SBP was 138.16 ± 21.11 mm Hg, mean DBP was 93.72 ± 16.20 mm of Hg, RR was 32.28 ± 7.10 BPM, temperature was 97.40 ± 1.84 F, SPO2 on RA was 85.86 ± 7.68%

Table 9: ABG Analysis

ABG analysis	Mean ± SD
PH 0 Hr	7.36 ± 0.10
Ph 1st Hr	7.37 ± 0.08
PH 4th Hr	7.39 ± 0.079
Ph 24th Hr	7.39 ± 0.06
Pco2 0 Hr	48.03 ± 14.88
Pco2 1 Hr	46.26 ± 13.88
Pco2 4 Hr	45.34 ± 13.29
Pco2 24 Hr	40.82 ± 6.02
Po2 0 Hr	60.06 ± 6.31
Po2 1 Hr	117.58 ± 31.67
Po2 4 Hr	113.37 ± 24.65
Po2 24 Hr	106.34 ± 28.58
HCO3 0 Hr	24.37 ± 6.15
Hco3 1 Hr	25.28 ± 6.12
Hco3 4 Hr	24.70 ± 6.26
Hco3 24 Hr	24.66 ± 5.56
Lactate 0 Hr	2.28 ± 1.26
Lactate 1 Hr	2.23 ± 1.41
Lactate 4 Hr	2.18 ± 1.33
Lactate 24 Hr	2.08 ± 1.41

Table 10: Mode of NIV

MODE	Frequency (n)	Percentage (%)
BIPAP	34	68%
CPAP	16	32%
Total	50	100%

In the present study, 68% had BIPAP, 32% had CPAP

Table 11: NIV – ABG analysis - PH

ABG analysis	Mean ± SD
PH 0 Hr	7.36 ± 0.10
Ph 24th Hr	7.39 ± 0.06
Paired t test = 2.28 , p= 0.02*, Statistically significant	

The mean PH was 7.36 0.10 at 0 hr and 7.39 0.06 at 24 hr, and 'there was no statistically significant difference in PH over time'

Table 12: NIV – ABG analysis - Pco2

ABG analysis	Mean ± SD
Pco2 0 Hr	48.03 ± 14.88
Pco2 24 Hr	40.82 ± 6.02
Paired t test = 2.98 p= 0.004*, Statistically significant	

Mean PCO2 at 0 hour was 48.03 ± 14.88 and the mean PCO2 at 24th hour was 40.82 ± 6.02. a statistically significant difference was observed in case of PCO2 across time

Table 13: NIV – ABG analysis - Po2

ABG analysis	Mean ± SD
Po2 0 Hr	60.06 ± 6.31
Po2 24 Hr	106.34 ± 28.58
Paired t test = 11.09, p= 0.0001*, Statistically significant	

The mean P02 at 0 hour was 60.06 ± 6.31 and the mean PO2 24 hr was 106.34 ± 28.58. Over time, there was a considerable variation in the mean value of Po2.

Table 14: NIV – ABG analysis - HCO3

ABG analysis	Mean ± SD
HCO3 0 Hr	24.37 ± 6.15
HCO3 24 Hr	24.66 ± 5.56
Paired t test = 0.31, p= 0.75, Not Statistically significant	

In mean HCO3 at 0 hour was 24.37 ± 6.15 and the mean HCO3 at 24 hours was 24.66 ± 5.56. The mean HCO3 levels did not change in a statistically meaningful way over time.

Table 15: NIV – ABG analysis - Lactate

ABG analysis	Mean ± SD
Lactate 0 Hr	2.28 ± 1.26
Lactate 24 Hr	2.08 ± 1.41
Paired t test = 1.13, p= 0.26, Not Statistically significant	

“In this present study, the mean lactate at 0 hour was 2.28 ± 1.26 and the mean lactate at 24 hours was 2.08 ± 1.41”. no statistically significant difference was observed in terms of lactate across the time.

Table 16: SPO2 on room air and on ventilation

	Mean ± SD
SPO2 on room air	85.86 ± 7.68
SPO2 on ventilation	95.12 ± 3.27
Paired t test = 8.17, p= 0.0001*, Statistically significant	

In the present study the mean SPO2 on room air was 85.86 ± 7.68 and the mean SPO2 on ventilation was 95.12 ± 3.27. A highly significant difference was observed in the mean SPO2 values with and without ventilation

Table 17: Duration of hospital stay

Duration of hospital stay	Frequency	Percentage
<5 days	14	28%
6 – 10 days	26	52%
11 – 15 days	10	20%
Total	50	100%

In the present study 28% of the patients stayed for <5 days, 52% stayed for a duration of 6-10 days, 20% stayed for a duration of 11-15 days

Table 18: Outcome of NIV

Outcome of NIV	Frequency	Percentage
Success	44	88%
Failure (Required Intubation)	6	12%
Total	50	100%

“In this present study, 88% had a successful ventilation “, whereas 12% had a failed ventilation and needed an intubation.

Table 19: Follow up ABG Values

	Day 1	Day 4	Day 7
Ph	7.39 ± 0.07	7.41 ± 0.06	7.41 ± 0.05
PCO2	51.49 ± 10.12	45.82 ± 9.09	39.77 ± 4.02
HCO3	25.28 ± 6.13	24.71 ± 6.26	24.70 ± 5.52
PO2	83.22 ± 7.05	91.45 ± 5.52	97.59 ± 2.17
Lactate	2.24 ± 1.41	2.18 ± 1.34	2.08 ± 1.42

DISCUSSION

The transmission of positive pressure to the lungs without the use of an endotracheal tube is referred to as noninvasive ventilation (NIV). Continuous positive airway pressure (CPAP) and all modalities of pressure controlled mechanical ventilation are examples of noninvasive ventilation. [Noninvasive positive pressure ventilation (NIPPV)] (NIPPV)].[11]
Acute respiratory failure is often marked by life-threatening changes in arterial blood gases and acid–base balance. Hypercapnic or hypoxemic acute respiratory failure are two types of acute respiratory failure. When the percentage of oxygen in inspired air (FiO2) is 0.60 or above, hypercapnic and hypoxemic respiratory failure are defined as PaCO2 greater than 45 mm Hg and PaO2 less than 55 mm Hg, respectively.[12]
The participants' average age was 56.06+_ 15.32 years, with 8% belonging to the age groups of 20-30 years, 12 percent to the age group of 31-40 years, 12 percent to the age group of 41-50 years, 26 percent to the age group of 51-60 years, 26 percent to the age group of 61-70 years, and 16 percent to the age group of 71-80 years. The average age of the participants did not differ significantly between the groups.

Gender predominance:

'In this study', 68% of the participants were male and 32% of the participants were female.
A male predominance was observed 'In this study' and the similar finding has been observed in the study done by Dave et al,[13] in which the ratio was observed to be 8.09, in Rachaiah et al,[14] it was 6.02 and this finding was in concordance with the findings made with the present study. Several other studies by Begum et al,[15] and Jain BK et al,[16] has also opined that COPD is having a male predominance.
This male preponderance observed in many studies has been attributed to the habit of smoking among men, which was found to be a very strong predisposing factor as well as risk factor for COPD. And only those female who were exposed to indoor air pollution had increased risk of having COPD.

Presenting complaints: In this study, all the cases had dyspnoea, chest pain and cough with expectoration. 42% of the cases had fever, 14% of the cases had vomiting, 18% of the cases had fatigue, 4% of the cases had loss of appetite, 6% of the cases had loss of weight.
Vikhe et al. did a study.17 Of 50 patients, 47 patients had cough, 45 patients had sputum and 44 patients had dyspnoea as symptoms on admission. And this sort of presentation is similar and in concordance to the presentation seen in the present study.
A similar symptomatology at presentation to the hospital is found in the study done by Singhal et al,[18] where the most common symptom was cough at the time of presentation followed by cough with sputum production.
At the time of admission, 12 percent of the patients in this research developed cyanosis. Pedal oedema was found in 6% of patients with predominant emphysema, while pursed lip breathing was found in 10% of patients with predominant emphysema. Intercostal and distended neck veins were found in 6% of the participants. Only 2% of the patients were found to have clubbing.
In Vikhe et al,[17] study. A total of 30 (60%) patients had signs of edema and 27 (54%) patients had Loud P2 on auscultation. This sort of presentation is discordant with the findings 'made in this study 'where cyanosis was the most prevalent sign whereas edema is seen in a meagre of 6%.

Past history/ risk factors: In the present study, 50% of the cases had previous history of smoking and 38% had history of biomass exposure.
In a similar study conducted by Tenmozhi et al,[19] prevalence of smoking is seen to be the single most important risk factor in 80% of the patients.
A similar conclusion was made in a study by Racahaih et al,[14] in which 88 percent of COPD patients smoked, all of whom were male and none of whom were female. This finding made was in concordance with the present study. They have also observed that the usage of fire wood has been the predisposing factor for COPD among female and this was also in concordance with the present study finding.

Types of respiratory failure: 54 %of the individuals in this study had type 1 respiratory failure, whereas 46 percent had type 2 respiratory failure. Arsude et al. discovered a near consonant discovery in their research.[20]

Co-morbidities: In the current study, 18 percent of the participants had diabetes, 30% had systemic HTN, 12% had CAD, and 4% had CKD.

BMI: 6% were having a BMI <18.5, 48% had a BMI of 18.6-24.9, 32% had BMI between 25-29.9 and 14% had BMI >30 in the current study.

Baseline vitals observed in the present study: The mean PR was 95.40 ± 20.33 BPM, mean SBP was 138.16 ± 21.11 mm Hg, mean DBP was 93.72 ± 16.20 mm of Hg, RR was 32.28 ± 7.10 BPM, temperature was 97.40 ± 1.84 F, SPO2 on RA was 85.86 ± 7.68%. In the current study, 68% had BIPAP, 32% had CPAP.

NIV- ABG analysis: The mean PH at 0 hr was 7.36 0.10, and after 24 hr it was 7.39 0.06, with no statistically significant difference between the two.
Mean PCO2 at 0 hour was 48.03 ± 14.88 and the mean PCO2 at 24th hour was 40.82 ± 6.02. a statistically significant difference was observed in case of PCO2 across time.
The mean P02 at 0 hour was 60.06 ± 6.31 and the mean PO2 24 hr was 106.34 ± 28.58. there was a significant difference in the mean value of Po2 over duration.
At 0 hour, the mean HCO3 was 24.37 6.15, and at 24 hours, it was 24.66 5.56. Over time, there was no statistically significant difference in mean HCO3 levels.
The mean lactate at 0 hour was 2.28 1.26, while the mean lactate at 24 hours was 2.08 1.41 in this study.

In terms of lactate, there was no statistically significant variation over time.

Saturations over time with ventilation: In the current study 'the mean 'SPO2 on room air was 85.86 ± 7.68 and the mean SPO2 on ventilation was 95.12 ± 3.27. A highly significant difference was observed in the mean SPO2 values with and without ventilation

Duration of hospital stay: 'In the current study' 28% of the patients stayed for <5 days, 52% stayed for a duration of 6-10 days, 20% stayed for a duration of 11-15 days.

Outcomes: With clinical and ABG improvement, success was defined as the avoidance of endotracheal intubation. Failure was defined as patients who did not improve clinically or on ABG values with NIPPV and required intubation.

In this study, 88 percent of the participants had successful ventilation, while 12 percent had failed ventilation and required intubation. Ventrella et al. and George et al. observed similar results in their investigations, claiming that NIV was successful in (81%) and (85%) of patients with acute respiratory failure, respectively.[21]

Improvement in baseline ABG values including pH, PCO2, and PO2 within or after 24 hours of NIV assistance exhibited a strong link with a favourable outcome in our study. In their investigation, Bhattacharyya et al104 recruited 100 patients with acute respiratory failure and discovered that improvements in pH, PCO2, and PO2 after 1, 4, and 24 hours of NIV assistance had a strong relationship with successful outcome (P 0.0001 for each). Singh et al.100 observed that improvement in ABG parameters such as pH and PCO2 after 1 and 4 hours of NIV assistance in patients with acute hypercapnic respiratory failure had a good outcome in their research of 50 patients with acute respiratory failure of various etiologies.[22]

Clinical and blood gas indicators improved significantly when NIPPV was used. In both type I and type II respiratory failure patients, the modified Borg's dyspnea score improved considerably with NIPPV at 4, 12, and 24 hours compared to baseline. A study by Kramer et al. backed up these findings.[23] At the end of 4, 12, and 24 hours, respiratory rate and HR in both type I and type II acute respiratory failure groups considerably decreased, indicating improvement with NIPPV compared to baseline. These findings were consistent with Brochard et al. and Agarwal et al research.

In this study, metrics on arterial blood gas analysis, such as pH and PaO2, improved with NIPPV at the end of 4, 12, and 24 hours compared to baseline in patients with type I respiratory failure. NIPPV improved pH, PaO2, and PaCO2 in type II respiratory failure patients after 4, 12, and 24 hours when compared to baseline. Ventrella et al 99 backed up these findings. In addition, Mclaughlin et al. found that in individuals with hypercapnic respiratory failure, ABG values such as pH and PaCO2 improved after 1 and 4 hours when compared to baseline.[24]

In addition, the difference in the mean length of stay in hospital between type I (9.17.79 days) and type II (8.53.69 days) respiratory failure patients was not significant. In a research published by Agarwal et al., the length of hospital stay in acute hypoxemic respiratory failure was (12 4.7) days.[25]

Rapid gas reversal, fewer problems, and a shorter weaning time all likely contribute to a shorter hospital stay. The cost-effectiveness of NIPPV is also due to the lower hospitalisation time.

There have been few previous studies that compare type I and type II failure as a whole. Many studies have compared individual disease groups (e.g., acute pulmonary edoema and acute COPD exacerbation), studied a single disease as a whole (e.g., NIV in COPD or NIV in ALI/ARDS), or compared modes of NIV among various types of acute respiratory failure (e.g., NIV in COPD or NIV in ALI/ARDS) (e.g., CPAP vs BIPAP).

Outcome predictors are critical for identifying patients who are less likely to improve with NIV, necessitating careful monitoring and the use of easily available intubation techniques.

In the NIV success group, there was a substantial decrease in dyspnea score, R, and HR at the end of 4 hours compared to baseline, but not in the NIV failure group. When compared to the NIV failure group, the improvement in pH and PaO2 in the NIV success group was statistically significant.

These findings are consistent with those of Bhattacharyya et al,[22] who found that the success group's heart and RRs, pH, and PaCO2 improved within the first hour, and that these parameters improved even after 4 hours and 24 hours after NIPPV treatment. RR improved by the end of 30 minutes with NIPPV, according to Lin et al 26, and can be used as a predictor of NIV success in patients with acute respiratory failure.

Limitations of the study

1. For future studies, larger study populations are needed.
2. As it was conducted in a tertiary care hospital set up, this study cannot be generalised.

CONCLUSION

Improvement in dyspnea score, RR, HR, and arterial blood gas indicators such as pH and PaO2 within 4 hours of NIPPV might be utilised to predict the response to NIPPV, according to the current study. As a result, patients on NIPPV with respiratory failure should be monitored for changes in HR, RR, pH, and PaO2 at regular intervals, so that patients who require invasive ventilation should be intubated as soon as possible to avoid an unnecessary increase in morbidity and death.

REFERENCES

1. Singh D, Agusti A, Anzueto A, Barnes PJ, Bourbeau J, Celli BR, Criner GJ, FrithP, HalpinDMG, Han M, López Varela MV, Martinez F, Montes de Oca M, PapiA, Pavord ID, Roche

N, Sin DD, Stockley R, Vestbo J, Wedzicha JA,Vogelmeier C. Global Strategy for the Diagnosis, Management, and Preventionof Chronic Obstructive Lung Disease: the GOLD science committee report2019. Eur. Respir. J. 2019 May;53(5).
2. Neder JA, Berton DC, O'Donnell DE. Pitfalls in the interpretation of pulmonaryfunction tests in neuromuscular disease. Jornal Brasileiro de Pneumologia.2020 Aug 26;46.
3. Chaouat A, Naeije R, Weitzenblum E. Pulmonary hypertension in COPD.European Respiratory Journal. 2008 Nov 1;32(5):1371-85.
4. Non-invasive ventilation in acute respiratory failure. Thorax. 2002 Mar1;57(3):192.
5. Wysocki M, Tric L, Wolff MA, Millet H, Herman B. Noninvasive pressure supportventilation in patients with acute respiratory failure. A randomized comparisonwith conventional therapy. Chest 1995;107:761-8.
6. Vitacca M, Rubini F, Foglio K, Scalvini S, Nava S, Ambrosino N. Non-invasive modalities of positive pressure ventilation improve the outcome of acuteexacerbations in COLD patients. Intensive Care Med 1993;19:450-5.
7. Elliott MW, Steven MH, Phillips GD, Branthwaite MA. Non-invasive mechanicalventilation for acute respiratory failure. BMJ 1990;300:358-60.
8. Bott J, Carroll MP, Conway JH, Keilty SE, Ward EM, Brown AM, et al.Randomised controlled trial of nasal ventilation in acute ventilatory failure due tochronic obstructive airways disease. Lancet 1993;341:1555-7.
9. Mehta S, Hill NS. Noninvasive Ventilation. Am J Respir Crit Care Med. 2001Feb 1;163(2):540–77.
10. Girou E, Brun-Buisson C, Taillé S, Lemaire F, Brochard L. Secular trends innosocomial infections and mortality associated with noninvasive ventilation inpatients with exacerbation of COPD and pulmonary edema. JAMA.2003;290:2985–2991.
11. Cabrini L, Landoni G, Oriani A, Plumari VP, Nobile L, Greco M, et al.Noninvasive ventilation and survival in acute care settings: a comprehensivesystematic review and metaanalysis of randomized controlled trials. Crit CareMed. 2015;43(4):880–888.
12. Grippi MA. Respiratory failure: an overview. Fishman's Pulmonary Diseasesand Disorders, 2017;2:2152–2161.
13. Dave L, Dwivedi P, Srivastava N, Yadav BS, Dohre R. A study of cardiovascularmanifestations of COPD. Int J Res Health Sci. 2014 Jul 31;2(3):812-7.
14. Rachaiah NM, Rachaiah JM, Krishnaswamy RB. A correlative study ofspirometric parameters and ECG changes in patients with chronic obstructivepulmonary disease. Int J Biol Med Res. 2012;3(1):1322-6.
15. Begum K, Begum MK, Sarker ZH, Dewan MR, Siddique MJ. Lipid profile statusof chronic obstructive pulmonary disease in hospitalized patients. BangladeshJournal of Medical Biochemistry. 2010;3(2):42-5.
16. Jain B, Pasari N, Songra A, Bajpai A. The Lipid Profile Parameter in ChronicObstructive Pulmonary Disease Patients and Correlation with Severity ofDisease. Journal of Cardio-Thoracic Medicine. 2017 Jun 1;5(2):564-8.
17. Vikhe VB, Shende PS, Patil RS, Tamakuwala KK, Patil AS, Gupta AP.Cardiovascular complications in chronic obstructive pulmonary disease withreference to 2D echocardiography findings. National journal of medicalresearch. 2013;3(4):385-8.
18. Singhal S, Kaur G, Sisodia V. A correlational study ofelectrocardiogram findings with severity of copd in atertiary care centre. Journal of evolution of medical anddental sciences-jemds. 2018 Jun 25;7(26):3000-.
19. Dr Thenmozhi. A Correlative Study of Spirometric Parameters, ECG Changes,Lipid Profile and Diabetes in Chronic Obstructive Pulmonary Disease. J Med SciClin Res [Internet]. 2019 Mar 28 [cited 2021 Dec 30];7(3). Available from:http://jmscr.igmpublication.org/v7-i3/211%20jmscr.pdf
20. Arsude S, Sontakke A, Jire A. Outcome of Noninvasive Ventilation in AcuteRespiratory Failure. Indian journal of critical care medicine: peer-reviewed,official publication of Indian Society of Critical Care Medicine. 2019Dec;23(12):556.
21. Ventrella F, Giancola A, Cappello S, Pipino M, Minafra G, Carbone M, et al. Useand performance of non-invasive ventilation in Internal Medicine Ward: a real-life study. Ital J Med. 2015;9(3):260–267.
22. Bhattacharyya D, Prasad B, Tampi PS Retd, Ramprasad R. Early predictorsof success of non-invasive positive pressure ventilation in hypercapnicrespiratory failure. Med J Armed Forces India 2011;67:315-9.
23. Kramer N, Meyer T, Meharg J, Cece R, Hill N. Randomized, prospective trial ofnoninvasive positive pressure ventilation in acute respiratory failure. Am JRespir Crit Care Med. 1995;151(6):1799–1806. doi:10.1164/ajrccm.151.6.7767523.
24. McLaughlin K, Murray I, Thain G, Currie G. Ward-based non-invasiveventilation for hypercapnic exacerbations of COPD: a "real-life" perspective.QJM. 2010;103(7):505–510. doi: 10.1093/qjmed/hcq063.
25. Agarwal R, Handa A, Aggarwal AN, Gupta D, Behera D. Outcomes ofnoninvasive ventilation in acute hypoxemic respiratory failure in a respiratoryintensive care unit in north India. Respir Care. 2009;54(12):1679–1687.
26. Lin M, Guo H, Huang M, Chen C, Wu C. Predictors of successful noninvasiveventilation treatment for patients suffering acute respiratory failure. J Chin MedAssoc. 2008;71(8):392–398. doi: 10.1016/S1726-4901(08)70089-3.