

Original Research Article

Non-invasive ventilation in acute exacerbation of COPD with mild to moderate type 2 respiratory failure

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ABSTRACT

Background: Chronic obstructive pulmonary disease is a syndrome of progressive airflow limitation caused by abnormal inflammatory reaction of airway and lung parenchyma. Risk factor for development of COPD is a complex interaction of genetic factors and many environmental exposures, with the cigarette smoking being the most common etiological agent.

Materials and Methods: It is a prospective observational comparative study conducted among patient with mild to moderate type 2 respiratory failure, secondary to acute exacerbation of Chronic Obstructive Pulmonary Disease admitted in chest ward department of Respiratory medicine, Late Shri Lakhi Ram Agrawal Memorial Medical College, Raigarh, Chhattisgarh, India for a period of one year from April 2020- April 2021.

Results: A total 60 patients were studied. Out of which, 30 patients in study group for whom noninvasive ventilation support along with conventional treatment was given and remaining 30 patients in comparison group, same treatment was given without non-invasive ventilation support. Both groups had similar demographic, clinical, biochemical profile at the time of admission. Distribution of comorbidities, smoking history were similar as shown below in tables. After application of Non-invasive ventilation along with conventional treatment in study group, the result showed that mean hours of NIV use in study group was 27 hours and mean hours of Oxygen use in comparison group was 98 hours.

Conclusions: Use of non-invasive ventilation in acute exacerbation of COPD, with mild to moderate type 2 respiratory failure, reduced tachypnoea, tachycardia, after 4 hours. There were improvement in oxygen saturation after 4 hours, improvement in PH also occurred after 4 hours by 0.04. Non-invasive ventilation gives rest to fatigued inspiratory muscle so work of breathing is reduced. It also restores functional and biochemical changes associated with fatigued muscle so all complication were reduced with use of non-invasive ventilation.

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1. Introduction

Chronic obstructive pulmonary disease is a syndrome of progressive airflow limitation caused by abnormal inflammatory reaction of airway and lung parenchyma. Risk factor for development of COPD are a complex interaction of genetic factors and many environmental exposure, with the cigarette smoking being the most common etiological agent.¹ The comorbid condition, is associated with pulmonary manifestation, pose additional problem in the management of the disease.²

The definition endorsed by Global Initiative for chronic Obstructive lung disease (GOLD), in its latest edition is "Chronic obstructive pulmonary disease is a common, preventable lung disorder characterised by progressive, poorly reversible airflow limitation often with systemic

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manifestations, in response to tobacco smoke and/or other harmful inhalational exposure."³

The prevalence of COPD in the adult population range between 4% and 10% and World Health Organisation projection predict that COPD related mortality and disability will continue to increase worldwide during the next two decades.⁴ It remain a major public health problem and is projected to be rank 5th in 2020 burden of disease worldwide. In India, it estimated that there are around 1.49 crore chronic case of COPD in the age group of 30 years and above.⁵ The prevalence rate of COPD in male varied from 2.12% to 9.4% in studies conducted in north India and from 1.4% to 4.08% in south India. These were projected to increase by nearly 50% by the year2016.⁶

1.1. Acute exacerbation of Chronic Obstructive Pulmonary disease

Exacerbations are episodes of acute worsening of clinical condition in patient with COPD. An episode of acute exacerbation of COPD (AECOPD) was defined at the Aspen workshop as "a sustained worsening of the patient condition from the stable state and beyond normal day-to -day variations, that is acute in onset and necessitates a change in regular medication in a patient with underlying COPD."⁷

It occurs despite adequate control. Most patient experience episodes of exacerbation at least one or more time during their life time. Many suffer one to three time a year. COPD exacerbation have a huge impact on the overall health condition of the patient both physically and emotionally. Present day successful management of COPD exacerbation can be done in majority of patients. COPD exacerbation is an event in the natural course of COPD characterised by an acute change in the patient baseline dyspnoea or breathing difficulty, sputum production sufficient to warrant change in management. Causes of COPD exacerbation are infections like viral and bacterial, environmental conditions like sudden change in temperature humidity air pollution, exposure to tobacco smoke and noxious gases, irritating chemicals, host factor such as patient with poor general health condition, poor nutritional status, immune compromised status, lack of compliance with prescribed medical therapy, lack of continuing with advised exercise, lack of compliance with long term oxygen therapy.⁸

Acute exacerbation of COPD can cause respiratory failure, that is, "inability of respiratory system to maintain metabolic demand of body, elimination of CO_2 and oxygenation of blood." It requires hospitalisation and mechanical ventilation, which has its own limitation because of complication like infection and also it becomes difficult to wean the patient. These causes increase the rate of morbidity and mortality. Non-invasive ventilation is a recently developed tool in managing respiratory failure, especially in mild and moderate cases, due to acute exacerbation of COPD and has shown good outcome in terms of reduction in mortality, reduction in need for intubation and mechanical ventilation, decrease duration of stay in the hospital.⁹

This clinical study aims to look into these aspects and see the efficacy of non-invasive ventilation. As in our area Chhattisgarh no such study has been carried out in past, we decided to conduct prospective comparative observational study from April 2020 to April 2021, to study efficacy of using non-invasive ventilation in mild to moderate type 2 respiratory failure with acute exacerbation of COPD.

2. Materials and Methods

It is a prospective observational comparative study conducted among patient with respiratory failure secondary to acute exacerbation of Chronic Obstructive Pulmonary Disease admitted in the chest ward of the Department of Respiratory Medicine, Late Shri Lakhi Ram Agrawal Memorial Medical College, Raigarh, Chhattisgarh, India for a period of one year from April 2020- April 2021.

2.1. Sampling technique

By simple random sampling method, patients who were admitted in chest ward and diagnosed as acute exacerbation of Chronic Obstructive Pulmonary Disease, of them 30 patents, who fulfilled our selection criteria were randomly selected in study group and another 30 patients in comparison group, after taking informed consent.

2.2. Inclusion criteria

- 1. Clinical diagnosis of Chronic Obstructive Pulmonary Disease
- 2. Respiratory rate >25/min
- 3. SPO2<80%
- 4. Respiratory acidosis PH<7 35
- 5. Hypercapnia PaCO2>45mmHg

2.3. Exclusion criteria

- 1. Impending or post respiratory arrest
- 2. Severe respiratory failure PH< 7 25, SPO2 <70%
- 3. Impaired consciousness (Glasgow coma scale <8
- 4. Cardiovascular instability
- 5. Copious secretions
- 6. Sever uncorrected hypoxia
- 7. Craniofacial trauma
- 8. Pneumothorax or pneumomediastinum
- 9. Non tolerance of Non-invasive ventilation
- 10. Patient did not willing to give consent

2.4. Methodology

- 1. Patients who fulfilled inclusion and exclusion criteria underwent following assessments:
- 2. Clinical profile assessment.
- 3. Detailed clinical history was taken, onset of symptoms like cough with expectoration, dyspnoea, fever, chest pain, and oedema feet.
- 4. Past history of tuberculosis and treatment history were taken.
- 5. History of addiction and comorbid conditions were taken.
- Complete physical examination was done. Vital parameter was noted. Dyspnoea was graded according to modified Medical Research Council (mMRC) dyspnoea scale.¹⁴⁰
- 7. Relevant investigations were done, these are as follows:
 - (a) Complete blood count.
 - (b) Renal and liver function test.
 - (c) Serum electrolytes.
 - (d) Random blood sugar.
 - (e) Sputum, smear for Acid Fast Bacillus (AFB) and culture.
 - (f) Chest X-ray.
 - (g) ECG.
 - (h) Pulse oximetry Monitoring.
 - (i) Arterial blood gas analysis.

2.5. Treatment intervention

Study group was put on Non-invasive CPAP (continuous positive airway pressure) ventilation. We used ResMed S-9 autoset CPAP machine for giving non-invasive ventilation. ResMed's autoset technology with expiratory pressure relief and monitors breathing and adapts breath by breath, to deliver the lowest pressure possible for comfortable breathing. Injectable antibiotics, oral corticosteroid, nebulisation with LABA, LAMA, ICS, SABA were given as per requirement. In comparison group same treatment was followed with oxygen inhalation, except NIV.

2.6. Statistical method

Data was recoded in structured format and entered in Microsoft Excel sheet. The statistical analysis had been performed using SPSS software version 22. The statistical analysis of quantitative data between two groups was done by using student 't' test (Unpaired 't' test). The qualitative data was analysed using chi- square test/Fisher-exact test. P-value <0.05 is considered to be statistically significant.

3. Results

A total 60 patients were studied. Out of which, 30 patients in study group where non-invasive ventilation support was given along with conventional treatment and remaining 30 patients in comparison group, were given same treatment without non-invasive ventilation support. Both groups had similar demographic, clinical, biochemical profile at the time of admission. Distribution of comorbidities, smoking history were similar as shown below in tables.

In Table 1, there is no statistically significant difference in age in both the groups.

There is equal and main presenting complain in both study and comparison group. Table 2

In Table 4, in study group tachypnoea improved after 4hours with application of NIV along with conventional treatment, while after 24 hours in comparison group without NIV.

The above table showed that the SPO₂ was improved by 10.46% in study group after giving NIV support for 4 hours. But SPO₂was improved by 8.53% in comparison group without NIV support. SPO₂ was found above 90% in both the groups after 24 hours. Table 5

The above table showed that mean hours of NIV use in study group was 27 hours and mean hours of Oxygen use in comparison group was 98 hours.Table 6

The above table showed that need of intubation and its complications was reduced in study group with the use of NIV, it was 3.33% whereas in comparison group it was 20%. Use of NIV reduces mortality in study group.Table 7

4. Discussion

Non-invasive ventilation is ventilator support without endotracheal intubation. In recent decades NIV is being increasingly used for hypercapnic respiratory failure due to acute exacerbation of COPD. We have done study on efficacy of NIV in acute exacerbation of COPD, with mild to moderate type 2 respiratory failure. We had recruited 60 patients who had fulfilled selection criteria after ethical approval and their consent. In study group of 30 patient's non-invasive ventilation support with required treatment was given and in comparison group, for remaining 30 patients same treatment without NIV support given.

In this study, both study group and comparison group had acute exacerbation of COPD. The mean age was 71.47years in study group and 71.6years in comparison group (P-value 0.93). Gender distribution wise, 56.67% (N₁=17) were male, 43.33% (N₁=13) were female in study group and 66.67% (N₂=20) were male, 33.33% (N₂=10) were female in comparison group. Male to female ratio was 2:1 in both groups (P value0.29).

All patients were presented with MMRC scale grade IV dyspnoea, with variable symptoms of fever, cough with or without expectoration, chest pain, pedal oedema.¹⁰

Parameters	Study group (N=30)	Comparison group (N=30)	p-value
Age (years)	71.47 ± 5.54	71.60 ± 4.65	0.92
Sex			
Male	17 (56.7%)	20 (66.7%)	0.29
Female	13 (43.3%)	10 (33.3%)	
Occupation			
Ex-employee	18 (60%)	20 (66.7%)	0.79
Housewife	12 (40%)	10 (33.3%)	
Addiction			
No addiction	14 (46.7%)	10 (33.3%)	0.07
Tobacco chewing	2 (6.7%)	0 (0%)	
Smokers	1 (3.3%)	7 (23.3%)	
Ex-smokers	13 (43.3%)	13 (43.3%)	
Fever			
Present	14 (46.7%)	17 (56.7%)	0.30
Absent	16 (53.3%)	13 (43.3%)	
Duration of fever	1.10 ± 1.27	1.30 ± 1.26	0.53
Cough			
Purulent	16 (53.3%)	16 (53.3%)	1.00
Mucoid	14 (46.7%)	14 (46.7%)	
Chest pain			
Present	1 (3.3%)	2 (6.7%)	0.83
Absent	16 (53.3%)	15 (50%)	
Post tussive	13 (43.3%)	13 (43.3%)	
Temperature			
Normal	14 (46.67%)	17 (56.6%)	0.61
Raised	16 (53.3%)	13 (43.3%)	
JVP			
Normal	9 (30%)	6(20%)	0.55
Raised	21 (70%)	24 (80%)	
Cyanosis			
Absent	8 (26.7%)	7 (23.3%)	1.00
Present	22 (73.3%)	23 (76.7%)	
Oedema			
Absent	12 (40%)	19 (63.3%)	0.14
Present	18 (60%)	11 (36.7%)	
Systolic BP	128.00 ± 13.75 mmhg	129.67 ± 11.29 mmhg	0.61
Diastolic BP	83.33 ± 8.44 mmhg	86.67 ± 6.06 mmhg	0.08
Gram stain			
Commensal	12 (40%)	13 (43.3%)	0.41
Gram positive cocci	10 (33.33%)	13 (43.3%)	
No organism	8 (26.66%)	4 (13.3%)	

Table 1: Comparative analysis of demographic and clinical profile between study and comparison group

Table 2: Breathlessness MRC scale¹⁴⁰ grad iv

Breathlessness	Present	Absent	P value
Study Group	30(100%)	70	
Comparison Group	30(100%)	70	

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Co-morbidities	Study group (N=30)	Comparison group (N=30)	p-value
Absent	10 (33.3%)	6 (20%)	
CCF	1 (3.3%)	0 (0%)	
Hypertension	2 (6.7%)	3 (10%)	
Diabetes mellitus	0 (0%)	1 (3.3%)	
Cardiac disease	1 (3.3%)	0 (0%)	
Cor pulmonale	1 (3.3%)	0 (0%)	
Hypothyroidism	0 (0%)	2 (6.7%)	
Tuberculosis	3 (10%)	4 (13.3%)	
Diabetes mellitus and psychiatric diseases	0 (0%)	1 (3.3%)	
Hypertension and Diabetes mellitus	4 (13.3%)	5 (16.7%)	
Tuberculosis and Diabetes mellitus	2 (6.7%)	0 (0%)	0.38
Tuberculosis and Hypertension	1 (3.3%)	0 (0%)	
Hypertension, cardiac disease and hypothyroidism	1 (3.3%)	0 (0%)	
Hypertension, Diabetes mellitus and CCE	0 (0%)	1 (3.3%)	
Hypertension, Diabetes	0 (0%)	1 (3.3%)	
Hypertension, Diabetes mellitus and diabetic	1 (3.3%)	0 (0%)	
nephropatny Hypertension, Diabetes mellitus and osteoporosis	0 (0%)	1 (3.3%)	
Hypertension, Diabetes mellitus and osteoarthritis	1 (3.3%)	0 (0%)	
Tuberculosis, Hypertension and Old CVA	1 (3.3%)	0 (0%)	
Tuberculosis, Hypertension and Diabetes mellitus	0 (0%)	2 (6.7%)	
Hypertension, Diabetes mellitus, cardiac disease and diabetic nephropathy	0 (0%)	1 (3.3%)	
Hypertension, Diabetes mellitus, cardiac disease and osteoporosis	0 (0%)	1 (3.3%)	
Tuberculosis, Hypertension, Diabetes mellitus and cor pulmonale	0 (0%)	1 (3.3%)	
Tuberculosis, Hypertension, Diabetes mellitus, cardiac disease and osteoporosis	1 (3.3%)	0 (0%)	

 Table 4: Respiratory rate/min at different time intervals between study and control groups

Respiratory rate	Study group	Comparison group	P-value
Respiratory rate at 0 hour	$30.53 \pm 2.01/\text{min}$	30.17 ± 2.20/min	0.51
Respiratory rate after 4 hours	21.77 ± 3.24/min	25.77 ± 2.34/min	0.0001*
Respiratory rate after 12 hours	20.53 ± 2.09 /min	22.40 ± 2.47/min	0.002*
Respiratory rate after 24 hours	$18.27 \pm 2.12/min$	$20.03 \pm 3.99/\text{min}$	0.01*

Table 5: Improvement in SPC	D_2			
Improvement/ Change in SPO ₂ %	Study Group (Mean ± SD)	Control group Mean ± SD	P value	
after 4hr	(7.73±4.5933)%	(5.167±4.23)%	0.0001 HS	
after 12hr	(10.46±5.8294)%	(8±5.311)%	0.0001 HS	
after 24hr	(12.33±6.0648)%	(10.867±5.94)%	0.0001 HS	
2nd day	(14.63±6.4353)%	(12.9±6.210)%	0.0001 HS	
3rd day	(16.46±7.3613)%	(14.767±6.50)%	0.0001 HS	
Hours of NIV/O2	-	Mean ± SD	P value	
Hours of NIV/O2		Mean ± SD	P value	
Study Group		27.07 ± 34.75	0.00001 HS	
Comparison Group		98.2±16.24	0.00001115	
Table 7: Comparison of outco	ome between study and comparison groups	oup		
	Study group	Comparison group		
INT and MV	1(3.33%)	6(20%)	0.04 SIG	

	Study group	Comparison group	
INT and MV	1(3.33%)	6(20%)	0.04 SIG
Total complication	1(3.33%)	6(20%)	
Laryngeal oedema	0	2	
VAP	1	2	
HCAP	0	2	

Smoking history was present (ex- smoker) in 43.33% (N₁=13) in both study and (N₂ =13) comparison group, tobacco chewing was present among6.7% (N₁=2) in study and 0% (N₂=0) in comparison group. Among study group, current smokers were 3.3% (N₁=1) and in comparison group they were 23.3% (N₂=7) (P-value0.07). Among females, 3.33% (N₁=1) were smoker in both study and (N₂=1) comparison group, non-smokers were 46.67% (N₁=14) in study group and 33.33% (N₂=10) in comparison group. Male non- smokers in study group were 3.33% (N₁=1) and in comparison group were 6.66% (N₂=2).

Fever was present in 46.67% (N₁=14) in study group and 56.67% (N₂=17) in comparison group, Duration of fever was 1.10 ± 1.27 days in study group and 1.30 ± 1.26 days in comparison group (P-value 0.53). Fever was absent in53.33% (N₁=16) in study group and 43.33% (N₂=13) in comparison group.(P-value 0.3).

Cough with mucoid sputum was 46.67% (N_1 =14) in study group and 46.67(N_2 =14) in comparison group. Purulent sputum was present among 53.33% (N_1 =16) in study group and 53.33% (N_2 =16) in comparison group (P-value 1).

Chest pain was present in 3.33% (N_1 =1) in study group and 6.67% (N_2 =2) in comparison group, post-tussive chest pain present 43.33% (N_1 =13) in study group and 43.33% (N_2 =13) in comparison group, absent 53.33% (N_1 =16) in study group and 50% (N_2 =15) in comparison group (Pvalue0.83).

All patients presented with tachypnoea, at the time of admission, in study group respiratory rate was 30.53 ± 2.01 /min and 30.17 ± 2.20 /min in comparison group, after 4 hour respiratory rate was 21.77 ± 3.24 /min

in study group and $25.77\pm2.34/min$ in comparison group (P value 0.0001), after 12 hour respiratory rate was $20.53\pm2.09/min$ in study group and $22.40\pm2.47/min$ in comparison group (P value 0.002), after 24 hour respiratory rate was $18.27\pm2.12/min$ in study group and $20.03\pm3.99/min$ in comparison group (P value 0.01).

All patient presented with tachycardia, at the time of admission in study group pulse rate was115.70 \pm 7.2/min and 117.37 \pm 13.42/min in comparison group, after 4-hour pulse rate was 99.93 \pm 7.33/min in study group and 114.37 \pm 13.62/min in comparison group (P value 0.0001), after 12 hours pulse rate was92.00 \pm 7.46/min in study group and 97.10 \pm 4.04/min in comparison group (P value 0.002) after 24 hours pulse rate was 91.43 \pm 6.72/min in study group and 94.80 \pm 3.84/min in comparison group (P value 0.02).

Blood Pressure, systolic BP was 128.00 ± 13.75 mmhg in study group and 129.67 ± 11.29 mmhg in comparison group (P-value 0.6). Diastolic BP was 83.33 ± 8.44 mmhg in study group and 86.67 ± 6.06 mmhg in comparison group (P-value 0.08).

Jugular venous pressure was normal among 30% ($N_{1=9}$) in study group and 20% ($N_{2}=6$) in comparison group, jugular venous pressure was raised among 70% ($N_{1=21}$) in study group and 80% ($N_{2}=24$) in comparison group (P-value0.55).

Temperature was raised among 46.67% (N_1 =14) in study group and 56.67% (N_2 =17) in comparison group, temperature was normal among 53.33% (N_1 =16) in study group and 43.33% (N_2 =13) in comparison group (P-value0.61).

Cyanosis was present among 73.33% (N₁=22) in study group and 76.66% (N₂=23) in comparison group, (p value-

1).

Bilateral pedal oedema was present among 60% (N₁=18) in study group and 50% (N₂=15) in comparison group (P-value0.14).

4.1. Examination of respiratory system

Examination of respiratory system bilateral rhonchi were present in all patient. Bilateral basal crepitation was present among 66.66% (N₁=20) in study group and 73.33% (N₂=22) in comparison group.

So in both groups, presenting signs and symptoms were almost same and there was significant improvement in tachypnoea tachycardia in study group after application of NIV within 4 hours.

4.2. Hours of NIV and oxygen use

In study group mean hours of NIV use was 27.07 ± 34.75 hrs. In comparison group mean hours of oxygen use was 98.2 ± 16.24 hrs. The p value was 0.00001.

4.3. Complications

In study group one patient underwent mechanical ventilation (MV) and developed ventilator associated pneumonia (VAP). In comparison group 6 patients underwent MV, among them 2 developed VAP, 2 developed laryngeal oedema and 2 developed health care associated pneumonia HCAP. The P value was 0.04 which was significant.

4.4. Number of hospitalisations in a year

In study group, mean hospitalisation was 1.27 ± 0.455 times in a year, and in comparison group, it was 3.067 ± 0.86 . The P value between two groups was 0.00001.

In our study, 3.33% required intubation among study (NIPPV) group and 16.6% among comparison group. Lesser number of intubation was required in study group. Complication was 3.33% in study group and 20% in comparison group. Complication was reduced in study group due to lesser number of intubation. Hospital stay was 5.2 days in study group and 7.9 days in comparison group. Hospital stay was reduced in study group and early improvement in respiratory acidosis was seen. Mortality was 0% in study group and 6.67% in comparison group. Mortality was decreased in NIPPV group. So, in our study we found: mortality, complication, hospital stay was decreased in NIPPV group.

In our study, there was no death in NIPPV group, similar to their study. Also, only one patient needed intubation among study group and 6 needed intubation among comparison group, same like their study result. In our study, length of hospital stay was 5.2 days in study group and 7.9 days in comparison group, whereas in their study it

was 7.2 days and 9.6 days respectively.

In both the studies 100% patients of NIPPV group were discharged, whereas 76% patients of comparison group in their study and 93% patients of comparison group, were discharged.

Rodriguez-Roisin et al., have reviewed a similar article, which showed similar results. In our study we found same results regarding mortality, need for intubation, duration of hospital stays and complication was reduced in NIV group, pH was increased within four hours, PaCO₂ was decreased within four hours, because we conducted our study in respiratory ward and performed arterial blood gas analysis after 4 hours. There was hardly any change in pH and PCO₂ in comparison group after four hours.¹¹

Sinuff T have reviewed 8 studies, and they found mortality and intubation was decreased in NIV group.¹² Respiratory rate was reduced within four hour in NIV group, while no change in comparison group, pH increased, PaCO₂ decreased within 4 hours, in NIV group. Whereas hardly any change was observed within 4 hours in comparison group. Hospital stays reduced by 3 days in NIV group. Complication reduced 17% in NIV group.

Ambrosino N has concluded hat NIV should be considered first-line therapy in the management of acute respiratory failure caused by chronic obstructive pulmonary disease (COPD) exacerbations based on evidence derived from multiple randomized trials.¹³ Meta-analyses by Ram FS concluded that intubation rate, hospital stay and mortality were reduced in non-invasive ventilation group. Although hypercapnic respiratory failure with coma is a contraindication for NIV, non-comatose patient with hypercapnic respiratory failure can be treated with NIV successfully.¹⁴ Outcomes of severe COPD exacerbations are no worse if treated with NIV than with endotracheal intubation, indicating that an initial trial with NIV is not deleterious, even in severely ill COPD patients.

Plant et al did a prospective multicentre randomised control trial, they took 236 patients, out of them 118 patients were allocated to standard treatment group and 118 patients to NIV.¹⁵ Monitoring was done by measuring arterial blood gas analysis after 1 and 4 hours of NIV. Respiratory rate was also monitored throughout the study. They found pH increased after 4 hours, respiratory rate decreased after 4 hours in NIV group, while hardly any change in comparison group. Death was 0(0%) in NIV group and 2(6.67%) in comparison group. Intubation was 1(3.33%) in NIV group and intubation was 6(20%) in comparison group. Hospital stay was 5.2 days in NIV group while 7.9 days in comparison group.

Thirty-four patients (94%) were successfully started with NIV and tolerated. Two patients (6%) failed to start on NIV due to inability to tolerate the mask. Of the 34 patients who were started on NIV, it was successful in 27 patients (79%) as defined by improvement in acidosis, hypercapnia and

respiratory rate. No patient developed any complications from NIV. Seven (21%) of those on NIV, failed to meet the above criteria of improvement.

In our study, all patients (n=30) tolerated NIV and out of them one patient did not show improvement and underwent mechanical ventilation. Rest all others (n=29) showed improvements in terms of improved acidosis, Hypercapnia and respiratory rate.

In their study, all those who failed to tolerate NIV (2 patients) or failed to improve once established on NIV, died (n=7). Mortality was therefore 25%. But in our study, death did not occur in NIV group. As we have excluded severe acidosis (pH<7.25).

Mean length of stay in hospital of survivors was 9.5 days. But in our study mean length of stay in hospital was 5.2 days. Their study demonstrated, NIV can be successfully used as an alternative to endotracheal intubation and mechanical ventilation in an intensive care unit in selected elderly patients with acute hypercaphic respiratory failure due to acute exacerbation of COPD. Its tolerability, success rate and associated low mortality are comparable to its use in younger patients.

Similarly, our study also showed, NIV can be used successfully in patients with respiratory failure due to AE of COPD, in general respiratory ward, where endotracheal intubation and mechanical ventilation are not readily available. Our study also showed NIV is better than simple oxygen delivery devices, while managing respiratory failure.

They concluded that, NIV should therefore be the treatment of choice for hypercapnic respiratory failure due to acute exacerbation of COPD in selected elderly patients. In addition, it may be used successfully in those patients in whom endotracheal intubation and ventilation on intensive care unit is not appropriate.

Thys et al, did a study on 190 patients with acute respiratory failure mainly due to COPD and Pulmonary oedema who received 200 trials of Non-invasive ventilation in an emergency department.¹⁶ Their results showed that the procedure was successful in 60.5% of patients and mortality was 34.5%. Patient who required intubation was 6.5% and tracheostomy rate was 1%. The improvement of pH within 6 hours after non-invasive ventilation was predictive of survival in the hypercapnic group. They concluded that their results confirmed the global efficacy of Non-invasive ventilation in an emergency department setting.

In our study we found that mortality was 0(0%) and Intubation was 3.33% in NIV group. Mortality was 2(6.67%) and intubation was 6(20%) in comparison group. pH was improved within 4 hours after non-invasive ventilation. So, efficacy of NIV was also proved in our study.

5. Conclusions

Use of non-invasive ventilation in acute exacerbation of COPD reduced tachypnoea, tachycardia, after 4hour, if patients can be selected properly with mild to moderate hypercapnic respiratory failure. Improvement in oxygen saturation occurred after 4 hours improvement in PH occurred after 4 hours by 0.04. Significant clinical improvement occurred after 4 hours. Acidosis and hypercapnia were corrected within 24 hours. Non-invasive ventilation gives rest to fatigued inspiratory muscle so work of breathing is reduced. It also restores functional and biochemical changes associated with muscle fatigued so all complication were reduced with use of non- invasive ventilation. Patient selection is important, NIV should be given early, before developing severe respiratory failure, to get maximum benefit in acute exacerbation of COPD.

6. Acknowledgments

None.

7. Conflict of Interest

The authors declare that there are no conflicts of interest in this paper

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None.

References

- Andrews JC, Schunemann HJ, Oxman AD, Pottie K, Meerpohl JJ, Coello PA, et al. GRADE guidelines: 15. Going from evidence to recommendation - determinants of a recommendation's direction and strength. J Clin Epidemiol. 2013;66(7):726–35. doi:10.1016/j.jclinepi.2013.02.003.
- Plant PK, Owen J, Elliott MW. One year period prevalence study of respiratory acidosis in acute exacerbation of COPD; implications for the provision of non-invasive ventilation and oxygen administration. *Thorax*. 2000;55(7):550–4. doi:10.1136/thorax.55.7.550.
- Roberts CM, Stone RA, Buckingham RJ. Acidosis, non-invasive ventilation and mortality in hospitalised COPD exacerbations. *Thorax*. 2011;66(1):43–8. doi:10.1136/thx.2010.153114.
- Nava S, Navalesi P, Conti G. Time of non-invasive ventilation. Intensive Care Med. 2006;32(3):361–70. doi:10.1007/s00134-005-0050-0.
- Keenan SP, Powers CE, Mccormack DG. Noninvasive positivepressure ventilation in patients with milder chronic obstructive pulmonary disease exacerbations: a randomized controlled trial. *Respir Care*. 2005;50(5):610–6.
- 6. Bardi G, Pierotello R, Desideri M. Nasal ventilation in COPD exacerbations: early and late results of a prospective, controlled study. *Eur Respir J.* 2000;15:98–104.
- O'driscoll BR, Howard LS, Davison AG. BTS guideline for emergency oxygen use in adult patients. *Thorax*. 2008;63(6):1–68. doi:10.1136/thx.2008.102947.
- Austin MA, Wills KE, Blizzard L. Effect of high flow oxygen on mortality in chronic obstructive pulmonary disease patients in prehospital setting: randomised controlled trial. *BMJ*. 2010;341:5462. doi:10.1136/bmj.c5462.
- Carrera M, Marin JM, Anton A, Chiner E, Alonso ML, Masa JF, et al. A controlled trial of noninvasive ventilation for chronic obstructive pulmonary disease exacerbations. *J Crit Care*. 2009;24(3):473.e7–14. doi:10.1016/j.jcrc.2008.08.007.

- Dikensoy O, Ikidag B, Filiz A. Comparison of non-invasive ventilation and standard medical therapy in acute hypercapnic respiratory failure: a randomised controlled study at a tertiary health centre in SE Turkey. *Int J Clin Pract.* 2002;56(2):85–8.
- Jurjevic M, Matic I, Sakic-Zdravcevic K, Sakić S, Danić D, Buković D, et al. Mechanical ventilation in chronic obstructive pulmonary disease patients, noninvasive vs. invasive method (randomized prospective study). *Coll Antropol.* 2009;33(3):791–7.
- Sinuff T, Cook DJ. Health technology assessment in the ICU: noninvasive positive pressure ventilation for acute respiratory failure. *J Crit Care*. 2003;18(1):59–67.
- Ambrosino N, Vagheggini G. Non-invasive ventilation in exacerbations of COPD. Int J Chron Obstruct Pulmon Dis. 2007;2(4):471–6.
- Ram FS, Picot J, Lightowler J, Wedzicha JA. Noninvasive positive-pressure ventilation for treatment of respiratory failure due to exacerbations of chronic obstructive pulmonary disease. *Cochrane Database Syst Rev.* 2004;(1):CD004104. doi:10.1002/14651858.CD004104.
- 15. Plant PK, Owen JL, Elliott MW. Non-invasive ventilation in acute exacerbations of chronic obstructive pulmonary disease: long term survival and predictors of in-hospital outcome. *Thorax.*

2001;56(9):708-12. doi:10.1136/thorax.56.9.708.

 Thys F, Roeseler J, Reynaert M, Liistro G, Rodenstein DO. Noninvasive ventilation for acute respiratory failure: a prospective randomised placebo-controlled trial. *Eur Respir J*. 2002;20(3):545– 55.

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