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Research Article

EFFECT OF NON-INVASIVE VENTILATION VS CONVENTIONAL THERAPY IN ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH TYPE -2 RESPIRATORY FAILURE

Gourab Nayak, Geetanjali Panda, Manoranjan Pattanaik, Jeetendra Kumar Patra, Hemanta Kumar Sethy, Thitta Mohanty and Bijaya Kumar Meher*

Department of Pulmonary Medicine, SCB Medical College, Cuttack, Odisha

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A B S T R A C T

Background: Acute exacerbation of COPD is managed with oxygen therapy, bronchodilators, systemic steroids, antibiotics and as needed Non-invasive ventilation (NIV) and/or invasive mechanical ventilation. NIV reduces rates of intubation, mortality, complications and duration of hospital stay.

Methods: A randomised case control study carried out on 100 patients admitted for acute exacerbation of COPD with type 2 respiratory failure to Department of Pulmonary Medicine, SCB MCH, Cuttack during the period from May 2016 to December 2017. Patients selected randomly into study group were shifted to respiratory ICU, and put on NIV (BIPAP S/T), along with other medical management and compared with treatment outcome of patients on conventional treatment for COPD.

Results: **A**verage age in study group and control group were 61.0yrs (SD=10.83) and 66.42yrs (SD= 8.58) respectively with male 68% and females 32%. **Smoking** was associated with patients in both study and control groups with overall mean of 58%. Most common presentation were breathlessness and cough with expectoration. 43 out of 50 patients (86%) in study group and 38 out of 50 patients (76%) in control group were successfully treated. 3 patients (6%) underwent endotracheal intubation and 4 patients (8%) died in study group, where as in control group 6 patients (12%) underwent endotracheal intubation and 6 patients (12%) died

Conclusion: Early use of NIV for acidotic patients with acute exacerbation of COPD leads to more rapid improvement in clinical condition ($p<0.05$) , blood gas parameters ($p<0.05$), reduces need for invasive mechanical ventilation and decreases in-hospital morbidity and mortality.

INTRODUCTION

As per the recent GOLD guidelines 2017 report, COPD is defined as "a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases"[1]. COPD is considered as systemic disease. It is not only disease of lung but is also associated with significant extrapulmonary effects that may contribute to the severity in individual patients[2]. An acute exacerbation of COPD is defined as an event in the natural course of the disease characterised by a change in the patient's baseline dyspnea, cough, and/or sputum that is beyond normal day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD[1].

Corresponding author:* **Bijaya Kumar Meher
Department of Pulmonary Medicine, SCB Medical College, Cuttack, Odisha

The conventional management in acute exacerbation of COPD includes long term oxygen therapy (LTOT), bronchodilators, systemic steroids, antibiotics and mucolytics. Non-invasive ventilation (NIV) and invasive mechanical ventilation are the add on therapies in the management of acute exacerbation of COPD. Patients with COPD are prone to develop acute exacerbations, which pushes them into acute respiratory failure. NIV in these circumstances reduces rates of intubation, mortality, complications and duration of hospital stay. The biggest advantage of these techniques is their simplicity, ease of implementation and improved patient comfort allowing them to retain important functions like speech, cough and swallowing[3].

We conducted a single centre, prospective, randomized trial to compare the efficacy of non invasive ventilation delivered through a face mask, with standard medical treatment, in patients admitted because of acute exacerbations of chronic obstructive pulmonary disease.

Aims and Objectives

To study the effect of non-invasive ventilation in management of acute exacerbation of COPD with type 2 respiratory failure and compare the treatment outcome with patients on conventional treatment for COPD.

MATERIALS AND METHODS

The study was a randomised case control study carried out on patients admitted to Department of Pulmonary Medicine, SCB MCH, Cuttack during the period from May 2016 to December 2017. 100 Diagnosed cases of Chronic Obstructive Pulmonary Disease according to GOLD guidelines who were admitted for acute exacerbation of COPD with type 2 respiratory failure, were included in the study.

Exclusion Criteria

- Bronchial asthma and other chronic respiratory diseases mimicking COPD.
- Cardiac comorbidity like myocardial infarction, arrhythmia, pulmonary edema, congestive cardiac failure.
- Active pulmonary tuberculosis
- Pneumonia
- Unconscious patients
- Refusal to NIV
- Patient not willing to participate in study
- pH<7.2

Study method

The patients were randomised into one of two groups, i.e. study group and control group. Each group had 50 patients. All patients were treated with controlled oxygen therapy, antibiotics, bronchodilators and corticosteroids. Patients selected randomly into study group were shifted to respiratory ICU, and put on NIV(BIPAP S/T), along with other conventional modalities of treatment.

Non invasive positive pressure ventilation was delivered by a ventilatory support system with the patient in a semirecumbent position. The expiratory pressure was set at the minimal pressure level (4 cmH2O) and the inspiratory pressure was set at 8 cmH2O. In all patients, the inspiratory pressure and expiratory pressure was increased by 2 cm of water steps, until the patient showed signs of discomfort (increasing sensation of dyspnoea) or a pressure of 20 cmH2O was reached.

At time of admission, all patients were evaluated with detailed history, general physical examination, chest X-ray, ECG and arterial blood gas parameters complete blood count, blood electrolytes, renal function test and liver function test. All patients were reassessed after 1 hour and 24 hours of treatment clinically and with arterial blood gas analysis.

End point of study for each patient was defined as either failure or success. Failure was defined in advance as a deterioration in clinical status and deterioration in blood gases and/or haemodynamic status. Treatment failure includes all patients who were either intubated or died. Success was defined as improvement in clinical status and blood gases.

Statistical analysis

Data were entered into Microsoft excel and analysed using SPSS software version 21. Results were displayed using appropriate graphs/tables. Appropriate tests (for dependent groups/ independent groups) for statistical significance would be used. P value <0.05 would be taken as the level of significance.

Ethical Considerations: All patients were being well informed about the procedure by an information sheet and written informed consent were obtained. Confidentiality of data were maintained and simultaneously Ethical clearance from the institution's ethical Committee was obtained.

Observation

A total of 100 cases were included in the study. The patients were divided into 2 groups: Study and Control. Maximum number of patients belong to the age group of 56-70 years (45%). Males outnumbered the Females with Male: Female = 2.12:1. Maximum no. of patients (37%) were farmers by their occupation and all females were housewives. The main causes related to the development of COPD were previous or current smoking (58%) and biomass fuel exposure (34%). Associated most common co-morbidities were corpulmonale (27%), hypertension (25%) and diabetes mellitus (25%). Dyspnea (100%) and cough with expectoration (98%) were the most common symptoms. The principal signs were prolonged expiration (93%), use of accessory muscles (93%) and rhonchi (84%).On chest x-ray the major findings were hyperlucency of lung fields(82%) and low flat diaphragm (62%).The most common ECG finding was sinus tachycardia (100%) followed by right axis deviation (75%). So, with regards to patient characteristics and presenting features, both groups are similar. ($p > 0.05$).Mean respiratory rate was 30.66±2.91 in study group and 30.28±2.59 in control group initially. Greater improvement was shown by the patients receiving additional NIV rather than only medical management. Mean initial heart rate was 115.3±6.94 in study group and 116.8±5.75 in control group. Patients receiving additional NIV showed more imprvement than in patients receiving only medical management. Mean pH was 7.258±0.035 in study group and 7.254±0.026 in control group initially. Notable improvement was shown in study group than in control group. Mean of pCO2 was 82.88±8.30 in study group and 83.24±5.70 in control group initially. The patients receiving additional NIV showed more improvement than in patients receiving only medical management. Mean pO2 was 48.99±4.62 in study group and 48.21±3.41 in control group initially. Faster improvement was shown in the patients of study group. Bicarbonate level was identical in both groups initially and it remained identical in both groups throughout the course of treatment. Oxygen saturation was 74.96±6.56 in study group and 76.82±4.88 in control group at time of admission. Patients of study group had better improvement than control group.

86% of patients in study group were successfully treated where as 76% of patients in control group were successfully treated. 6% patients in study group needed endotracheal intubation and mechanical ventilation where as 12% in control group needed the same.

Table 1 Respiratory Rate

Respiratory rate	Study group	Control group	P value
Admission	30.66±2.91	30.28±2.59	0.492
1 hour	23.12±4.86	29.58±2.90	<0.001
24 hour	21.0±5.39	24.78±5.42	0.001

Table 1 shows a comparison between mean of respiratory rates between study group and control group, at time of admission, after 1 hour and after 24 hours. Respiratory rate is similar (p=0.492) in both groups at time of admission (30.66±2.91 vs 30.28±2.59). After 1 hour the improvement in study group is very significantly higher (p<0.001) than in control group (23.12±4.86 vs 29.58±2.90). After 24 hours there is more significant (p=0.001) improvement in study group (21±5.39 and 24.78±5.42).

Table 2 Heart Rate

Heart rate	Study group	Control group	P value
Admission	115.3±6.94	116.8±5.75	0.242
1 hour	98.76±16.26	114.86±8.58	<0.001
24 hour	95.62±17.34	106.08±16.54	0.003

Table 2 shows a comparison between mean of heart rates between study group and control group, at time of admission, after 1 hour and after 24 hours. Heart rate is similar in both groups at time of admission (115.3±6.94 vs 116.8±5.75, with p=0.242). After 1 hour the improvement in study group is much greater than in control group(98.76±16.26 vs 114.86±8.58). This is statistically very significant. After 24 hours there is remarkable improvement in both groups as compared to base line(95.62±17.34 and 106.08±16.54 respectively), with very significant improvement in study group (p=0.003).

Table 3 Change in Vitals

Vitals	Change	Study group	Control group
RR	1 hour	7.54	0.70
	24 hour	9.66	5.50
HR	1 hour	16.54	1.94
	24 hour	19.68	10.72

Table 3 shows a comparison between change of respiratory rate and heart rates between study group and control group, after 1 hour and after 24 hours. Decrease in respiratory rate by end of 1 hour is 7.54 and 0.70 in study group and control group respectively in comparison to baseline. At end of 24 hours, the decrease is 9.66 and 5.50 respectively. Decrease in heart rate by end of 1 hour is 16.54 and 1.94 in study group and control group respectively in comparison to baseline. At end of 24 hours, the decrease is 19.68 and 10.72 respectively.

Table 4 pH

pH	Study group	Control group	P value
Admission	7.258±0.035	7.254±0.026	0.499
1 hour	7.296±0.039	7.271±0.030	<0.001
24 hour	7.349±0.055	7.301±0.053	<0.001

Table 4 shows a comparison between mean of pH between study group and control group, at time of admission, after 1 hour and after 24 hours. pH is similar in both groups (p=0.499) at time of admission(7.258±0.035 vs 7.2540.026). Improvement in study group is highly significant (p<0.001) than in control group at end of 1 hour (7.296±0.039 vs 7.271±0.030). After 24 hours there is highly significant (p<0.001) improvement in study group as compared to control group (7.349±0.055 and 7.301±0.053 respectively).

Table 5 pCO2

pCO2	Study group	Control group	P value
Admission	82.88±8.30	83.24±5.70	0.802
1 hour	69.64±10.52	82.15±5.92	<0.001
24 hour	62.49±13.58	71.39±12.97	0.001

Table 5 shows a comparison between mean of pCO2 between study group and control group, at time of admission, after 1 hour and after 24 hours. pCO2 is similar (p=0.802) in both groups at time of admission(82.88±8.30 vs 83.24±5.70). After 1 hour the improvement in study group is much greater than in control group(69.64±10.52 vs 82.15±5.92). This improvement is highly significant (p<0.001). After 24 hours too, there is very significant (p=0.001) improvement shown in the study group as compared to control group(62.49±13.58 vs 71.39±12.97).

Table 6 pO2

pO2	Study group	Control group	P value
Admission	48.99±4.62	48.21±3.41	0.339
1 hour	54.81±4.13	51.84±3.97	<0.001
24 hour	59.16±5.73	55.34±5.24	0.001

Table 6 shows a comparison between mean of pO2 between study group and control group, at time of admission, after 1 hour and after 24 hours. pO2 is similar (p=0.339) in both groups at time of admission(48.99±4.62 vs 48.21±3.41). After 1 hour highly significant (p<0.001) improvement is noticed in study group (54.81±4.13 vs 51.84±3.97). After 24 hours also there is very significant (p=0.001) improvement in study group as compared to control group (59.16±5.73 and 55.34±5.24 respectively).

Table 7 Bicarbonate

HCO3	Study group	Control group	P value
Admission	37.17±3.21	37.68±2.96	0.422
1 hour	36.84±3.11	37.65±2.90	0.18
24 hour	34.45±3.04	35.17±2.90	0.22

Table 7 shows a comparison between mean of bicarbonate between study group and control group, at time of admission, after 1 hour and after 24 hours. Bicarbonate is similar in both groups at time of admission (37.17±3.21 vs 37.68±2.96), after 1 hour(36.84±3.11 vs 37.65±2.90) and after 24 hours(34.45±3.04 vs 35.17±2.90).(p > 0.05)

Table 8 SPO2

SPO2	Study group	Control group	P value
Admission	74.96±6.56	76.82±4.88	0.11
1 hour	82.48±5.67	80.44±5.10	0.06
24 hour	86.44±6.21	85.04±6.21	0.26

Table 8 shows a comparison between mean of SPO2 between study group and control group, at time of admission, after 1 hour and after 24 hours. SPO2 is similar in both groups at time of admission (74.96±6.56 vs 76.82±4.88). After 1 hour there is more improvement in study group(82.48±5.67 vs 80.44±5.10). After 24 hours there is similar improvement in both groups as compared to base line(86.44±6.21 and 85.04±6.21). But difference in the improvement in these two groups is not significant statistically. (p > 0.05)

Table 9 Change in Blood Gas

Blood gas	Change	Study group	Control group
pH	1 hour	0.038	0.017
	24 hour	0.091	0.047
pCO2	1 hour	13.24	1.09
	24 hour	19.95	11.85
pO2	1 hour	5.82	3.63
	24 hour	10.17	7.13

Table 9 shows a comparison between change in blood gas parameters between study group and control group, after 1 hour and after 24 hours. Increase in pH at end of 1 hour is 0.038 and 0.017 in study group and control group respectively in comparison to baseline. At end of 24 hours, the increase is

0.091 and 0.047 respectively. Decrease in pCO2 by end of 1 hour is 13.24 and 1.09 in study group and control group respectively in comparison to baseline. At end of 24 hours, the decrease is 19.95 and 11.85 respectively. Increase in pO2 at end of 1 hour is 5.82 and 3.63 in study group and control group respectively in comparison to baseline. At end of 24 hours, the increase is 10.17 and 7.13 respectively.

Table 10 Treatment outcome

Outcome		Study group	Control group	P value
Success		43	38	0.202
Failure	ETI	3	6	0.295
	Death	4	6	0.505

Table 10 shows a comparison between treatment outcomes of study group and control group. 43 patients in study group and 38 patients in control group were successfully treated. 3 patients were intubated in study group where as 6 patients were intubated in control group. 4 patients in study group and 6 patients in control group died. But this difference in outcome in the two groups is not statistically significant. ($p > 0.05$)

Table 11 Length of Hospital Stay (LOHS)

	Study group	Control group	P value
LOHS	12.88±4.11 days	16.36±5.16 days	<0.001

Table 11 shows a comparison between mean of length of hospital stay in study group and control group. Patients in study group stayed an average of 16.88±4.11 days in hospital, and patients in control group stayed an average of 16.36±5.16 days in hospital. This difference is highly significant ($p<0.001$).

Table 12 NIV Complications

Complication	No of pts	%
Pressure sores	5	10
Gastric distension	6	12
Dry mouth	3	6
Claustrophobia	7	14

Table 12 shows that in patients receiving NIV, claustrophobia is the most common complication(14%), followed by gastric distension(12%). Other complications are pressure sores(10%) and dry mouth(6%).

DISCUSSION

There was no significant difference in the age of the subjects in the both study and control groups. Patients in the study group averaged 61.0yrs (SD=10.83) while those in the control group averaged 66.42yrs (SD= 8.58). The maximum number of patients were within the age group of 56-70 years (45%) followed by age group of 71-85 years (30%).

In our study prevalence of male gender is 68% and females is 32%, that corroborates with study by Julio Cesar Mendes de Oliveria *et al* (2013)[4] where the prevalence of male was 79%. The majority of patients in both study and control groups were farmers (34% and 40% respectively) with a mean of 37%.This is because farming is the main occupation of Indian population. All females were housewives.

In our study, smoking was associated with majority of patients in both study and control groups (56% and 60% respectively) with overall mean of 58%.According to study by Dr.Vithalnarayandhadke *et al*.(2014)[5] smoking was associated with 75% of patients. The most common risk factor for female patients was biomass fuel exposure as evidenced in this study (90.62% of female patients, 34% overall).According to study by Julio Cesar Mendes de Oliveria *et al*. (2013)[4], the exposure to biomass combustion was present in 9.4% of patients.

In the present study corpulmonale, diabetes mellitus and hypertension were the major co-morbidities observed in both groups of patients. In the study group 26% had corpulmonale, 24% had diabetes mellitus and 22% had hypertension; similarly in the control group 28% had corpulmonale, 26% and 28% had diabetes mellitus and hypertension respectively. According to study by Gupta *et al*. (2011)[6] corpulmonale was observed in 41.7% of cases. According to study by Mapel DW (2000)[7] association of diabetes mellitus was 3-12% with COPD patients. In a study by Garica-Olmos L *et al*. (2013)[8] 20% patients had associated diabetes mellitus. According to study by Almagro P *et al*. (2010)[9] and Fumagalli G *et al*. (2013)[10] association of hypertension was 6-50% in COPD patients.

In this study, patients admitted with acute exacerbation of COPD presented mainly with breathlessness and cough with expectoration which was present in 100% and 98% of cases respectively. Roche *et al*.(2008)[11] found dyspnea in 99.2% patients of COPD and cough was present in 78.1% of the patients. According to more recent study by J P Singh *et al*[12], cough was present in 100% cases followed by dyspnea (92%) and sputum production (68%).

The majority of patients in both study and control groups were found to have prolonged expiration, accessory muscle use and rhonchi. In study group these findings were reported in 96%, 90% and 82% respectively; in control group the findings were reported in 90%, 96% and 86% respectively. The next common finding was reduced breath sound (76% and 80% in study and control group respectively).Other findings were barrel shaped chest(37%), crepitations (51%),pedal edema (19%), loud P2(14%) and elevated JVP(10%) etc. According to study by Dr Vithalnarayandhadke *et al*. (2014)[5]; 70% patients had rhonchi ,40% patients had barrel shaped chest , 30% patients had loud P2 and systolic murmur in tricuspid area. Elevated JVP, Pedal edema, Crepitations were present in 20% patients.

The Chest x-ray findings mainly included hyperlucency of lung fields(82%), low flat diaphragm(62%),increased bronchovascular markings(36%) and tubular heart (48%).Other findings were CTR>50% (17%) and peripheral vascular pruning(23%). In a study by Dr Vithalnarayandhadke *et al*.(2014)[5], increased bronchovascular markings in 39% of cases, changes of emphysema in 36% of cases, prominent central pulmonary artery in 30% of cases, cardiomegaly in 30% of cases and normal chest x-ray in 25% of cases.

In the present study, the most common ECG finding was sinus tachycardia (100%), Right axis deviation (in study and control group 80% and 70% respectively) followed by P pulmonale (in study and control group 34% and 30% respectively). Other finding was Poor progression of R wave (26%) .According to Hina Banker *et al* (2013)[13], the ECG findings were Right axis deviation (65% cases), P Pulmonale (35% of cases) and Low voltage QRS complex (22% of cases).ventricular hypertrophy (25%) and right axis deviation (19%) and bundle branch block (7%).

In this study, respiratory rate at time of admission was similar in study and control group (30.66±2.91/min and 30.28±2.59/min respectively). In study done by Celikel *et al*[14], baseline RR was 34±8.1 and 35±5.8. Follow up respiratory rates in our study are 23.12±4.86 and 29.58 ±2.90 in study group and control group at end of 1 hour, and 21±5.39 and 24.78±5.42 respectively at end of 24 hours which is similar to study of Brochard *et al*[15] where follow up respiratory rate was 33±7 and 25±8. Follow up RR in study by Celikel *et al*[14] were 24±6 in study group and 30±5 in control group at end of 1 hour. At end of 6 hours the RR were 19±6 and 27±8 respectively. So findings of our study are similar to above studies.

Heart rate in our study at time of admission was similar in study and control group (115.3±6.94 and 116.8±5.75 respectively). In study done by Celikel *et al*[14], baseline HR was 99±18 and 108±19. In study done by Thys *et al*[16] baseline HR was 122.4±22 and 122±20. So baseline HR in our study is similar to baseline HR in studies by Thys *et al*[16] and Brochard *et al*[15]. Follow up heart rates in our study are 98.76±16.26 and 114.86 ±8.58 in study group and control group at end of 1 hour, and 95.62±17.34 and 106.08±16.54 respectively at end of 24 hours. Follow up HR in study by Celikel *et al*[14] were 98±16 in study group and 104±21 in control group at end of 1 hour, and at theend of 6 hours the HR were 96±10 and 93±18 respectively. So findings of baseline and follow up heart rates of our study are similar to above studies.

In our study, the initial pH was similar in study group(7.258±0.035) and control group(7.254±0.026). In study done by Brochard *et al*[15], baseline pH was 7.27±0.10 and 7.28±0.11 respectively. In study of Khinlani *et al*[17], baseline pH was 7.23±0.07 in both study and control groups. Plant *et al*[18] found in their study that baseline pH was 7.32 in study group and 7.30 in control group. In study of Wood *et al*[19] baseline pH was 7.35±0.08 and 7.34±0.09 respectively. On follow up, in our study pH was 7.296±0.039 in study group and 7.271±0.030 in control group at end of 1 hour. At end of 24 hours, the respective values were 7.349±0.055 and 7.301±0.053. The follow up values in study of Brochard *et al*[15] were 7.31±0.09 in study group and 7.26±0.11 in control group at end of 1 hour. In study of Khinlani *et al*[17] the follow up pH values were 7.27±0.08 in study group and 7.22±0.09 in control group at end of 1 hour. At end of 24 hours, the follow up values were 7.37±0.08 and 7.35±0.1 respectively. In study by Plant *et al*[18] the follow up pH values were 7.342 and 7.324 in study and control group respectively at end of 1 hour, and 7.345 and 7.350 respectively at end of 4 hours. Wood *et al*[19] in their study found that follow up pH values were 7.42±0.06 and 7.39±0.12 in study and control group respectively at end of 1 hour. So our study is more or less similar to the above mentioned studies.

In our study, the initial pCO2 was similar in study group(82.88±8.30) and control group(83.24±5.70). In study done by Brochard *et al*[15],baseline pCO2 was 70±12 and 67±16 respectively. In study of Khinlani *et al*[17], baseline pCO2 was 85.4±14.8 and 81.1±11.6 in study and control groups respectively. Plant *et al*[18] found in their study that baseline pCO2 was 66.31 in study group and 65.03 in control group. In study of Wood *et al*[19] baseline pCO2 was 56.5±22.3 and 56.3±26.5 respectively. On follow up, in our study pCO2 was 69.64±10.52 in study group and 82.15±5.92 in control group at end of 1 hour. At end of 24 hours, the respective values were 62.49±13.58 and 71.39±12.97. The follow up values in study of Brochard *et al*[15] were 68±13 in study group and 67±16 in control group at end of 1 hour. In study of Khinlani *et al*[17]the follow up pCO2 values were 65.1±37.6 in study group and 86.2±20.6 in control group at end of 1 hour. At end of 24 hours, the follow up values were 58.1±24.3 and 68.4±18.5 respectively. In study by Plant *et al*[18] the follow up pCO2 values were 61.50 and 63.53 in study and control group respectively at end of 1 hour, and 60.45 and 60.82 respectively at end of 4 hours. Wood *et al*[19] in their study found that follow up pCO2 values were 53.3±17.6 and 49.3±19.6 in study and control group respectively at end of 1 hour. So result of our study is similar to studies of Brochard *et al*[15] and Khinlani *et al*[17].

In our study, the initial pO2 was similar in study group(48.99±4.62) and control group(48.2±3.41). In study done by Brochard *et al*[15],baseline pO2 was 41±10 and 39±12 respectively. In study of Khinlani *et al*[17],baseline pO2 was 61.18±14.73 and 61.50±15.06 in study and control groups respectively. Plant *et al*[18] found in their study that baseline pO2 was 51.73in study group and 52.63 in control group. In study of Wood *et al*[19] baseline pO2 was 59.8±20.7 and 71.3±22.7 respectively. On follow up, in our study pO2 was 54.81±4.13 in study group and 51.84±3.97 in control group at end of 1 hour. At end of 24 hours, the respective values were 59.16±5.73 and 55.34±5.24. The follow up values in study of Brochard *et al*[15] were 66±17 in study group and 58±24 in control group at end of 1 hour. In study of Khinlani *et al*[17] the follow up pO2 values were 67.4±20.09 in study group and 64.10±26.07 in control group at end of 1 hour. In study by Plant *et al*[18] the follow up pO2 values were 56.39 in both study and control group at end of 1 hour, and 56.24 and 60.97 respectively at end of 4 hours. Wood *et al*[19] in their study found that follow up pO2 values were 96.5±78.3 and 79.2±30.3 in study and control group respectively at end of 1 hour. So result of our study is identical to above studies.

In our study, the initial bicarbonate levels were similar in study group(37.17±3.21) and control group(37.68±2.96). In study done by Brochard *et al*[15], baseline bicarbonate was 32±7 and 33±7 respectively. In study of Khinlani *et al*[17],baseline bicarbonate was 35.40±6.16 and 35.65±4.69 in study and control groups respectively. Bardi*et al*[20] found in their study that baseline bicarbonate was 34.2±4.2 in study group and 31.6±5.9 in control group. On follow up, in our study bicarbonate was 36.84±3.11 in study group and 37.65±2.90 in control group at end of 1 hour. At end of 24 hours, the respective values were 34.45±3.04 and 35.17±2.90. In study of Khinlani *et al*[17] the follow up bicarbonate values were 35.47±7.53 in study group and 36.67±5.78 in control group at end of 1 hour. In study by Bardi*et al*[20] the follow up bicarbonate values were 30.1±3.4 and 28.0±3.6 in study and control group respectively at time of discharge. So the result of our study is comparable to studies by Brochard *et al*[15],Khinlani *et al*[17]and Bardi *et al*[20].

In our study the baseline SPO2 was 74.96±6.56 and 76.82±4.88 in study group and control group respectively at time of admission. In study by Thys *et al*[16],baseline SPO2 of COPD patients was 84±14.7 and 88.4±8.8 in study and control groups respectively. Khinlani *et al*[17] in his study found baseline SPO2 to be 88.78±4.96 in study group and 90.05±6.02 in control group. On follow up, our study revealed SPO2 of 82.48±5.67 in study group and 80.44±5.10 in control

group at end of 1 hour. At end of 24 hours, SPO2 in study group was 86.44±6.21 and that in control group was 85.04±6.21. In study of Thys *et al*[16] follow up SPO2 in study group was 83.2±18.7 and that in control group was 91.3±3.8. In study by Khinlani *et al*[17] follow up SPO2 at end of 1 hour was 90.78±5.57 in study group and 92.00±5.94 in control group. Result of our study is in line with study of Khinlani *et al*[17].Butresults of our study seem different from results of study by Thys *et al*[16]. This may be attributed to very small sample size of COPD patients, total sample size being 12 patients only.

In our study, 43 out of 50 patients (86%) in study group and 38 out of 50 patients (76%) in control group were successfully treated. 3 patients (6%) underwent endotracheal intubation and 4 patients (8%) died in study group, hence failure being 14%. 6 patients (12%) underwent endotracheal intubation and 6 patients (12%) died in control group, hence failure being 24%. In study by Avdeev *et al*[21] the need of endotracheal intubation was found in 12% in study group and 28% in control group, and mortality was 8% and 31% respectively. Hence failure was observed in 20% patients in study group and 59% in control group which is at par with our study. In study by Brochard *et al*[15] the need of endotracheal intubation was found in 26% in study group and 31% in control group, and mortality was 9% and 29% respectively. Hence failure was observed in 35% patients in study group and 60% in control group. So this study is also similar to our study. In study by Khinlani *et al*[17] the need of endotracheal intubation was found in 15% in study group and 60% in control group, and mortality was 10% and 15% respectively. Hence failure was observed in 25% patients in study group and 75% in control group. These findings are in line with the findings of our study. In study of Bardi *et al*[20] failure was seen in 7% of patients in study group and 20% of patients in control group, which is similar to finding of our study. In study by Barbe *et al*[22] no patient needed endotracheal intubation and no patient died. This may be due to the fact that the subjects were relatively more stable as compared to other studies.

The mean duration of hospital stay in our study is 12.88±4.11days in study group and 16.36±5.16 days in control group. Mean hospital stay in study by Celikel *et al*[14] was 11.7±3.5 days and 14.6±4.7 days, which is similar to our study. In study by Avdeev *et al*[21],mean hospital stay was 26±7 days in study group and 34±10 days in control group, which is longer than mean hospital stay of our study. In study of Brochard *et al*[15], mean hospital stay in study group was 23±17 days and that in control group was 35±33 days. This is similar to finding of Avdeev *et al*[21] .In study by Khinlani *et al*[17] mean hospital stay was 9.4±4.3 days in study group and 17.8±2.6 days in control group, which is at par with the finding of our study.

In our study most common complication was in form of claustrophobia, which was seen in 7 out of the 50 patients (14%) receiving NIV. 6 patients (12%) complained of gastric distension on receiving NIV. Pressure sores over bridge of nose or malar areas was observed in 5 out of the 50 patients (10%) subjected to NIV. Dry mouth was observed in 3 patients (6%). Overall these complications were seen in 11 patients (22%), which is similar to the complications found (16%) in the study by Brochard *et al*[15].

Practically most clinical studies and meta-analysis agree to the efficacy of non-invasive ventilation in management of acute exacerbation of chronic obstructive pulmonary disease; however data regarding the exact usefulness of non-invasive ventilation in acute exacerbation of chronic obstructive pulmonary disease have been inconclusive.

Despite many studies of the effectiveness of non-invasive ventilation for different population, few have been performed exclusively on patients with COPD. The use of non-invasive ventilation in patients of acute exacerbation of COPD is beneficial as compared to conventional management alone, as discussed in our study.

CONCLUSION

86% of patients in study group were successfully treated where as 76% of patients in control group were successfully treated. 6% patients in study group needed endotracheal intubation and mechanical ventilation where as 12% in control group needed the same.

Hence, the early use of NIV for acidotic patients with acute exacerbation of COPD leads to more rapid improvement in clinical condition ($p<0.05$) and blood gas parameters ($p<0.05$). There is also a reduction in the need for invasive mechanical ventilation (with objective criteria), and a reduction in in-hospital morbidity and mortality, although this was not found to be significant statistically. ($p>0.05$).

Our study analysed the management of acute exacerbation of COPD, with special reference to use of non-invasive ventilation in tertiary care hospital. We established the differences in the treatment outcomes of patients receiving non-invasive ventilation in addition to medical management, and those receiving only medical management for COPD. There was reduction in number of patients undergoing endotracheal intubation and death (but not significant statistically), in the patients who received add on non-invasive ventilation as compared to those receiving only medical management. The patients receiving non-invasive ventilation also showed rapid improvement in clinical condition and blood gas parameters (significant statistically).

So according to our study, use of NIV is an important strategy in management of exacerbations in COPD patients, particularly in respiratory acidosis with type 2 respiratory failure. We therefore support the recommendation that NIV should be used in management of acute exacerbations of COPD.

LIMITATIONS OF THE STUDY

Lack of a blind placebo comparison group, Small sample size were the limitations of our study. Again our study does not predict which patients are likely to benefit most from use of NIV, not evaluate role of NIV in stable COPD patients, and does not consider the cost effectiveness of use of NIV against the additional benefit it confers.

Hence further studies are needed across different centres to examine exact advantage of use of NIV in patients of acute exacerbation of COPD. Studies also need to be conducted to predict which subset of patients are most likely to benefit from use of NIV, and which subset are not. Use of NIV in stable COPD patients needs to be evaluated. In addition, cost effectiveness also needs to be analysed. This would permit the

effectiveness of NIV to be determined with greater precision. Hence, it is essential that we continue the research to evaluate the effectiveness of NIV in COPD patients, so that management can be optimized.

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