

**ORIGINAL ARTICLE**

**CHANGES IN ARTERIAL BLOOD GASES AND RESPIRATORY RATE**

**BEFORE AND AFTER NON INVASIVE POSITIVE PRESSURE**

**VENTILATION IN ACUTE EXACERBATION OF COPD**

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**ABSTRACT:****Objectives:**

To compare Respiratory Rate, pH, pCO<sub>2</sub> before and after Non Invasive Positive Pressure Ventilation in Acute Exacerbation of COPD

**Design of study:**

An Interventional study (Quasi Experimental)

**Place and duration of study:**

Pulmonology department of Post Graduate Medical Institute Lady Reading Hospital Peshawar, from Oct. 2003 to Oct, 2004.

**Material and methods:**

Fifty patients were recruited in the study. The patients were clinically assessed and relevant investigations done before starting on NIPPV. The parameters i.e. respiratory rate, pH and PCO<sub>2</sub> were regularly monitored. The NIPPV was given for at least 6-8 hours in the first 24 hours. It was continued longer according to the patient response and tolerance. Clinical signs and arterial blood gases level were checked 6-8 hours after start of NIPPV.

**Results:**

The mean respiratory rate decreased from 24-16 $\pm$  3.47 per minute to 17.40 $\pm$  3.14 per minute (P <0.0001). There was significant improvement

in pH from  $7.28 \pm 4.39$  to  $7.35 \pm 8.28$ . ( $P < 0.0001$ ), and reduction in  $p\text{CO}_2$  from  $63.750 \pm 10.01$  mmHg to  $56.22 \pm 11.40$  mmHg, ( $P < 0.0001$ ).

**Conclusion:**

There were remarkable improvement in pH,  $\text{PCO}_2$  and respiratory rate after Non-Invasive positive pressure ventilation (NIPPV). NIPPV is a effective, simple and affordable addition to the treatment of life threatening chronic obstructive pulmonary disease.

**Key words:**

Non invasive positive pressure ventilation, chronic obstructive pulmonary disease, Arterial blood gases.

## **INTRODUCTION**

Chronic obstructive pulmonary disease (COPD) is associated with prolonged morbidity and high mortality. And acute exacerbation of C.O.P.D is a major cause of admission to hospital through out the world.<sup>1</sup> A proportion of these patients have more severe disease with respiratory failure and acidosis and this has poor treatment outcome and increased mortality.<sup>2</sup> During these episodes a major deterioration in gas exchange is accompanied by a worsening in the clinical condition of the patient, characterized by a rapid and shallow breathing pattern, severe dyspnoea, right ventricular failure and encephalopathy. The pathophysiological pathway of all these features is the inability of the respiratory system to maintain adequate alveolar ventilation in the presence of major abnormalities in respiratory mechanics. Unfortunately, the ability of medical treatment to reverse severe respiratory failure in these patients is limited. When hypoventilation become so severe that several organ dysfunction occur, there is no choice other than to provide “artificial” ventilation to avoid a fatal outcome. The traditional way has been to use endotracheal intubation as a mean of access to the lower airways and to deliver ventilation to the patient lung. Endotracheal intubation and mechanical ventilation can be a life saving procedure in such patients. But unfortunately it also carries risk of causing trauma and inducing severe infection to the airways, thereby increasing mortality by itself. To avoid

such risks, non invasive, positive pressure ventilation. (NIPPV) has been in use for over a decade<sup>2-4</sup>.

NIPPV refers to the provision of ventilatory support through the patients upper airway using mask or similar device. It is mainly used in acute hypercapnic respiratory failure. It has a number of potential advantages, particularly the avoidance of tracheal intubation with its associated morbidity and mortality from problems such as pneumonia. Intermittent ventilatory assistance is possible with NIV allowing gradual weaning and also normal eating, drinking and communication<sup>6,7</sup> NIPPV decrease inspiratory muscle effort and respiratory rate and increases tidal volumes and O<sub>2</sub> saturation in patients with COPD both when stable and during acute exacerbation. Arterial Pa O<sub>2</sub> increases and Pa CO<sub>2</sub> decreases with NIPPV<sup>1-5</sup>.

This study was conducted to assess the role of NIPPV in acute exacerbation of COPD. The main objective of the study is to see improvement in respiratory rate and arterial blood gases

### **AIMS AND OBJECTIVES:**

Objective of the study is to compare Respiratory rate, pH and pCO<sub>2</sub> before and after NIPPV.

### **HYPOTHESIS**

1. The respiratory rate before NIPPV will be significantly more than after NIPPV
2. pH before NIPPV will be significantly lower than after NIPPV

3.  $PCO_2$  before NIPPV will be significantly higher than after NIPPV.

## **MATERIAL AND METHODS**

### **Sample size:**

Total number of patients=50

### **Type of study:**

Interventional study (quasi experimental)

### **Sampling Technique:**

Convenient non probability

We included 50 patients, diagnosed as COPD with acute exacerbation admitted in chest unit LRH. A detailed history of the duration and spectrum of symptoms was taken. Full details of past, medical illness drugs taken, family, personal history particularly smoking history was included, followed by a detailed clinical examination. After that blood for arterial blood gases (ABG) analysis was taken. After 6 to 8 hours of non-invasive ventilation ABG were repeated. Monitoring included clinical assessment combined with pulse oximetry and arterial blood gas tensions.  $O_2$  saturation was monitored continuously for at least 24 hours after commencing NIPPV. NIPPV was provided with Bipap S/T-D Ventilatory support system (Respironics, Inc. USA) with Inspiratory positive airway pressure (IPAP) in range of 4-30cm  $H_2O$  and Expiratory positive

airway pressure (EPAP) range 4-30cm H<sub>2</sub>O and having spontaneous (S), Spontaneous/Timed (S/T) or timed (T) mode.

We started our patients initially on Expiratory positive airway pressure (EPAP) 4cm H<sub>2</sub>O and Inspiratory positive airway pressure (IPAP) 9cm H<sub>2</sub>O, then slowly increasing IPAP, up to 15cm H<sub>2</sub>O on S/T mode.

The NIPPV was given for at least 6-8 hours in the first 24 hours. It was continued longer according to the patient response and tolerance. Clinical signs and arterial blood gases were checked 6-8 hours after start of NIPPV.

If there was deterioration of patient clinical condition and pulmonary function variables despite treatment, patient was shifted to I.C.U for more definitive treatment; if arterial blood pH remained stable for 12 hours after last pause, treatment with NIPPV was terminated and declared successful improvement and patient discharged accordingly.

NIPPV was applied as continuously as possible during its initial application. When weaning was attempted after improvement in patient's respiratory status, this was done either by gradual reduction in the levels of ventilator supports or by initiating brief periods without NIPPV, which were increased in duration as tolerated by the patient.

## **INCLUSION CRITERIA:**

Patients who were eligible to enter into study, were known cases of, or had a high probability of, COPD (on basis of clinical history, physical examination, Chest radiograph and spirometry) and presented in acute exacerbation with hypercapnoeic respiratory failure with:

- Dyspnoea at rest or respiratory rate > 25 breaths per minute,
- FEV<sub>1</sub> 50% predicated or PEFR ≤ 100 lit per minute.
- ABGs showing pH < 7.35.

Pa Co<sub>2</sub> > 45 mmHg

PaO<sub>2</sub> < 60 mmHg

### **EXCLUSION CRITERIA:**

Patients were excluded:

- i. who had arterial blood pH < 7.20
- ii. who were unconscious.
- iii. Who were unable to spontaneously clear their airways.
- iv. with other medical condition requiring immediate tracheal intubation.
- v. having haemodynamic instability.
- vi. unable to cooperate with NIPPV.

### **DATA ANALYSIS:**

Data was analyzed on statistical package software SPSS version 10. Frequency and percentage was computed for qualitative variables like sex, smoking status, age groups and complications. Mean and standard deviation was computed for quantitative

variables, age, respiratory rate, pH and PCO<sub>2</sub> before and after non invasive ventilation. Pair t-test was used to check the difference between before non invasive ventilation and after non invasive ventilation for respiratory rate, pH and PCO<sub>2</sub> with 0.05 level of significance.

## **RESULTS:**

In these 50 patients 46 (92%) were male and 4 (8%) were female. The age range was from 40-85 years and the mean age was  $64.98 \pm 10.32$ . The age distribution of population is given in Table 1.

Duration of study was 1 year. Among the 46 male patients 42 (91.3%) were smoker and 4 (9.3%) were non smoker, while among the 4 female patients only 1 (25%) was smoker, so total smoker patients were 43 (86%) and non smoker were 7 (14%).

The mean respiratory rate was  $24.16 \pm 3.47$  per minute before and  $17.40 \pm 3.14$  per minute after NIPPV ( $P < 0.0001$ ). The mean PH before NIPPV was  $7.28 \pm 4.39$  and afterwards was  $7.35 \pm 8.28$  ( $P < 0.0001$ ). The mean PCO<sub>2</sub> was  $63.750 \pm 10.01$  mmHg before and  $56.22 \pm 11.40$  mmHg after NIPPV ( $P < 0.0001$ ).

Regarding complications directly associated with non invasive positive pressure ventilation (NIPPV) in our study, mucus plug obstruction of airways occurred in 1 patient (2%). In 5 (10%) patients there were eye irritation and in 1 (2%) patient there was transient

hypotension. In 4(8%) patients there were skin abrasions. Nasal congestion was reported in 4(8%) patients after NIPPV. In 10(20%) patients there were mask intolerance.

**Table-1: Age Distribution**

<b>Age range</b>	<b>No of patients</b>	<b>Percentage</b>
40-50	4	8%
51-60	26	52%
61-70	10	20%
71-80	7	14%
≥81	3	6%



**Table No 2**

**Analysis of Variable amongst 50 patients studied**

<b>Variables</b>	<b>Mean</b>	<b>Std. Deviation</b>	<b>P. Value</b>
Resp rate before	24.16	3.47	<0.0001
Resp rate after	17.40	3.14	
Ph before	7.28	4.39	<0.0001
Ph after	7.35	8.28	
PCO <sub>2</sub> before	63.75	10.01	<0.0001
PCO <sub>2</sub> After	56.22	11.40	

**Table No 3**

**Complications of non invasive ventilation**

<b>Complications</b>	<b>Number of patients</b>	<b>Percentage</b>
NIPPV related mucus plugs	1	2%
Eye irritation	5	10%
Transient Hypotension	1	2%
Mask Intolerance	10	20%
Skin Abrasion	4	8%
Nasal Congestion	4	8%

NIPPV: Non invasive positive pressure ventilation.

## DISCUSSION

Our study confirms the benefits expected of NIPPV in acute exacerbation of COPD. The patients were assessed for the change in respiratory rate, pH and  $PCO_2$  before and after NIPPV. Acidosis being the main indicator of the severity of acute exacerbation of COPD, was the main parameter of our study. In 45 out of fifty patients pH improved to normal value. This is compatible with other trials<sup>12-18</sup>.

According to Brochad et al<sup>8</sup> and Confalonieri<sup>18</sup> there is eighty percent success rate and according to Anton<sup>14</sup>, 77% of patients were successfully treated with NIV.

In study by Soo Hoo<sup>15</sup> it has been shown that NIV is not effective in acute respiratory failure in COPD<sup>10</sup>. Only one randomized study has failed to show a benefit for NIPPV and this study was very small<sup>21</sup>.

In 46 out of 50 patients in our study,  $PCO_2$  decreased from initial high values which is also compatible with other trials<sup>5-7</sup>. According to Kramer and Meyar<sup>11</sup> there is decrease in  $PCO_2$  in about 94% of cases.<sup>11</sup>

A change in respiratory rate is also reported as a prognostic factor. In our study the respiratory rate declined in all 50 patients. The fall in respiratory rate is probably explained by an increase in tidal volume which allows the respiratory rate to fall, thus protecting against fatigue, while allowing minute ventilation to be maintained. The improvement in tidal volume may occur with medical treatment and/or with NIV. It has been shown that NIV reduces respiratory rate<sup>1,4,7</sup> which was also found in our

study. According to Meduri GU and Turner there is 90% success in decreasing respiratory rate.<sup>9</sup>

Weaning from NIV was started when no clinical signs of respiratory distress were observed during the last phase. Then if arterial blood gas level were stable ( $\text{pH} > 7.35$ ) after a 12 hours pause, therapy was considered terminated, provided the original cause of exacerbation was under control.

The rate of complication directly associated with NIPPV therapy in our study was acceptably low. A large mucus plug obstructed airways in 1 patient (2%) which was probably due to reduced ability to cough during NIPPV. Lajer and Janniquin reported large mucus plugs in 10% of their patients<sup>19</sup>. In 4 patients (8%) there were eye irritation and in 1 patient (2%) there was transient hypotension in our study. Meduri and Conoscent showed also transient hypotension in 2% cases and eye irritation in 8% cases.<sup>20</sup> In 2 patients (4%) there were skin abrasions. Nasal congestion was reported in 2 patients (4%) after NIPPV. In 10 patients (20%) there were mask intolerance and it was also reported in Thomas J Martin study<sup>5</sup>.

## **CONCLUSION**

Both the clinical and biochemical variables of acute exacerbation of COPD improved significantly after the application of non invasive positive pressure ventilation (NIPPV).

## REFERENCES

1. British Thoracic Society Standards of Care Committee. Guideline non-invasive ventilation in acute respiratory failure. *Thorax* 2002;57:192-211.
2. Elliot MW. Non invasive ventilation in acute exacerbation of COPD. In: Simonds Ak, Non-invasive respiratory support. 2<sup>nd</sup> ed. New York' Oxford University Inc, 2002:30-42.
3. Plant PK, Owen J, Elliot MW. One year period prevalence study of respiratory acidosis in acute exacerbation of COPD; implications for the provision of NIV and O<sub>2</sub> administration. *Thorax* 2000;55:550-4.
4. Plant PK, Owen JL, Elliott MW. A multi center randomized control trial of the early use of non invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards. *Lancet* 2000;355:1931-5.
5. Martin TJ, Havis JD, Costantino JP. A randomized prospective evaluation of non invasive ventilation for acute respiratory failure. *Am J Respire Crit Care Med* 2000;161:807-13.
6. Confalonieri M, Parigi P, Scartaballati A. Non-invasive mechanical ventilation improve the immediate and long-term outcome of COPD patients with acute respiratory failure. *Eur Respir J* 1996;9:722-30.

7. Diaz O, Iglesia R, Frerrer M. Effects of noninvasive ventilation on pulmonary gas exchange and hemodynamics during acute hypercapnic exacerbations of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 1997;156:1840-5.
8. Brochard L, Mancebo J, Wysocky M. Non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease, *N Engl J Med.* 1995;333:817-22.
9. Medurin GU, Turner E, Abou-Shala N. Non-invasive positive pressure ventilation via face mask: first-line intervention in patients with acute hypercapnic and hypoxemic respiratory failure. *Chest* 1996;109:179-93.
10. Soo Hoo G, Santiago S, Williams A. Nasal mechanical ventilation for hypercapnic respiratory failure in chronic obstructive pulmonary disease, determinants of success and failure *Crit Care Med.* 1994;22:1253-61.
11. Kramer N, Meyer TJ, Meharg J. Randomized prospective trial of noninvasive positive pressure ventilation in acute respiratory failure. *Am J Respir Crit Care Med.* 1995;151:1799-806.
12. Poponick JM, Renstom JP, Bennett RP. Use of a ventilatory support system for acute respiratory failure in the emergency department. *Chest* 1999;116:166-71.

13. Benhamou D, Girault C, Faure C. Nasal mask ventilation in acute respiratory failure. *Chest* 1992;102:912-17.
14. Anton A, Guell R, Gomez J. Predicting the result of noninvasive ventilation in severe acute exacerbations of patients with chronic airflow limitation. *Chest* 2000;117:828-33.
15. Soo Hoo GW, Hakimian N, Santiago SM. Hypercapnic respiratory failure in COPD patients. *Chest* 2000;117:169-77.
16. Jeffery AA, Warren PM, Flenley DC. Acute hypercapnic respiratory failure in patients with chronic obstructive lung disease: risk factors and use of guidelines for management. *Thorax* 1992;47:34-40.
17. Warren PM, Flenley DC, Millar Js. Respiratory failure revisited: acute exacerbation of chronic bronchitis between 1961-68 and 1970-76. *Lancet* 1980;:467-70.
18. Confalonieri M, Potena A, Carbone G. Acute respiratory failure in patients with severe community acquired pneumonia. A prospective randomized evaluation of noninvasive ventilation. *Am J Respir Crit Care Med* 1999;160:1585-91.
19. Lager P, Jenniquin J, Gausergues P, Robert D. Acute respiratory failure in COPD patients treated with non invasive intermittent mechanical ventilation control mode with nasal mask. *Am rev of respir dis* 1988;137-63.

20. Meduri GU, Conscent CC, Menaski P, Nair S. Noninvasive face mask ventilation inpatients with acute respiratory failure. Chest 1989;95:865-70.
21. Barbe F, Togoires B, Rubi M. Non invasive ventilatory support does not facilitate recovery from acute respiratory failure in COPD. Eur Respir J 1999;9:1240-5.