ORIGINAL RESEARCH PAPER

Pulmonary Medicine

ROLE OF NON INVASIVE VENTILATION IN TYPE1 AND TYPE 2 RESPIRATORY FAILURE

KEY WORDS: NIV, Respiratory Failure, Mechanical Ventilation.

Dr. Ameet H Senior Resident, Dept. of Pulmonary Medicine, AIIMS Patna

Dr. Ajay Kumar Verma Senior Resident, Dept. of Anaesthesia, JNUIMSRC, Jaipur, Rajasthan.

Dr. Akanksha Monil Parsana* Assistant Professor, Dept. of Anaesthesia, PSMC, Karamsad, Gujarat. *Corresponding Author

ABSTRACT

Introduction- NIV has role in the management of acute respiratory failure. NIV can reduce intubation and mortality rates in patients with severe respiratory failure. **Aim-** To assess effectiveness of NIV for respiratory failure. **Materials and Methods-** prospective observational study on 50 patients who received NIV as the initial ventilator support for respiratory failure. **Conclusion-** NIV is a safe and effective for ventilatory support in patients with acute respiratory failure. **Result-** NIV reduced need for intubation and mechanical ventilation. It also reduces the mortality and morbidity and shortens the ICU and hospital stay of patients.

INTRODUCTION:

Non-invasive ventilation (NIV) refers to the application of artificial ventilation without any conduit access to the airway. NIV has role in the management of acute respiratory failure[1-4]. NIV prevents complications associated with invasive ventilation like intubation problems and ventilator associated pneumonia[5-8] . Patient selection, appropriate application of interface and proper monitoring determine the success of NIV. NIV can reduce intubation and mortality rates in patients with severe respiratory failure[4-5].

AIM:

1. To study the clinical application of NIV in respiratory failure.
2. To assess ABGA before and after NIV.
3. To assess effectiveness of NIV for respiratory failure.

MATERIALS AND METHODS:

This prospective observational study was conducted on 50 patients who received NIV as the initial ventilator support for acute respiratory failure.

Inclusion Criteria:

1. Age >18yrs
2. Alert, cooperative, conscious patients.
3. Respiratory rate > 24/min.
4. Patients with respiratory failure

Exclusion Criteria:

1. Hemodynamic instability
2. Uncooperative patient.
3. Life threatening refractory hypoxemia.
4. Facial deformity.
5. Need for airway protection.
6. Electrocardiogram instability
7. Severe acidosis (pH<7.10)

METHODOLOGY:

BIPAP was started IPAP 8 cmH2O and EPAP 4 cm H20.

Technique:

1. Head end elevated to 45°angle
2. Clear face mask.
3. Strap well adjusted
4. Nasogastric tube in place.
5. Fio2 to keep SpO2≥90%.
6. Adjust ventilator settings SpO2 and ABGA

If there was hypercapnia IPAP was increased by 2cmH2O and if there was hypoxemia IPAP and EPAP were increased by 2 cm of H2O. Maximum IPAP 18cmH2O and EPAP 10cmH2O. Continuous application of NIV was encouraged. Bronchodilators, steroids, antibiotics and diuretics were given as indicated. Monitoring of vitals, SpO2 and ABGA was done. On this basis ABGA at 1 hour of and at subsequent intervals at weaning and 6 hours after weaning were done. Upon deterioration at any point during NIV, endotracheal intubation was considered. Weaning failure was defined as the need for invasive mechanical ventilation or inability to wean the patient off NIV due to worsening.

1. Loss of consciousness.
2. Persistent hypotension.
3. Worsening of respiratory distress.
4. Respiratory rate>40/ min.
5. SpO2<90% despite FiO2of 100%.
6. Cardiorespiratory arrest.
7. Worsening ABGA.

Upon improvement in ABGA, the pressure was decreased in gradations of 2cmH20 till IPAP was 8-10cmH20 and EPAP was 4cm H2O and weaning was initiated when the patient had

1. Clinical improvement respiratory rate to <24/min.
2. Normal ABGA
3. SpO2≥90%.

Then the application was switched over to intermittent use.
End point of study:

1. Relief of symptoms
2. Need for intubation
3. Successful weaning

OBSERVATIONS AND RESULTS:

In our study 41outof 50 patients were in the age group of 41- 69 (82%) with mean age of 55 years. In our study, males 44(88%) with female 6(12%). In the present study the most common underlying clinical condition was Chronic obstructive pulmonary disease 34(68%) followed by pneumonia 7(14%), Interstitial lung disease 4(8%), bronchiectasis 3(6%) and Acute pulmonary oedema 2(4%).

The mean PO2 for Type1 Respiratory failure was 50.68 at admission, 68.38 at 1hour post NIV, 83.6 at weaning and 103.15 at 6 hrs post weaning. For Type 2 Respiratory failure the mean PO2 at the time of admission was 53.61, 74.43 at 1 hour post NIV, 113.47 at the time of weaning and 124.08 at 6 hours post weaning.

The mean PCO2 for Type 1 Respiratory failure was 36.61 at admission, 36.77 at 1 hour post NIV, 36.43 at the time of

weaning and 36.38 at 6 hours post weaning. For Type 2 Respiratory failure the mean PCO2 at the time of admission was 72.78, 60.01 at 1 hour post NIV, 47.16 at the time of weaning and 42.58 at 6 hours post weaning.

The mean pH for Type 1 Respiratory failure was 7.327 at admission,7.395 at 1hour postNIV, 7.400 at weaning and 7.400 at 6 hours post weaning. For Type 2 Respiratory failure the mean pH at the time of admission was 7.268, 7.319 at 1 hour post NIV,7.378at weaning and7.386 at 6hours post weaning.

The mean PO2 in NIV successful patients for Type 1 Respiratory failure was 50.68 and for failure patients was 47.48. The mean PO2 in NIV successful patients for Type 2 Respiratory failure was 53.61 and for failure patients was 50.12. The mean PCO2 in NIV successful patients for Type 1 Respiratory failure was 36.61 and for failure patients was 33.80.The mean PCO2 in NIV successful patients for Type 2 Respiratory failure was 72.78 and for failure patients was 78.57.

The mean pH in NIV successful patients for Type 1 Respiratory failure was 7.327 and for failure patients was 7.279. The mean pH in NIV successful patients for Type 2 Respiratory failure was 7.268 and for failure patients was 7.203.

The final outcome of NIV for type 1 respiratory failure was a success rate of 56% and failure rate of 44% while the success rate of NIV for type 2 respiratory failure was 88% and failure rate was 12%.

The most common reason for discontinuation of NIV was Hemodynamic instability seen in 5 patients (46%) followed by inability to decrease dyspnoea in 2 patients (18%), intolerance of mask in two patients (18%) followed by inability to correct ABG value in one patient (9%) and skin erosion in one patient (9%).

The most common clinical condition leading to NIV failure were those associated with hypoxemic respiratory failure like acute pulmonary oedema (50%), interstitial lung disease (50%) followed by pneumonia (42%) followed by bronchiectasis (33%). The clinical condition associated with hypercapnic respiratory failure was found to be least failure with Chronic obstructive pulmonary disease at 11%.

The duration of NIV in Type 1respiratory failure was found to be 48 hours and in type 2 respiratory failure was found to be 72 hours. The mean entire duration of NIV was found to be 64 hours.

DISCUSSION:

Respiratory failure is a common cause of illness and death. Treatment of patients who develop acute respiratory failure often mandates mechanical ventilatory assistance. This traditionally required endotracheal intubation. In an effort to avoid the morbidity and mortality associated with endotracheal intubation and mechanical ventilation modalities have been developed to provide non invasive positive pressure ventilation.

Our study population comprised of 50 patients. Most of the patients were in the age group of 40-69(82%) mean age55 years which was comparable to other studies by Sailaja et al, Arnaud et al and Ige et al. The Sex distribution in the present study showed a male predominance of 88% and a similar study by Vishal et al and Shailaja et al.

The study population was divided into two groups those with Hypoxemic (type 1) respiratory failure and those with Hypercapnic respiratory failure (type 2). The study population in the group with Hypoxemic (type 1) respiratory failure was 16 and those with hypercapnic respiratory failure was 34. The underlying respiratory illness in those patients with type 1 respiratory failure was pneumonia in 7 patients (14%), interstitial lung disease in 4 patients (8%), bronchiectasis in 3 patients (6%), acute pulmonary oedema in 2 patients (4%) and the underlying respiratory illness in those patients with type 2 respiratory failure was chronic obstructive pulmonary disease in 34 patients (68%).Thus the predominant group was those with type 2 respiratory failure.

The mean PO2 of the present study on admission was 50.68 for type 1 respiratory failure and 53.61 for type 2 respiratory failure. A similar study by Baraa et al showed mean PO2 43.8 for type 1 respiratory failure and 60.06 for type 2 respiratory failure which is comparable with the present study.

The mean PCO2 of the present study on admission was 36.61 for type 1 respiratory failure and 72.78 for type 2 respiratory failure. A similar study by Thomas et al showed mean PCO2of 37 for type 1 respiratory failure and 79 for type 2 respiratory failure on admission. Study by Baraa etal showed mean PCO2 on admission of 36.9 for type 1 respiratory failure and 72.6 for type 2 respiratory failure which is comparable to our study.

The mean pH of the present study on admission was 7.327 for type 1 respiratory failure and 7.268 for type 2 respiratory failure. A similar study by Ige et al showed a mean pH on admission of 7.316for type 1 respiratory failure and 7.231 for type 2 respiratory failure which is comparable to the present study. Another study by Baraa et al showed a mean pH on admission of 7.31 for type 1 respiratory failure and 7.38 for type 2 respiratory failure.

On comparison of physiological parameters at various intervals showed the mean PO2 on admission was 50.68 for type1 and was 53.61 for type 2 respiratory failure, at 1 hour post NIV was 68.38 for type 1 and 74.43 for type 2 respiratory failure, at the time of weaning was 83.6 for type1and113.47 for type2 respiratory failure, post Weaning it was 103.15 for type1 and124.08 for type2 respiratory failure. A similar study by Baraa et al showed a mean PO2 on admission of 43.8 for type1 and 60.06 fortype 2respiratory failure, at 1 hour post NIV it was 50.19 for type 1 and 61.7 for type 2 respiratory failure, at the time of weaning it was 56.7 for typeland 70.3 for type 2 respiratory failure, post weaning it was 65.1 for type 1 and 75.14 for type 2 respiratory failure which is comparable to the present study. Another study by Paolo et al [72] showed a mean PO2on admission of 51.8 for type 1 and 64.6 for type 2 respiratory failure, at 1 hour post NIV it was 70.03 for type 1 and 81.16 for type 2 respiratory failure, at weaning it was 76.06 for type1and 76.2 for type2 respiratory failure.

The mean PCO2 on admission was 36.61 for type 1 and 72.78 for type 2 respiratory failure, at 1 hour post NIV it was 36.77 for type 1 and 60.01 for type 2 respiratory failure, at the time of weaning it was 36.43 for type1 and47.16 for type2 respiratory failure, post weaning it was 36.38 for type 1 and 42.58 for type 2 respiratory failure. A similar study by Baraa et al showed a mean PCO2 on admission of 36.9 for type 1 and 72.6 for type 2 respiratory failure, at 1 hour post NIV it was 34.3 for type 1 and 56.6 for type 2 respiratory failure, at the time of weaning it was 33.2 for type 1 and 53.4 for type 2 respiratory failure, post weaning it was 33.0 for type 1 and 46.3 for type 2 respiratory failure which is comparable to the present study. Another study by Paolo et al showed mean PCO2 on admission of 51.6 for type1 and 69.3 for type2 respiratory failure, at1hour post NIV it was 46.67 for type 1 and 67.64 for type 2 respiratory failure, at the time of weaning it was 47.1 for type 1 and 60.5 for type 2 respiratory failure.

The mean pH on admission was 7.327 for type 1 and 7.268 for type 2 respiratory failure, at 1 hour post NIV it was 7.395 for type 1 and 7.319 for type2 respiratory failure, at the time of weaning it was 7.400 for type 1 and 7.378 for type 2 respiratory failure, post weaning it was 7.400 for type 1 and 7.386 for type 2 respiratory failure. A similar study by Baraa et

al showed a mean pH on admission of 7.38 for type 1 and 7.29 for type 2 respiratory failure, at 1 hour post NIV it was 7.39 for type 1 and 7.35 for type 2 respiratory failure, at the time of weaning it was 7.400 for type 1 and 7.35 for type 2 respiratory failure, post weaning it was 7.39 for type 1 and 7.35 for type 2 respiratory failure which is comparable to the present study. Another study by Paolo et al showed a mean pH on admission of 7.22 for type 1 and 7.28 for type2 respiratory failure, at 1hour post NIV it was 7.38 for type 1 and 7.32 for type 2 respiratory failure, at the time of weaning it was7.400 for type1 and7.38 for type 2 respiratory failure.

The most common cause of NIV failure in our study was Hemodynamic instability in 5 patients (46%) followed by mask intolerance in 2patients (18%)and inability to decrease dyspnoea in2 patients (18%). A similar study by Baraa et al also showed the most common cause of NIV failure was Hemodynamic instability(58%) followed by mask intolerance (16%) and inability to decrease dyspnoea (8%) which is comparable to the present study.

The most common underlying respiratory illness associated with NIV failure were those associated with type 1 respiratory failure like Acute pulmonary oedema seen in 1 patient (50%), Interstitial lung disease seen in two patients (50%), Pneumonia seen in three patients (42%).These are comparable with other studies where type 1 respiratory failure was more likely to be associated with NIV failure. Pneumonia was the most common underlying respiratory illness to be associated with NIV failure in studies by Arnaud et al (54%), Antonelli et al [73] (46%), Baraa et al (53%). The mean duration of NIV usage in successful cases was 82 hours in the present study. In a similar study by Ige et al the mean duration of NIV usage for successful cases was 72 hours which is comparable to the present study.

CONCLUSION:

NIV is a safe and effective for ventilatory support in patients with acute respiratory failure. It is very effective for reversing the physiological abnormalities due to respiratory failure. In our study we studied the clinical application and effectiveness of NIV in patients with type 1 and type 2 respiratory failure. NIV was associated with a significantly reduced need for intubation and mechanical ventilation. It also reduces the mortality and morbidity associated with intubation and mechanical ventilation and also shortens the ICU and hospital stay of patients. This study demonstrated that non invasive ventilation is a promising therapeutic modality that is associated with significant improvement in physiological parameters and clinical outcomes.

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