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Ventilatory Strategy: Useful in Acute Severe Asthma

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Abstract: ***When people talk about bronchial asthma, they are really talking about asthma, A chronic inflammatory disease of the airways that causes periodic "attacks" of coughing, wheezing, shortness of breath, and chest tightness. We hereby, Report a case on Ventilator strategy in 3 different patients of acute severe asthma who were not benefited much by conservative management and found relief by ventilatory support in National Institute Of Medical Science, Jaipur.***

Keywords: Acute severe asthma; Mechanical ventilation

1. Introduction

Bronchial asthma is a common disorder that affects about 300 million people worldwide. Its signs and symptoms however, can be present in other pulmonary and/or airway diseases and therefore a careful workup of patient with respiratory symptoms that might be due to asthma is required. Frequently experience shortness of breath or you hear a whistling or wheezy sound in your chest when you breathe, you may have asthma — a chronic condition that causes inflammation and narrowing of the bronchial tubes, the passageways that allow air to enter and leave the lungs. If people with asthma are exposed to a substance to which they are sensitive or a situation that changes their regular breathing patterns, the symptoms can become more severe..In most of the patients with acute severe asthma intensive therapy with inhaled beta-agonist, systemic corticosteroids and oxygen is usually sufficient enough to improve pulmonary function and ameliorate symptoms. However, despite maximal medical therapy some patients develop life-threatening features and require ventilatory support. Less than five percent of patients admitted with an acute asthmatic attack require intubation and ventilation [1.] Mechanical ventilation in case of status asthmaticus is a life support system associated with substantial morbidity and should be instituted only when it becomes evident that maximal therapy will not be efficacious. To avoid excess morbidity and mortality however, one must be precise in management of these patients. I would like to present our experience with management of three cases of acute severe asthma, who required intubation and mechanical ventilation and were successfully managed.

2. Case Report-1

87-year old lady who was a known case of bronchial asthma for last 45 years was admitted to intensive care unit following acute exacerbation of breathlessness for last 3days. She was on regular treatment with inhaled salbutamol, beclomethasone and oral deriphylline. On admission she was in severe respiratory distress with pulse rate 124/min, blood pressure144/92mm Hg, breath rate 32/min, pulsus paradoxus 15mm Hg and peak expiratory flow rate 102 l/min. Oxygen saturation by pulse oximetry (SpO2) was 89% and blood gases showed pH 7.266, PaCo2 64.1 mm Hg and PaO2 58 mm Hg. She also had low grade fever and cough with thick mucoid expectoration. She was hospitalized more than four times during the last one year, and required parental steroids for control of bronchospasm. Chest auscultation was done which revealed bilateral extensive wheeze with occasional crepitations. Diagnosis of acute severe asthma was made and she was treated aggressively with nebulisation of salbutamol 2.5mg with ipratropium 0.25mg 4 hourly, intravenous methylprednisolone 125mg given 8 hourly, nasaloxygen and antibiotics. After 24 hours of treatment the patient's condition worsened, she became cyanosed and exhausted. Her pulse rate was 146/min, BP-152/100 mm Hg breath rate 34/min, pulsus paradoxus 20 mm Hg and peak expiratory flow rate (PEFR) of 80 l/min. At this time SpO2 had fallen to 70% and arterial blood gases showed pH 7.20, PaCO2 81 mm Hg and PaO2 38.7 mm Hg. Diagnosis of respiratory failure was made and ventilatory support was considered. Patient was anaesthetized with midazolam 2mg, ketamine 80mg and vecuronium 4mg. Trachea was intubated with 8.0 mm cuffed oral endotracheal tube. Patient was placed on Seimensservo 900C ventilator in control mode. Small doses of midazolam and vecuronium were given intermittently to maintain adequate sedation and muscle relaxation. Tidal volume and peak inspiratory flow rate were adjusted to keep plateau pressure below 30cm H2O. Patient was monitored with continuous ECG along with HR, SpO2, hourly NIBP and frequent ABGs. After 24 hours of ventilation patient improved clinically and vital parameters were HR 108/min, breath rate 30/min, BP-140/90 mm Hg and SpO2 96%. On second day vecuronium was stopped and ventilator mode was changed to synchronized intermittent mandatory ventilation (SIMV-8breaths/min) with pressure support of 10 cm H2O and PEEP 5cm H2O. Arterial blood gases showed pH 7.35, PaCO2 64 mmHg, PaO2 134 mm Hg and oxygen saturation 98%.Humidification of inspired gases was done using heated humidifier in inspiratory limb of ventilator circuit. Also frequent instillation of saline 2-4 ml through endotracheal tube, followed by chest physical therapy was used to mobilize secretions and mucus plugs. On third day of continued treatment, patient's condition further improved with HR 92/min, breath rate 22/min and airway secretions were thin and scanty. After four days of ventilation bronchospasm was minimal and arterial blood gases were within acceptable range (pH 7.37, PaCO2 54 mm Hg, PaO2 95mm Hg with FiO2 0.35). Patient was gradually weaned off the ventilator and extubated on fifth day. Subsequently

patient was put on oral medication and made uneventful recovery.

3. Case Report-2

48 year old man, who was a known case of bronchial asthma presented with acute exacerbation of breathlessness. Within few minutes of admission to the intensive care unit patient started deteriorating rapidly. He had profuse sweating, became cyanosed (SpO2 80%) and drowsy. Realizing the criticality, subcutaneous adrenaline 0.3ml of 1:1000 solution was given before securing intravenous line. Simultaneously, salbutamol nebulization and nasal oxygen were started. But patient deteriorated further, became comatose and oxygen saturation dropped to 50%. Patient was immediately intubated with 8.5mm endotracheal tube after injecting ketamine 100mg and vecuronium 4mg. Initially ventilation was carried out using anaesthesia circuit and 100% oxygen. Resistance was felt while doing hand ventilation, once oxygen saturation improved patient was placed on Siemens servo 900C ventilator. Salbutamol nebulization was given 4 hourly using T-piece connection. Methylprednisolone and antibiotics were administered in usual doses. After 24 hours of ventilation patient improved, and there was minimal bronchospasm. Patient was extubated and recovered uneventfully.

4. Case Report-3

60 year old woman was admitted to intensive care unit with sudden onset of breathlessness. He was diagnosed as a case of acute severe asthma. Appropriate treatment was started with salbutamol nebulization and intravenous corticosteroids. As there was no improvement clinically salbutamol nebulization in 100% oxygen was repeated every30 minutes. After 2 hours of aggressive therapy patient deteriorated further, became cyanosed and obtunded. At this point mechanical ventilation was considered; patient was intubated and connected to T-bird ventilator on SIMV mode. Tidal volume was adjusted to keep peak airway pressure less than 50 cm of H2O. Within 24 hours of ventilation patient improved significantly, bronchospasm was minimal and airway pressures had come down to 30cm of H2O. Weaning from mechanical ventilation started by gradually reducing SIMV controlled breath rate. After 48 hours of ventilation, patient was extubated and had an uneventful recovery.

5. Discussion

As observed Acute severe asthma that does not respond to intensive bronchodilator therapy can result in fatality, if timely intervention is not undertaken. Clinicians also feel reluctant to intubate and ventilate because of lack of expertise. We have observed that acute asthmatic exacerbation that requires intubation follows one of two basic patterns. Most often, acute deterioration follows a gradual worsening of airflow obstruction over 2-3 days due to fatigue of respiratory muscles (case 1). A second presentation is characterized by an explosive onset of asthma, causing hypoxia and restlessness (case 2 and3). Molfino et al had similar observations [2].The decision to proceed with intubation and ventilation is best based on the integrative clinical assessment of the patient's ability to continue ventilation till medical therapy becomes effective. The features of life threatening asthma include impending respiratory muscle fatigue, depressed mental state, central cyanosis, bradycardia, silent chest, PEFR < 100 l/min or unrecordable and increasing hypercapnia or respiratory acidosis (pH<7.2). Patients having life threatening features require intubation and mechanical ventilation. Oral intubation is preferred because it allows for placement of a large endotracheal tube, which decreases airway resistance and facilitates removal of tenacious mucus plugs. To achieve sedation and relaxation before intubation, midazolam, ketamine and vecuronium may be used. Ketamine being a good bronchodilator helps ineffective ventilation and reduction of airway pressures. Muscle relaxants like vecuronium should not be used for more than 24 to 48 hrs. If used over several days with high dose of corticosteroids, muscle relaxants appear to contribute to the development of an acute, diffuse myopathy [3]. The resulting muscle weakness can be profound and recovery may require many months of rehabilitation and even then may be incomplete. Until 1990, barotrauma was a common complication of the high pressures required to maintain normal alveolar ventilation, resulting in high mortality. The recently introduced concept of 'permissive hypercapnia' is responsible for the sharp reduction in mortality. The ventilator is set to deliver small tidal volume (6 to 8 ml/kg) at low frequency of 10 to 12 breaths/min to keep peak end-inspiratory plateau pressure under 30 cm H2O. This regime is continued as long as necessary provided oxygenation can be maintained and PaCO2 does not rise above 90 mm Hg. Severe limitation of expiratory flow in these patients when matched with an inadequate exhalation time can result in dynamic hyperinflation, also known as auto positive end expiratory pressure (auto-PEEP). The adverse cardiopulmonary effects of this hyperinflation include hypotension, hypercapnia, weaning failure and in extreme cases, cardiac arrest with electromechanical dissociation [4]. An approach to avoid lung hyperinflation is to minimize inspiratory time by setting peak inspiratory flow rates of 80 to 100 l/min, thereby prolonging the time permitted for exhalation and lung emptying. Permissive hypercapnia is associated with respiratory acidosis and an intense drive to breathe. The pH disturbance itself appears to be tolerated well by most patients. If the systemic pH remains above 7.15, the acidosis that accompanies need not be corrected [5] .Low levels of PEEP, in amounts just adequate to match the patient's intrinsic PEEP, are of value in aiding patient ventilatory synchrony during weaning[6]. Frequent aerosol administration and chest physical therapy is required to mobilize mucus plugs; even therapeutic bronchoscopy with lavage has been used as an additional supportive measure[7]. In our institute we use frequent instillation of 2-4 ml of saline through endotracheal tube and chest physical therapy to mobilize mucus plugs. The duration of mechanical ventilatory support in acute severe asthma is highly variable, ranging from a few hours to a week or more. In patients with gradual worsening of symptoms, who have prominent airway edema and mucus plugging, typically respond slowly and require 3 to 5 days of ventilatory support. On the other hand patients, who present with sudden asphyxial asthma due to pure bronchospasm, may rapidly respond to bronchodilators, allowing successful extubation within a few hours of intubation.

6. Conclusion

Bronchial asthma, although one of the most common respiratory diseases, can be minicked by a number of other pulmonary and airway diseases, and especially patients with so called severe or treatment refractory asthma should receive a detailed diagnostic workup with a rather broad differential diagnosis. Among our patients, the first patient had gradual worsening of symptoms and required 5 days of ventilatory support. The other two patients had sudden bronchospasm and required shorter duration of ventilatory support. Status asthmaticus severe enough to warrant admission to the ICU marks the patient as being in the high-risk group for recurrent admission and possibly even death due to asthma. Counselling and aggressive outpatient management needs to be carefully arranged to avoid multiple episodes of severe asthma.

References

[1] Braman SS, Kaemmerlen JT. Intensive care of status asthmaticus: A 10-year experience. JAMA 1990; 264:366-8.
[2] Molfino NA, Nannini LJ, Martelli AN, Slutsky AS. Respiratory arrest in near fatal asthma. N Engl J Med 1991; 324:285-8.
[3] Hansen-Flaschen J, Cowes J, Raps EC. Neuromuscular blockade in the intensive care unit: More than we bargained for. Am Rev Respir Dis 1993; 147:234-6.
[4] Rosergarten PL, Tuxen DV, Dziukas L, Scheinkestel C, Merret K, Bowes G. Circulatory arrest induced by intermittent positive pressure ventilation in a patient with severe asthma. Anaesth Intensive Care 1991; 19:118-21.
[5] Feil F, Perret C. Permissive hypercapnia. Am J Resp Crit Care Med 1994; 150:1722-37.
[6] Tan TK, Bhatt SB, Tam YH, Oh TE. Effects of PEEP on dynamic hyperinflation in patients with airflow limitation. Br J Anaesth 1993; 70:267-72.
[7] Smith DL, Deshazo RD. Bronchoalveolar lavage in asthma. Am Rev Respir Dis 1993; 148:523-7.