Case Report

Early tracheotomy for acute severe asthma

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BACKGROUND: Few studies have reported the effects of early tracheotomy in acute severe asthmatic patients. We report two patients with acute severe asthma who were successfully treated with early tracheotomy.

METHODS: The two patients with acute severe asthma were retrospectively reviewed. They had been treated at the Department of Emergency and Critical Care, Renji Hospital, Shanghai Jiaotong University School of Medicine.

RESULTS: They developed progressively hypercapnia and severe acidosis, and were not improved after conventional therapies. Early tracheotomy after mechanical ventilation decreased airway resistance and work of breathing, and corrected hypercapnia and acidosis. Adequate gas exchange was maintained after tracheotomy. The two patients were subsequently weaned from mechanical ventilation and discharged.

CONCLUSION: Early tracheotomy could be a valuable approach in certain patients with severe asthma.

KEY WORDS: Early tracheotomy; Acute severe asthma; Extubation; Mechanical ventilation; Work of breathing

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INTRODUCTION

Acute severe asthma (status asthmaticus) is a life-threatening emergency because of refractory respiratory failure. Patients are severely dyspneic, cyanosed, and often moribund. Although there appears to be a trend toward increased survival after mechanical ventilation for patients with acute asthma, ventilation of critically ill asthmatic patients continues to be a potentially perilous venture associated with significant morbidity and mortality.[1] Tracheotomy is often performed when patients fail to wean from ventilation. As reported by Lee et al,[2] patients with early tracheotomy compared with those with delayed tracheotomy showed significantly lower hospital mortality (31.7% vs. 61.7%, $P<0.005$), pneumonia (5% vs. 25%, $P<0.005$), and accidental extubation (0% vs. 10%, $P=0.03$). Patients with early asthma spent less time in the intensive care unit (4.8 vs. 16.2 days, $P<0.001$) and on mechanical ventilation (7.6 vs. 17.4 days, $P<0001$) and there was less damage to the mouth and larynx, but not the trachea. The mean duration of mechanical ventilation in patients with severe asthma was about 3 days,[3] but few studies reported the effects of early tracheotomy under this specific condition. Herein, we report two patients with acute severe asthma who were successfully treated with early tracheotomy.

Case reports

Case 1. A 50-year-old man with asthma was admitted to the emergency department of our hospital because of sudden onset of dyspnea, cough and chest tightness. On arrival he complained of severe shortness of breath and was only able to speak with short sentences. Examination revealed widespread wheeze and accessory muscle breathing. Chest radiograph showed bilateral infiltration. The results of labortory examination including biochemistry analysis of blood were nothing abnormal.

Initial arterial blood gas (ABG) was pH 7.11, PaO_2/FIO_2 151.6 mmHg, and $PaCO_2$ 101.0 mmHg. The patient was transferred to ICU and administered with repeated nebulized bronchodilators, high-dose of corticosteroids, antibiotics, and noninvasive ventilation, but he was not improved. At 7 hours after admission, arterial blood gas analysis revealed worsening of respiratory acidosis and hypercapnia (pH 7.1, $PaCO_2$ 109 mmHg, PaO_2 58 mmHg), indicating the need of tracheal intubation. Combined with sedation and analgesia, neuromuscular blocking agents, mechanical ventilation was performed with a low respiratory rate, prolonged expiratory time, and low extrinsic positive end-expiratory pressure. However, adequate oxygenation and ventilation were not achieved with poor compliance and high resistance. After 48 hours of ventilation, the airway pressure of the patient remained high (Plateau airway pressure>40 cmH_2O) and tidal volumes<300 mL. $PaCO_2$ remained 85 mmHg with a corresponding pH value of 7.20. Tracheotomy was performed thereafter. One day after tracheotomy, the plateau airway pressure decreased (25 cmH_2O), tidal volume increased (450 mL) gradually with further stabilization of other vital signs. A third ABG analysis revealed improvement of hypercarbia and acidosis ($PaCO_2$ 66.8 mmHg, pH 7.39). He was extubated on the 7th day and discharged 17 days later.

Case 2. A 24-year-old man with asthma was admitted to ICU for sudden onset of dyspnea and tightness. Physical examination revealed widespread wheeze and subcutaneous emphysema on the face, neck, shoulders and upper part of the chest. Chest radiograph showed extensive subcutaneous emphysema and pneumomediastinum. Arterial blood gas analysis revealed acute hypercapnic respiratory failure (pH 7.329, PCO_2 57.1 mmHg, PO_2 55.2 mmHg). He was initially treated with conventional medication, but failed. At 20 hours after admission, repeated chest radiograph revealed pneumomediastinum with left pneumatothorax. A second arterial blood gas analysis revealed increased hypercapnia (pH 7.183, $PaCO_2$ 98.2 mmHg, PaO_2 51.2 mmHg). He was intubated, and closed drainage was performed on the left pneumatothorax. Despite multiple ventilatory mode changes, maximal bronchodilator therapy, and antibiotic treatment, PaO_2 was still lower than 60 mmHg. At 72 hours after intubation, $PaCO_2$ rose to 73.7 mmHg, airway pressure remained high, and tidal volumes were unacceptably low. Then tracheotomy was performed. At 48 hours after tracheotomy, the plateau airway pressure decreased (23 cmH_2O), tidal volume increased (440 mL), and $PaCO_2$ value decreased accordingly ($PaCO_2$ 62.3 mmHg, pH 7.358). He was extubated on the 10th day and discharged from the hospital 18 days later.

DISCUSSION

The causes of death from severe asthma include arrhythmia, hypoxemia secondary to ventilation/perfusion (V/Q) mismatch, and alveolar hypoventilation. The major physiologic changes associated with severe asthma are airflow limitation, bronchial hyperresponsiveness, airway closure, loss of elastic recoil, and hyperinflation (or air trapping). Optimal treatment includes nebulized or intravenous beta-2 agonists, nebulized anticholinergics, intravenous corticosteroids, and sometimes intravenous aminophylline and magnesium sulphate. Mechanical ventilation is an effective measure to correct hypoxemia and hypercapnea. Once the patient was intubated, primary focus must be on avoiding excessive airway pressure and minimizing lung hyperinflation. To achieve this goal it is often necessary to hypoventilate the patient and thus to tolerate hypercapnia, which is termed "controlled hypoventilation" or "permissive hypercapnia". Despite progress in mechanical ventilation and newer drug therapies, there remains a significant mortality and morbidity in severe cases.[4]

Among those refractory patients, additional methods have been attempted such as buffer therapy with bicarbonate or amino alcohol tromethamine, inhaled anesthesia, airway clearance with bronchoscopy and mucolytics, helium-oxygen mixture (heliox), and extracorporeal membrane oxygenation. However, the efficacy of the aforementioned modalities has not been confirmed.

In our study, the two patients were irresponsive to all conventional therapies until they gained significant improvement at 48 hours after tracheostomy. It seems that tracheostomy can minimize airway hypersensitivity due to endotracheal tubes, and improve secretion drainage that prevents sputum plug. Several studies[2-4] have focused on the benefit of early tracheostomy in intubated patients. Jaeger et al[5] suggested the larger the diameter and the shorter the tube, the lower the resistance. On the other hand, irregular tube walls (with secretions) and the sharper any curve in the tube will present greater resistance. After measuring the internal volume (dead space) of standard endotracheal tubes and tracheostomy tubes, Davis et al[6] found there were a difference of<20 mL and no significant effect on

spontaneous tidal volume. Why tracheostomy might benefit a patient in terms of weaning is reduced work of breathing (WOB) due to decreased gas flow resistance in a tracheostomy tube and improvement in secretion drainage. These investigators also found intrinsic positive end-expiratory pressure (PEEPi) was slightly (mean difference 1.3 cmH_2O) but significantly lower after tracheotomy.[6] In addition, Diehl et al[7] found resistive and elastic work as well as PEEPi and ventilatory drive were significantly reduced as measured by airway occlusion pressure. Compared with delayed tracheotomy in ICU patients who needed a prolonged mechanical ventilation, satisfactory outcomes (including duration of mechanical ventilation, mortality in hospital, incidence of ventilator-associated pneumonia) were found in the patients undergoing early tracheotomy.[8]

We consider that if Pplat under 30 cmH_2O or pH above 7.2 can not be maintained (despite the use of deep sedation/paralysis), the ventilated asthmatic patient must be examined for causes of decreased respiratory system compliance. If there are no such causes, tracheotomy must be performed within 72 hours.

Early tracheotomy is rarely described as a therapy for patients with life-threatening asthma. Our data suggest that early tracheotomy may be beneficial for asthmatic patients resistant to conventional therapy including mechanical ventilation. But additional evidence especially randomized controlled trial is needed in order to guide decision-making in clinical practice.

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