Bronchoscopy as a rescue therapy in patients with status asthmaticus: Two case reports and review of literature

Muhammad Faisal Khan, Hadil A. Al Otair, Alaa F. Elgishy, Abdulaziz Hamad Alzeer

Department of Critical Care, King Khalid University Hospital, King Saud University, Riyadh, Saudi Arabia

Address for correspondence:
Dr. Muhammad Faisal Khan, Department of Critical Care, 95, 3rd Level Critical Care Building, King Khalid University Hospital, P.O. BOX: 2925, Riyadh 11461, Saudi Arabia.
E-mail: mfkdr.icu@gmail.com

ABSTRACT

Management of status asthmaticus requires more than guidelines-guided therapy. Occasionally, uncommon therapeutic interventions and procedures may provide significant benefits. The role of bronchoscopy in fatal asthma is still not clearly defined. We illustrate two cases of severe status asthmaticus on mechanical ventilation who showed remarkable improvement after therapeutic bronchoscopy.

Key words: *Mechanical ventilation, status asthmaticus, therapeutic bronchoscopy*

INTRODUCTION

Mortality of severe asthmatic attack ranges from 1 to 10%.[1] The management of life-threatening asthma is an immense challenge for the Intensivist, especially with those patients whose course is complicated with severe bronchospasm leading to air trapping, respiratory fatigue, and requiring ventilatory support. Numerous guidelines are available for managing asthma; however, standardized guidelines are lacking for the management of status asthmaticus which is resistant to conventional therapies and requiring mechanical ventilation.[2,3] Several factors are responsible for status asthmaticus such as resistant bronchospasm and altered viscoelastic properties of mucus leading to airway plugging with thick mucus secretions.[4] The role of bronchoscopy in these cases is obscure and not well documented in the literature.

Therefore, we report two cases of severe status asthmaticus on mechanical ventilation who showed marked improvement after therapeutic bronchoscopy.

Access this article online	
Quick Response Code:	Website: www.saudija.org
	DOI: 10.4103/1658-354X.115323

CASE REPORTS

Case 1

A 43-year-old woman, known to have asthma for 5 years, is presented to the Emergency Department (ED) in a gasping state. Her clinical assessments include: heart rate, 140 bpm; blood pressure, 70/42 mmHg; respiratory rate, 35 per min; and SpO_2 78% on plethysmography. Initial resuscitation was done with intubation and fluid management. Her initial arterial blood gases showed severe respiratory and metabolic acidosis (pH 6.90; $PaCO_2$ 114 mmHg; PaO_2 30.0 mmHg; and HCO_3 13.4 mEq/L). After securing the airway, the clinical exam revealed, tachycardia, unstable hemodynamics, peripherally mottled skin, and silent chest with no air entry. The case was diagnosed as a severe life-threatening asthma leading to type II respiratory failure and shock. Initial laboratory exams showed hemoglobin, 13.4 g/dL; hematocrit 40.8%; leukocyte count 15.7×10^9/L and platelet 225×10^9/L. Chest X-ray revealed hyper-inflated lung fields. The patient was sedated with ketamine and relaxed with rocuronium infusion. She was placed on mechanical ventilation (Draeger C500) in assist control mode, respiratory rate 12 per min; tidal volume 7 mL/kg (according to ideal body weight); inspiratory flow rate 70 L/min; Positive end expiratory pressure (PEEP) 5 cm H_2O; and 1.0 FiO_2. Lung

mechanics revealed high-peak airway and plateau pressure (80 and 30 cm H_2O respectively). The Intrinsic-PEEP (PEEPi) measured at that time was non-significant. The treatment was started with bronchodilators (salbutamol and ipratropium), inhaled and intravenous corticosteroids, and bolus of magnesium sulfate (2 g).

Subsequently, the patient was treated with salbutamol infusion and intermittent doses of 0.5 mL (1:1000) subcutaneous adrenaline. Despite being on maximum support, the patient showed no significant improvement. Lung mechanics still displayed high peak and plateau pressures (75 and 35 cm H_2O respectively). Bicarbonate infusion was started to maintain the pH near normal. The patient continued to have mixed acidosis with pH 7.01; PCO_2, 71.8; PaO_2 87; and HCO_3 13.4. Urgent flexible bronchoscopy was performed for diagnostic purposes and removal of mucus plug as warranted. Bronchoscopy revealed hyperemic airways, purulent secretions in central airways. Lavage was done and mucus plugs were removed. The process of bronchoscopy was unremarkable.

Immediately after bronchoscopy, the patient's respiratory condition began to improve. Acidemia improved from 7.01 to 7.24, PCO_2 decreased from 72 to 32 mmHg and PaO_2 improved from 87.2 to 186 mmHg [Graph 1]. FiO2 was also reduced from 1.0 to 0.4. Peak and plateau airway pressures decreased to 25 and 18 cm H_2O respectively. Patient's severe bronchospasm had remarkably improved.

At the same time, the patient was also treated for sepsis. The presence of Gram-positive cocci in blood was noted in gram stains and vancomycin was started. Hematological assessment showed elevated leukocyte (21.2×10^9/L), low platelet (98×10^9/L) count, and normal hemoglobin.

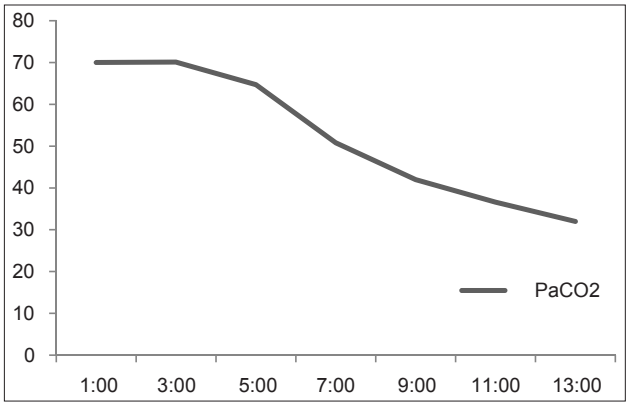
Furthermore, patient's sepsis worsened. Patient was resuscitated with fluids, vasopressor, and broad spectrum antibiotics. Despite improvement in respiratory status her course was complicated with septic shock and multi-organ failure. Patient expired after 5 days of admission.

Case 2

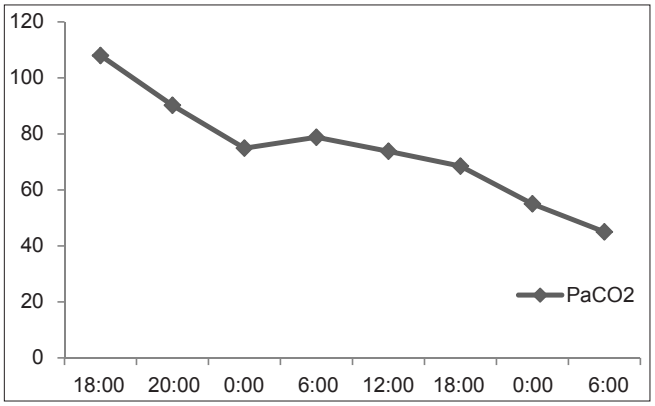
A 36-year-old male, who was asthmatic since childhood, presented in the ED with shortness of breath for 1 week. The patient also had a history of yellowish sputum with fever for 1 week. Clinical exam revealed bilateral wheeze with prolonged expiration throughout the chest and a roentgenogram showed hyper-inflated lungs and right lower lobe infiltrate. He was intubated in the ED in response to worsening respiratory failure. The initial diagnosis was exacerbation of asthma and community acquired pneumonia. Blood gases before (pH 7.01; $PaCO_2$ 108; PaO_2, 106 HCO_3 27.8) and after the intubation showed acute respiratory acidosis (pH 7.16; $PaCO_2$ 90.2; PaO_2 110; HCO_3 23.7). Initial treatment included inhaled bronchodilators, inhaled and intravenous steroids, antibiotics (ceftriaxone and azithromycin), and a bolus dose of magnesium sulfate. Patient was placed on mechanical ventilation (Draeger infinity 500) with settings of assist control, rate 12 per min; tidal volume 6-7 mL/kg; PEEP 05 cm H_2O; inspiratory flow rate 70 L/min and FiO_2 1.0. Lung mechanics revealed high peak and plateau pressures of 70 and 28 cm H_2O respectively, whereas intrinsic PEEP ($PEEP_i$) was measured 12 cm H_2O. Inspiratory flow rate was increased to 80 L/min to reduce the $PEEP_i$.

Intravenous infusion of salbutamol and intermittent subcutaneous adrenaline 0.5 mL (1:1000) was started due to progressive worsening of clinical condition. $PEEP_i$ and peak airway pressure improved from 12 to 4 cm H_2O and 70 to 56 cm H_2O respectively. Arterial blood gases showed little improvement (pH 7.26; $PaCO_2$ 74.9; and PaO_2 146). On 3rd day of hospital admission bronchoscopy was planned for bronchial toilet. It revealed inflamed airways and obstruction of middle-sized airways with mucus plugs [Figure 1]. Effective suction and lavage were done.

The patient's clinical condition and ventilator requirement were reduced and ABG was improved [Graph 2] after therapeutic bronchoscopy. The patient was successfully



Graph 1: Change in $PaCO_2$ during 12 h period in case 1



Graph 2: Change in $PaCO_2$ during 12 h period in case 2

Figure 1: Mucus plug removed after bronchoscopy in case 2 patient

weaned on the following day. Patient was discharged from intensive care 48 h after extubation.

DISCUSSION

Asthma is prevalent in the adult population with a broad clinical spectrum ranging from mild to fatal asthma.[5] The management of severe cases of asthma may demand more than guidelines-guided therapy and use of the innovative therapy that is not the part of routine clinical practice. These two cases mentioned in this case report clearly highlight the potential role of bronchoscopy in resistant cases of status asthmaticus.

Asthma is a reversible obstructive airway disease characterized by a triad of bronchial smooth muscle contractions, airway inflammation, and increased secretion.[5] Acute severe asthma is a life-threatening condition that can cripple the whole respiratory process. The mortality rate of patients admitted in ICU with status asthmaticus was noted approximately 8.3%.[6] The clinical course of severity in symptoms follows the sequence of air flow limitation leading to ventilation perfusion mismatch, hypoxemia, hypercarbia, and respiratory failure. Risk factors that can contribute significantly include infections and severe mucus plugging; these may lead to severe air flow obstruction, atelectasis, and air way inflammation. It has already been observed in a study[7] that the necropsic finding of patients who died after fatal asthmatic attack revealed blockade of airway; this blockade was primarily due to thick mucus plugs that lead to airway segments collapse. The cause of the death in this study was owing to severe air flow limitation, generation of intrinsic PEEP, hypoxemia, and then respiratory failure and shock.

It is now more evident that the pathophysiology of fatal asthma depends on the hypothesis of luminal obstruction leading to life-threatening attack of asthma. This could be due to cumulative effects of the blending of cellular contents, mucus, and protein exudates.[8] Kuyper *et al.* investigated the characteristic of mucus plugging in fatal asthma patients and it was observed that cellular content was a mixture of migratory inflammatory cells and shed epithelial cells. The cellular content with mucus exudates could initiate inflammation in airways and epithelial disruption. The higher the epithelial disruption, the greater the risk of mucus retention and impairment of mucociliary clearance and increase in sputum viscosity.[9] This thick and tenacious secretion can adhere to the bronchi that resist airflow and also produce turbulent air movement in airways. The worsening of this mechanism not only increases work of breathing but also interferes with gas exchange and increase chance of infection.[9] This vicious cycle can interfere with inhaled drug delivery and slows the pace of weaning from ventilator. Thus, the role of removal of this mucus impaction could be a life-saving.

Early reports highlighted the successful use of bronchoscopy in patients with severe asthma who were on mechanical ventilation.[10,11] Removal of mucus plug and broncho-alveolar lavage helped in improvement of PCO_2 and expedites ventilator withdrawal. A recent report of 41 pediatric patients with status asthmaticus showed improvement in ventilator weaning, reduced time of ventilation, and decreased length of ICU stay after performing therapeutic bronchoscopy.[12]

Flexible bronchoscopy is routinely performed in recent days in ICU. The presence of thick impacted sputum in central as well as small airways makes its use more promising.

These two cases illustrate that when standard therapies fail, flexible bronchoscopy could have significant role in evaluation of central airways and removal of mucus plug. These cases were initially managed with bronchodilators, steroids, antibiotics, and mechanical ventilation. Bronchoscopy was done to rule out other causes of severe airflow limitation including mucus plug and foreign body. In case 1, airways were inflamed, thick and tenacious secretions were successfully removed. In case 2, bronchoscopy also revealed thick mucus plugs and inflamed airway walls. These two cases prudently describe the cause of ventilatory failure because after successful liberation of thick sputum, airway compliance improved and bronchospasm was relieved.

Despite positive reports of the benefits of bronchoscopy and lavage in severe asthmaticus, its use continues to be documented only in case reports[4,8,9] and some observational studies.[9] Therefore, concrete evidence

supporting the effectiveness of this potentially beneficial therapy is still awaited.

CONCLUSION

In conclusion, these two cases support that bronchoscopy may facilitate respiratory recovery in patients with resistant status asthmaticus who are on mechanical ventilation. Robust studies are warranted to explore the potential role of bronchoscopy and lavage in these patients.

REFERENCES

1. Papiris S, Kotanidou A, Malagari K, Roussos C. Clinical review: Severe asthma. Crit Care 2002;6:30-44.
2. British Guideline on the Management of Asthma. British Thoracic Society Scottish Intercollegiate Guidelines Network. Thorax 2008;63:iv1-121.
3. Kazani S, Israel E. Update in asthma 2010. Am J Respir Crit Care Med 2011;184:291-6.
4. Durward A, Forte V, Shemie SD. Resolution of mucus plugging and atelectasis after intratracheal rhDNase therapy in a mechanically ventilated child with refractory status asthmaticus. Crit Care Med 2000;28:560-2.
5. Expert Panel Report 3 (EPR-3). Guidelines for the Diagnosis and Management of Asthma-Summary Report 2007. National Asthma Education and Prevention Program. J Allergy Clin Immunol 2007;120:S94-138.
6. Afessa B, Morales I, Cury JD. Clinical course and outcome of patients admitted to an ICU for status asthmaticus. Chest 2001;120:1616-21.
7. Houston JC, De Navasquez S, Trounce JR. A clinical and pathological study of fatal cases of status asthmaticus. Thorax 1953;8:207-13.
8. Zamanian M, Marini JJ. Pressure-flow signatures of central-airway mucus plugging. Crit Care Med 2006;34:223-6.
9. Kuyper LM, Paré PD, Hogg JC, Lambert RK, Ionescu D, Woods R, *et al*. Characterization of airway plugging in fatal asthma. Am J Med 2003;115:6-11.
10. Thompson HT, Pryor WJ, Hill J. Bronchial lavage in the treatment of obstructive lung disease. Thorax 1966;21:557-9.
11. Walker PE, Marshall M. Bronchial lavage in status asthmaticus. Br Med J 1969;3:31-2.
12. Maggi JC, Nussbaum E, Babbitt C, Maggi FE, Randhawa I. Pediatric fiberoptic bronchoscopy as adjunctive therapy in acute asthma with respiratory failure. Pediatr Pulmonol 2012;47:1180-4.

How to cite this article: Khan MF, Al Otair HA, Elgishy AF, Alzeer AH. Bronchoscopy as a rescue therapy in patients with status asthmaticus: Two case reports and review of literature. Saudi J Anaesth 2013;7:327-30.
Source of Support: Nil, **Conflict of Interest:** None declared.