

Noninvasive Ventilation in Severe Acute Asthma

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Noninvasive ventilation (NIV) in severe acute asthma is controversial but may benefit this population by preventing intubation. We report on a 35-year-old male asthma patient who presented to our emergency department via emergency medical services. The patient was responsive, diaphoretic, and breathing at 35 breaths/min on 100% oxygen with bag-mask assistance, with S_{pO_2} 88%, heart rate 110–120 beats/min, blood pressure 220/110 mm Hg, and temperature 35.8°C. NIV at 12/5 cm H_2O and F_{IO_2} 0.40 was applied, and albuterol at 40 mg/h was initiated. Admission arterial blood gas revealed a pH of 6.95, P_{aCO_2} 126 mm Hg, and P_{aO_2} 316 mm Hg. After 90 min of therapy, P_{aCO_2} was 63 mm Hg. Improvement continued, and NIV was stopped 4 h following presentation. NIV tolerance was supported with low doses of lorazepam. The patient was transferred to the ICU, moved to general care the next morning, and discharged 3 days later. We attribute our success to close monitoring in a critical care setting and the titration of lorazepam. Key words: noninvasive; ventilation; asthma; ARF; continuous albuterol; capnography; mechanical ventilation; intubation; sedation. [Respir Care 2014;59(10):e149–e152. © 2014 Daedalus Enterprises]

Introduction

The use of noninvasive ventilation (NIV) in acute respiratory failure (ARF) has been well established in preventing intubation for COPD, congestive heart failure, and immunocompromised patients.¹ The successful application for these populations can be attributed to the relatively rapid reversibility of the underlying cause of the distress. Other increasing applications of NIV include pneumonia, weaning from mechanical ventilation, and acute-on-chronic respiratory failure. The application for acute severe asthma does not have the same degree of support. A 2012 Cochrane review on the use of NIV for severe asthma reports some promising preliminary results, but the use of NIV in this population still remains controversial due to a scarcity of

larger controlled trials.² Acute severe asthma is characterized by severe air-flow limitations due to excessive secretions, airway inflammation, and bronchospasm. Definitive therapy includes bronchodilators, corticosteroids, and supportive care. These patients can present severely hypercarbic, hypoxemic, and acidotic. Supportive care has included endotracheal intubation and positive-pressure ventilation to reverse the hypercarbia-associated acidosis, correct hypoxemia, and provide rest for the respiratory musculature. These therapies are associated with risk. Endotracheal intubation in the critically ill patient requires the risk of sedatives and/or paralytics, and the intubation procedure itself carries risk.³ The subsequent application of mechanical ventilation to the asthma patient requires ongoing sedation and is associated with its own relative difficulties.

We applied NIV to an acute severe asthma patient to provide the required ventilatory assistance without exposing the patient to the risks of endotracheal intubation and invasive mechanical ventilation. The environment of care was an academic medical center emergency department that included continuous cardiac monitoring, capnography, ventilator graphics, one-to-one nursing, a registered respiratory therapist, and a physician. NIV was provided via a Vela ventilator (CareFusion, San Diego, California) in the NIV mode and a full face mask (ResMed, San Diego, California).

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Case Report

On day 1, a 35-y-old male with a well established history of asthma, with no prior intubations for exacerbations, and well known to our facility developed shortness of breath while at home getting out of the shower. The patient had attempted to self-medicate but was unable to get relief and activated emergency medical services for an asthma exacerbation. On arrival, emergency medical services found the patient in respiratory distress with diffuse wheezes; treated the patient with 3 standard doses of albuterol, one standard dose of ipratropium bromide nebulizer, 125 mg of intravenous Solu-Medrol, 0.3 mg of subcutaneous epinephrine, and oxygen; and transported him to our emergency department for an acute severe asthma exacerbation. On presentation, the patient was a well developed male and diaphoretic, with a breathing frequency of 35 breaths/min. He was unable to speak but would nod “yes” and “no” to verbal questioning. Initial pulse oximetry was 88% with bag-mask assistance, with F_{IO_2} 1.0. The patient had an intravenous line, and initial vital signs included a heart rate of 110–120 beats/min, a blood pressure of 220/110 mm Hg, and a temperature of 35.8°C. His pulmonary exam was remarkable for severe respiratory distress, intercostal retractions, and faint wheezes throughout, with little air exchange appreciated. The home medication list included only those for asthma.

Blood laboratory samples were immediately drawn on arrival and included an arterial blood gas (ABG), and the patient was placed on NIV: pressure support 5 cm H_2O , PEEP 5 cm H_2O , F_{IO_2} 0.40, and noninvasive breath mode via an oronasal mask. The settings were titrated for patient tolerance, targeted exhaled tidal volumes (V_T), and $S_{pO_2} > 92\%$. The pressure support level was set at 12 cm H_2O to achieve targeted V_T values (4–8 mL/kg), and PEEP 5 cm H_2O optimized comfort and synchrony. Total delivered inspiratory pressure was 17 cm H_2O . Initial medications included 4 g of intravenous magnesium, 0.5 mg of subcutaneous epinephrine, and continuous nebulized albuterol at 40 mg/h. A chest x-ray was obtained. The patient was also given antibiotics prophylactically for potential community-acquired pneumonia. Within minutes of therapy, the patient appeared more comfortable, his expiratory time began increasing, and his capnographic curve demonstrated improved alveolar ventilation. The systolic blood pressure was > 220 mm Hg and was treated effectively with a nitroglycerin drip. ABG obtained on arrival prior to NIV during bag-valve-mask assistance on 100% oxygen revealed a pH of 6.95, P_{aCO_2} 126 mm Hg, and P_{aO_2} 316 mm Hg. The chest x-ray (Fig. 1) revealed no focal infiltrates or pneumothoraces, well aerated lung fields, and a hyperinflated chest. The complete blood count and electrocardiogram were unremarkable. Despite the severe acidosis, the patient appeared more comfortable on NIV, and support was continued with close monitoring that included serial ABG



Fig. 1. Anteroposterior chest radiograph taken at admission.

Table 1. Timeline of Arterial Blood Gas Analysis

Time	pH	P_{aCO_2} (mm Hg)	P_{aO_2} (mm Hg)	Support
14:40	6.95	126	316	NIV
15:14	7.06	96	205	NIV
15:40	7.18	63	190	NIV
16:45	7.30	44	179	NIV
17:56	7.32	40	139	NC
18:56	7.34	40	131	NC

NIV = noninvasive ventilation
NC = nasal cannula

analysis, continuous capnography, and cardiac monitoring for signs of deterioration. The continued improvement was evidenced by a decreasing breathing frequency, improved expiratory waveforms, and an increasing expiratory time. A second ABG revealed an improving acidosis with a pH of 7.06 and significantly improved ventilation (P_{aCO_2} 96 mm Hg). As the patient’s mental status improved, his tolerance for NIV diminished, and periodic lorazepam was used in 0.5-mg aliquots. While awaiting a bed in the medical ICU, the patient continued to improve. Pressure support levels were weaned to maintain exhaled V_T within target range. The patient’s ABG was essentially normal at 2 h post-arrival, with a pH of 7.30 and P_{aCO_2} 44 mm Hg on NIV. The systolic blood pressure had been normalized, and he was able to be weaned from the nitroglycerin drip. NIV was discontinued, and the patient was placed on a nasal cannula of 4 L/min at 2.5 h after presenting to the emergency department. He was subsequently transferred to the ICU, where he was observed overnight and then transferred to a general floor on day 2. He was discharged 3 days after his initial presentation. Table 1 shows the timeline of ABG analysis in the emergency department.

Discussion

The application of NIV in supporting ARF has become widely accepted for COPD, congestive heart failure, and immunocompromised patients in acute respiratory distress¹ but remains controversial for severe acute asthma.² The immediate use of NIV in our case was not solely for the ventilatory support of the asthma exacerbation. Delay et al⁴ and Baillard et al⁵ have demonstrated improvements in pre-oxygenation for endotracheal intubation with NIV. Use of NIV in this setting may decrease the relative risks and complications associated with orotracheal intubation of a patient in extremis by optimizing oxygenation and ventilation. The application of NIV for asthma support alone is not without precedent. Fernández et al⁶ performed a 7-year observational review and concluded that NIV appears to be a suitable method to improve ventilation in a subgroup of asthma patients. In 2009, Hess et al⁷ performed a survey of emergency department physician use of NIV, and of the 89% who responded, NIV had been used for asthma (although < 10% reported its usage). In a review of NIV in asthma, Soroksky et al⁸ reported that benefits include bronchodilation, offset of intrinsic PEEP, recruitment of collapsed alveoli, an improved ventilation/perfusion relationship, and reduction in the work of breathing. The authors also stated that a cautious trial of NIV may be applied in a severe asthma attack.⁸ A retrospective cohort study by Murase et al⁹ in 2010 of severe asthma treated with NIV reported a trend toward a decrease in intubation rates and concluded that the ready availability of NIV enables the rapid commencement of mechanical ventilation and may decrease the need for tracheal intubation. Also in 2010, Gupta et al¹⁰ reported that adding NIV for this population may accelerate improvement in lung function, decrease inhaled bronchodilator requirement, and shorten ICU and hospital stay. In 2011, Williams et al¹¹ enrolled 165 children with moderate to severe asthma and weighing ≤ 20 kg in a retrospective and prospective descriptive analysis. The results of this study demonstrated that NIV for these patients is safe and may improve clinical outcomes. Although NIV in asthma lacks large randomized controlled studies, there is an increasing body of evidence reporting benefits for this population.

Hess¹² reports that the identification of those likely to benefit from NIV should include their need for mechanical ventilator support and assessing the existence of exclusions (eg, need for airway protection, inability to fit an interface, cardiopulmonary arrest, uncooperative). In our case, the patient presented with a primary inability to ventilate due to a severe asthma exacerbation and associated hypertension. Severe asthma presents with a high work of breathing and hyperinflated lungs due to an inability to exhale. Ventilatory priorities include unloading the work of breathing, improving alveolar ventilation, avoidance of hyperinflation, providing supplemental oxygen, minimiz-

ing the positive-pressure impact on hemodynamics, administering inhaled therapeutic agents, and close monitoring of ventilatory mechanics.¹³ We applied a critical care transport ventilator (Vela) in the noninvasive mode with a full face mask to address the patient's ventilatory dysfunction. Pressure settings were initiated low and titrated for patient synchrony, compliance, and exhaled V_T values. This noninvasive application of ventilatory support unloaded the inspiratory muscles and provided a means to administer oxygen and inhaled medications and to closely monitor response to therapy without incurring the relative risks of orotracheal intubation and subsequent invasive mechanical ventilation. In maintaining the spontaneous respiratory drive, we were able to optimize total lung compliance and keep airway positive-pressure support to a minimum. Albuterol was administered utilizing the ventilator's nebulization function to power a standard jet nebulizer placed after the inspiratory filter on the ventilator output limb. Use of this function added flow to the circuit only during inspiration and did not negatively impact ventilator response sensitivity to the patient's inspiratory effort. The delivery and deposition of the albuterol given in this manner were questionable, and we opted to initiate therapy with our highest protocol dose, 40 mg/h. Ventilator graphical analysis allowed us to monitor the ventilator's ability to meet the patient's inspiratory demands, ensuring us of decreasing the work of breathing. Observing the expiratory limb through both ventilator graphics and capnography provided "real-time" monitoring of response to therapy, CO_2 removal, and helped guide decision making.

The use of sedation to facilitate tolerance of NIV in ARF is not commonly accepted due to concerns of decreasing the patients' respiratory drive, but it has been reported. In a survey of sedation practices for NIV in ARF, Devlin et al¹⁴ found that only 15% of respondents have never used sedation for NIV patients. Among the agents most frequently used were benzodiazepines. In 2008, Akada et al¹⁵ reported a preliminary study that successfully used dexmedetomidine in 10 patients to facilitate NIV tolerance. Takasaki et al¹⁶ reported the successful treatment of 2 severe asthmatic patients with the use of dexmedetomidine to facilitate NIV. Our decision to support NIV tolerance with small doses of lorazepam was due to the patient's definitive need for ventilatory support as evidenced by the arterial pH and P_{aCO_2} ; the relative risks associated with orotracheal intubation and invasive positive-pressure ventilation; and the acknowledgment that if there were any signs of deterioration, orotracheal intubation and invasive ventilation would be initiated immediately. Lorazepam was used in this case for anxiolysis but may have provided relief of dyspnea by impacting the patient's respiratory drive and allowing for an increase in expiratory time, adding additional benefit to alveolar ventilation.

The rapid positive response to therapy may be more a result of the rapid onset of the asthma exacerbation. Rapid-

onset asthma exacerbations have been reported to respond more quickly to emergency medical management and require fewer admissions than do exacerbations that present with longer onset flares.^{17,18}

Limitations of this report include maintaining P_{aO_2} at > 100 mm Hg during care and the lack of reported V_T values and related NIV device adjustments. Table 1 shows the timeline of ABG analysis during care. Although the blood gas results are supportive of improved ventilation and acidemia, the P_{aO_2} was consistently > 100 mm Hg. The first result on NIV revealed a P_{aO_2} of 205 mm Hg. This high value for the delivered F_{IO_2} of 0.40 may be attributed to lack of alveolar oxygen equilibration with what was being delivered relative to when the sample was drawn and/or a time documentation discrepancy. The remaining results, all > 100 mm Hg, do not support adherence to recommendations that minimizing F_{IO_2} delivery in asthma exacerbations can decrease potential CO_2 retention, as evidenced by Rodrigo et al.¹⁹ P_{aCO_2} was supported and treated through NIV. Our choice of F_{IO_2} was based primarily on support of oxygenation due to the patient's extremis. The lack of monitored V_T values, end-tidal carbon dioxide levels, and subsequent titration steps of ventilator parameters does not clarify the dynamic management involved in NIV for severe asthma.

What does make this case unique from previous case reports is the setting, the severity of the presentation, the use of anxiolysis, and the choice of the noninvasive ventilator in supporting the severe dyspnea. Our resuscitation bays are staffed with dedicated physicians, nurses, and respiratory therapists. Our emergency department ventilators have an NIV mode. This equipment has a distinct advantage in both monitoring and patient response capabilities compared with what has been used in other reported studies.²⁰ The immediate application of NIV with advanced mode and monitoring capabilities provided the patient with similar ventilatory support had the patient received orotracheal intubation, and without delay.

The application of NIV in this case may have stabilized the patient's ventilatory status and provided time for response to the usual and customary care of severe acute asthma without the complications and risks of tracheal intubation, sedation, and controlled mechanical ventilation. Real-time bedside diagnostics of the patient's ventilatory response to therapy provided us with information that was essential in managing the patient and designing the plan of care. It is impossible to know the extent to which this application of NIV contributed to the outcome. Use of NIV as an adjunct in severe acute asthma should be done only by experienced personnel and in a critical care setting with experienced support personnel and resources readily available. The benefit and safety of NIV therapy for severe acute asthma have not yet been established through randomized controlled trials.

REFERENCES

- Hill NS, Brennan J, Garpestad E, Nava S. Noninvasive ventilation in acute respiratory failure. *Crit Care Med* 2007;35(10):2402-2407.
- Lim W, Mohammed Akram R, Carson K, Mysore S, Labiszewski N, Wedzicha J, et al. Non-invasive positive pressure ventilation for treatment of respiratory failure due to severe acute exacerbations of asthma. *Cochrane Database Syst Rev* 2012;12:CD004360.
- Jaber S, Amraoui J, Lefrant JY, Arich C, Cohendy R, Landreau L, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study. *Crit Care Med* 2006;34(9):2355-2361.
- Delay JM, Sebbane M, Jung B, Nocca D, Verzilli D, Pouzeratte Y, et al. The effectiveness of noninvasive positive pressure ventilation to enhance preoxygenation in morbidly obese patients: a randomized controlled study. *Anesth Analg* 2008;107(5):1707-1713.
- Baillard C, Fosse JP, Sebbane M, Chanques G, Vincent F, Courouble P, et al. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. *Am J Respir Crit Care Med* 2006;174(2):171-177.
- Fernández MM, Villara A, Blanch L, Fernández R. Noninvasive mechanical ventilation in status asthmaticus. *Intensive Care Medicine* 2001;27(3):486-492.
- Hess DR, Pang JM, Camargo CA Jr. A survey of the use of noninvasive ventilation in academic emergency departments in the United States. *Respir Care* 2009;54(10):1306-1312.
- Soroksky A, Klinowski E, Ilgyev E, Mizrahi A, Miller A, Ben Yehuda TM, et al. Noninvasive positive pressure ventilation in acute asthmatic attack. *Eur Respir Rev* 2010;19(115):39-45.
- Murase K, Tomii K, Chin K, Tsuboi T, Sakurai A, Tachikawa R, et al. The use of non-invasive ventilation for life-threatening attacks: changes in the need for intubation. *Respirology* 2010;15(4):714-720.
- Gupta D, Nath A, Agarwal R, Behera D. (2010). A prospective randomized controlled trial on the efficacy of noninvasive ventilation in severe acute asthma. *Respir Care* 2010;55(5):536-543.
- Williams AM, Abramo TJ, Shah MV, Miller RA, Burney-Jones C, Rooks S, et al. Safety and clinical findings of BiPAP utilization in children 20 kg or less for asthma exacerbations. *Intensive Care Med* 2011;37(8):1338-1343.
- Hess DR. Noninvasive ventilation for acute respiratory failure. *Respir Care* 2013;58(6):950-972.
- Oddo M, Feihl F, Schaller MD, Perret C. Management of mechanical ventilation in acute severe asthma: practical aspects. *Intensive Care Med* 2006;32(4):501-510.
- Devlin WJ, Nava S, Fong JJ, Bahady I, Hill NS. Survey of sedation practices during noninvasive positive-pressure ventilation to treat acute respiratory failure. *Crit Care Med* 2007;35(10):2298-2302.
- Akada S, Takeda S, Yoshida Y, Nakazato K, Mori M, Hongo T, et al. The efficacy of dexmedetomidine in patients with noninvasive ventilation: a preliminary study. *Anesth Analg* 2008;107(1):167-170.
- Takasaki Y, Kido T, Semba K. Dexmedetomidine facilitates induction of noninvasive positive pressure ventilation for acute respiratory failure in patients with severe asthma. *J Anesth* 2009;23(1):147-150.
- Rodrigo GJ, Rodrigo C. Rapid-onset asthma attack: a prospective cohort study about characteristics and response to emergency department treatment. *Chest* 2000;118(6):1547-1552.
- Woodruff PG, Emond SD, Singh AK, Camargo CA Jr. Sudden-onset severe acute asthma: clinical features and response to therapy. *Acad Emerg Med* 1998;5(7):695-701.
- Rodrigo GJ, Rodriguez Verde M, Peregalli V, Rodrigo C. Effects of short-term 28% and 100% oxygen on P_{aCO_2} and peak expiratory flow rate in acute asthma. *Chest* 2003;124(4):1312-1317.
- Agarwal R, Malhotra P, Gupta P. Failure of NIV in acute asthma: case report and a word of caution. *Emerg Med J* 2006;23(2):e9.