Case Report

Noninvasive ventilation-neurally adjusted ventilator assist for management of acute exacerbation of chronic obstructive pulmonary disease

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ABSTRACT

Patient–ventilator asynchrony is common with noninvasive ventilation (NIV) used for management of acute exacerbation of chronic obstructive pulmonary disease (COPD). Neurally adjusted ventilator assist (NAVA) is a mode of ventilatory support which can minimize the patient–ventilator asynchrony. Delivering NIV with NAVA (NIV–NAVA) during acute exacerbation of COPD seems a logical approach and may be useful in reducing patient–ventilator asynchrony. However, there are no published reports which describe the use of NIV–NAVA for management of acute exacerbation of COPD. We describe the successful management of a 56-year-old gentleman presenting to the emergency department of our hospital with acute exacerbation of COPD with hypercapnic respiratory failure with NIV–NAVA.

KEY WORDS: Chronic obstructive pulmonary disease, exacerbation, neurally adjusted ventilator assist, noninvasive ventilation, patient–ventilator asynchrony

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INTRODUCTION

Noninvasive ventilation (NIV) for management of chronic obstructive pulmonary disease (COPD) with acute hypercapnic respiratory failure (AHRF) is currently the standard of care.[1] Over the last few decades, NIV has avoided enumerable endotracheal intubation and complications associated with it, including mortality.[1] However, despite these positive results, NIV fails in a subset of patients.[2-4] Conventional NIV with pressure support (NIV–PS) provides ventilatory support by augmenting either flow or pressure of patient-initiated breath. It is very difficult to maintain synchrony during flow- or pressure-sensed ventilation; therefore, patient–ventilator asynchrony is common during NIV–PS. This asynchrony may be responsible for NIV failure in a subset of patients.[5]

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Neurally adjusted ventilator assist (NAVA) provides ventilatory support based on the diaphragmatic contraction instead of flow or pressure changes in airways.[6] NAVA has been shown to be associated with better patient–ventilator synchrony;[7-9] therefore there is less likelihood of NIV failure. NAVA has also been used successfully for weaning purposes.[10] Therefore, delivering NIV with NAVA (NIV–NAVA) during acute exacerbation of COPD seems a logical approach and may be useful in reducing patient–ventilator asynchrony.[11,12] However, there are no published reports that describe the use of NIV–NAVA for management of acute exacerbation of COPD. We herein present a case of COPD with AHRF who was successfully managed with NIV–NAVA.

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CASE REPORT

A 56-year-old male diagnosed case of COPD for the past 2 years presented to the emergency department of our hospital with shortness of breath (Medical Research Council (MRC) Grade 3) and cough with expectoration for the last 3 days and decrease in level of consciousness for 1 day. There was no history of fever, chest pain, hemoptysis, orthopnea, pedal edema, and focal neurological deficit or seizure. He had no history of hypertension, diabetes, or pulmonary tuberculosis. There was no history of previous hospitalization, though he consulted his primary care physician for worsening of respiratory symptoms, two times in a preceding year. He was a reformed smoker with a smoking index of 450. He worked as a security guard in a hospital and left the job 2 years back due to his illness. Spirometry done 2 years ago showed evidence of moderately severe obstruction with no bronchodilator response. His medications included inhaled corticosteroids and long-acting beta agonist (ICS/LABA). He used to take alcohol on social occasions with no other illicit drug use. There was no significant family history of respiratory illness.

On examination, he was drowsy, but arousable and flapping tremors were present. He was using accessory muscles of respiration and had a respiratory rate of 34/min. Pulse oximetry showed an oxygen saturation of 84% on breathing in ambient air. Heart rate and blood pressure were 102/min (regular) and 130/80 mmHg, respectively. Respiratory system examination revealed barrel-shaped chest, bilaterally reduced chest wall movements, and hyper-resonant note on percussion with obliterated cardiac and liver dullness. On auscultation, the intensity of breath sounds was grossly reduced on both sides with presence of bilateral diffuse expiratory wheeze. There was no clinical evidence of heart failure or deep-venous thrombosis.

Arterial blood gas (ABG) analysis showed evidence of acute on chronic hypercapnic respiratory failure (pH = 7.251, $PaCO_2$ = 87.9 mmHg, and HCO_3 = 39 meq/dl). His blood counts and routine biochemistry were within normal limits. His chest radiography showed the presence of hyperinflated lung fields without consolidation or effusion. Electrocardiography showed right-axis deviation and P-pulmonale. Troponin-I and pro-b-type natriuretic peptide levels were within normal limits. Screening two-dimensional-echocardiography revealed mild dilatation of right ventricle and right atrium with moderate pulmonary arterial hypertension. Both ventricles were contracting normally. There were no features suggestive of pulmonary thrombosis. There was A-profile on lung ultrasound with no effusion or consolidation. Deep-venous systems of lower limbs were free from thrombus. For management of AHRF, the patient was started on standard medical care (bronchodilators, systemic steroids, and antibiotics) in addition to NIV. NIV was provided by NAVA ventilator with dedicated software (Servo-i Maquet, Sweden) after written informed consent was obtained from the next of kin. EdiA catheter was inserted through the nose, and its accurate position (to pick up diaphragmatic signals) was confirmed by characteristic waveforms on the ventilator screen. Proper fitting nonvented NIV face mask was used as interface to provide ventilation. Edi signals were used to select the initial NAVA level such that the estimated pressure curve (to provide a tidal volume of approximately 6–8 ml/kg of ideal body weight of 57 kg) overlapped with the pressure delivery curve. The NAVA level of 2.0 cm of H_2O/μv was required for this patient to generate the target tidal volume of around 486 ml. The airway pressure limit of 25 cm of H_2O, positive end-expiratory pressure of 6 cm of H_2O, and the FiO_2 of 0.4 were set to achieve oxygen saturation of at least 92% with adequate respiratory rate and tidal volume. Baseline vital signs, ventilator parameters, and ABG parameters were recorded [Table 1]. Subsequent assessment of vital signs, ventilator parameters, and ABG parameters were recorded at prespecified time intervals (initial at 30 min and then 6 hourly). Various types of asynchronies from recorded waveforms were noted and asynchrony index (ASI) was calculated [Table 1]. Patient comfort was noted using visual analog scale (VAS). Patient reported no discomfort to ventilator, with mild discomfort to the presence of EdiA catheter that resolved within 24 h of ventilation. Analysis of patient–ventilator asynchrony revealed only few asynchronies, the most common being double triggering [Table 1]. Once EdiA peak reduced without decrease in tidal volume (suggested improvement in respiratory failure), NAVA level was reduced by steps of 0.1 cm of H_2O/μv. Total duration of NIV use was 4 days and weaning was started on the 3rd day after respiratory failure improved. On discharge, his oxygen saturation was 93% on room air. ABG showed pH 7.445, $PaCO_2$ 48.5 mmHg, PaO_2 67.5 mmHg, and HCO_3 37.99 meq/dl. He was discharged home on ICS/LABA plus long-acting antimuscarinic agents.

DISCUSSION

This case report describes the successful use of NIV–NAVA as the sole modality of ventilation for the management of acute exacerbation of COPD. Over the years, NIV has earned Grade 1A recommendation for the management of acute exacerbation of COPD.[13] However, NIV failure does occur and rate may be as high as 40%.[2,14] Data also suggest that up to 46% patients may exhibit severe asynchronies (defined as ASI >10%) during the use of NIV for varying causes of respiratory failure, including COPD.[9,15,16] Fighting with the ventilator or patient–ventilator asynchrony is one of the causes of immediate NIV failure.[5] Patient–ventilator synchronization is therefore critical for improving the NIV success. In this context, NAVA as a mode of ventilation provides better patient–ventilator interaction and reduces asynchrony. Successful use of NIV–NAVA in this patient suggests the feasibility of this mode during acute exacerbation of COPD.

Table 1: Trends of various parameters during noninvasive ventilation–neurally adjusted ventilator assist

Parameters	Time interval				
	At presentation	30 min	Day 2	Day 3	Day 4
pH	7.251	7.322	7.391	7.405	7.445
$PaCO_2$ (mmHg)	87.9	82.4	65.9	57.4	48.5
HCO_3 (meq/l)	39.0	41.8	38.2	36.3	37.9
PaO_2 (mmHg)	58.6	63.4	82.5	63.2	75.3
SpO_2 (%)	96	95	95	94	94
FiO_2 (%)	40	40	35	30	28
Respiratory rate (min)**	34	26.3±3.2	20.6±2.8	14.5±2.3	14.9±2.0
Tidal volume (ml)**		460.4±82.4	475.6±76.6	581.2±96.8	468.6±77.4
NAVA ($cmH_2O/\mu v$)	2*	2	1.3	0.8	0.4
EdiA (μv)**		10.22±5.22	10.77±4.62	9.88±2.80	8.58±2.36
Leak (%)**		68.3±11.5	56.2±10.3	45.2±6.2	52.2±8.4
PEEP (cmH_2O)	6	6	6	6	6
ASI (%)#		4.13	3.25	2.82	2.63
VAS for ventilator		No discomfort	No discomfort	No discomfort	No discomfort
VAS for Edi catheter		Mild discomfort	Mild discomfort	No discomfort	No discomfort

*Initial setting of NAVA level=2 $cmH_2O/\mu v$ (adjsuted to generate tidal volume=456 ml), **All values at 30 min and afterwards are presented as mean±SD, #ASI is the total number of asynchronies (ineffectively triggered breaths plus double triggered breaths plus short cycled breaths plus prolonged cycled breaths) divided by the number of triggered and ineffectively triggered breaths × 100. NAVA: Neutrally adjusted ventilator assist, SD: Standard deviation, ASI: Asynchrony index, PEEP: Positive end-expiratory pressure, VAS: Visual analogue scale

For administration of NAVA, insertion of a special nasogastric catheter (EdiA catheter) is required. There may be two concerns while using EdiA catheter during NIV–NAVA – discomfort in the nose and leak around the interface (both are due to the presence of catheter). We monitored the patient closely for discomfort/pain (using VAS) and leak. We observed that there was mild patient-rated discomfort related to the presence of EdiA catheter for the initial 2 days. It indicates that the catheter was well tolerated, and the concern regarding discomfort related to it should not be a major limitation during NIV–NAVA. Second, due to the presence of catheter between the bed of the mask and skin, there may be leak. In this patient, we observed leaks ranging from 45% to 68%. However, no significant change was observed in the tidal volume. Also, leaks during NIV have been associated with asynchrony; however, we did not observe any significant asynchrony in this patient. Probably, NAVA has inbuilt compensatory mechanism for leak.

In a prospective, multicenter, observational study including patients with hypercapnic respiratory failure, severe asynchrony was observed in 43% of patients.[16] Autotriggering was seen in 13%, double triggering in 15%, ineffective breaths in 13%, premature cycling in 12%, and late cycling in 23% of patients. During NAVA, we can monitor the patient for the presence of all of these, if present. The severity is usually expressed as ASI, which is defined as the total number of asynchronies divided by the number of triggered and ineffectively triggered breaths × 100. In the index case, the ASI range was 4.1%–2.6%. These values indicate that patient–ventilator asynchrony may be minimized by using NAVA. Indeed, NAVA use has been shown to improve patient–ventilator interaction with ASI of 4.9% versus 15.8%; $P = 0.03$.[9]

To the best of our knowledge, this is the first case where NIV–NAVA was used as the sole ventilatory support during the entire management of acute exacerbation of COPD. Previous case reports or case series have used NAVA for patients on NIV to demonstrate the physiological effects or asynchrony using NIV–PS and NAVA, alternatively.[9,17] Our case report has shown that NIV–NAVA is feasible during acute exacerbation of COPD without significant incease in patient discomfort. Additinally, it can serve as weaning mode also. Improved patient–ventilator synchonization and benefit assosciated with that may be expected with this. Properly designed studies are required to assess the benefits of NIV–NAVA on outcomes related to improved patient–ventilator interactions such as NIV failure rate and duration of hospital and Intensive Care Unit stay.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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