

CASE REPORT

High intensity non-invasive positive pressure ventilation for refractory decompensated acute hypercapnic respiratory failure in advanced chronic obstructive pulmonary disease

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SUMMARY

Chronic obstructive pulmonary disease (COPD) is a debilitating progressive lung disease characterised by irreversible airflow obstruction. In addition to an increase in morbidity and mortality, exacerbation also results in frequent hospital visits, which place a burden on healthcare systems. Non-invasive positive pressure ventilation (NPPV) with conventional inspiratory pressures is the standard ventilatory support for patients in exacerbation. At present, the use of higher inspiratory pressures through high intensity non-invasive positive pressure ventilation (Hi-NPPV) during an exacerbation remains unknown. We describe a novel application of Hi-NPPV in a patient with acute exacerbation who was refractory to conventional NPPV.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common obstructive airway disease with the hallmark of irreversible airflow obstruction. Hypercapnia is defined by an elevation in the arterial carbon dioxide tension (PaCO₂) of ≥ 45 mmHg. Non-invasive positive pressure ventilation (NPPV) is established as the preferred treatment for COPD patients with acute decompensated hypercapnic respiratory failure (AHRF). Despite being known to help to reduce the length of hospital stay, rates of endotracheal intubation and overall mortality, the conventional low inspiratory airway pressures (IPAP) may have contributed to a quarter of non-NPPV responders. Conversely, the role of high inspiratory pressures via high intensity non-invasive positive pressure ventilation (Hi-NPPV) for COPD cases with AHRF remains unknown.

CASE REPORT

The patient was a 79-year-old man with advanced COPD grade 2, Global initiative for chronic obstructive lung disease (GOLD) D severity and was using domiciliary oxygen therapy. He had no other medical comorbidities. He was on optimal inhaler therapy, which included long-acting anticholinergics and β_2 -agonist. He had a history of five hospital admissions in the previous 12 months. He had an advance care directive against invasive ventilation.

He presented with COPD exacerbation. His Glasgow Coma Scale was 15, body temperature (T) of 37.5°C, blood pressure of 105/69mmHg and respiratory rate of 26 breaths per minute. He had a progression of his Modified medical research council (mMRC) dyspnoea scale of 3 to 4. He had widespread expiratory rhonchi on both lungs. His chest radiograph showed hyperinflated lung fields and no features of consolidation. Apart from an arterial blood gas (ABG), which showed features of AHRF, there were no significant abnormalities in all his blood results.

He was treated with inhaled bronchodilators, intravenous corticosteroids, a seven-day course of empirical intravenous Ceftriaxone and initiated on conventional NPPV. Despite on NPPV, the serial ABGs of the patient failed to show full compensation and he was not able to be weaned off NPPV. A trial of Hi-NPPV was attempted from day 15. The mode of bi-level ventilatory support (ResMed™ Stellar 100) was adjusted between pressure support ventilation and pressure control ventilation to achieve adequate tidal volumes, minute ventilation and appropriate oxygenation of SpO₂ 88%–92%. NPPV was connected to the patient's interface using a single limb circuit (Figure 1A). A total face mask (Figure 1B) was used transiently for three days (day 18 till 20). The patient was also closely monitored for potential complications, in particular worsening hypercapnia, barotrauma and hypotension. He was switched back to oral-nasal mask on day 21 (Figure 1C). The inspiratory pressures were titrated according to his tolerance and to achieve compensation in the blood gas results (Table I) and (Figure 2).

A diagnosis of deventilation dyspnoea was made (day 21 onwards) when the patient experienced dyspnoeic and lethargic on cessation of nocturnal Hi-NPPV in the morning with no other explanation identified. The symptoms resolved with the introduction of small doses of aqueous morphine (1 mg twice daily), nebulised bronchodilators and extending the duration of Hi-NPPV for an extra one to two hours, depending on the patient's symptoms.

The patient was also put on a multidisciplinary rehabilitation program and he was successfully weaned off daytime Hi-NPPV during the next 15 days. On day 40, he was discharged home

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Table III: Baseline information on treatment for patients according to GINA assessment of asthma control

Day of Admission	NPPV						ABG 1 hour after nocturnal NPPV						VAS 0-10
	Mode	Interface	IPAP (cmH ₂ O)	EPAP (cmH ₂ O)	O ₂ flow (L/min)	Backup Rate (breaths/min)	i-Time	pH	PaCO ₂ (mmHg)	PaO ₂ (mmHg)	HCO ₃ ⁻ (mmol/L)	BE (mmol/L)	
Admission	-	-	-	-	-	-	-	7.283	91.0	58.6	34.5	14.6	10
Day 1	ST	Oro-nasal	12	5	15	12	1.2	7.330	74.7	68.8	32.9	12.0	7
Day 3	ST	Oro-nasal	12	5	15	12	1.2	7.357	69.6	63.0	34.3	12.2	7
Day 5	ST	Oro-nasal	12	5	15	12	1.2	7.258	93.5	67.2	32.7	13.1	8
Day 7	ST	Oro-nasal	14	6	15	12	1.1	7.298	85.1	101.0	35.1	13.6	8
Day 9	ST	Oro-nasal	16	6	15	12	1.1	7.308	86.2	86.5	35.6	15.2	8
Day 11	ST	Oro-nasal	18	8	15	12	1.2	7.324	85.5	68.2	37.3	16.5	7
Day 13	ST	Oro-nasal	18	8	15	12	1.2	7.304	82.2	93.8	34.7	12.9	7
Day 15	ST	Oro-nasal	20	5	15	12	1.2	7.304	83.7	62.0	32.8	13.7	7
Day 17	ST	Oro-nasal	20	5	15	12	1.2	7.324	78.8	74.0	34.9	13.4	7
Day 18	PCV	Total-face	20	5	15	14	1.0	7.349	78.7	72.0	38.4	15.9	7
Day 20	PCV	Total-face	20	5	15	14	1.0	7.272	92.6	55.0	34.9	14.1	7
Day 21	ST	Oro-nasal	22	6	11	15	1.0	7.316	77.3	88.8	32.6	11.9	7
Day 22	PCV	Oro-nasal	26	7	6	15	0.8	7.369	67.2	67.1	33.4	12.1	7
Day 24	PCV	Oro-nasal	26	6	6	14	0.8	7.347	69.9	60.1	32.5	11.4	6
Day 26	PCV	Oro-nasal	28	4	5	14	0.8	7.410	64.9	64.9	36.4	14.9	4
Day 28	PCV	Oro-nasal	26	4	4	12	1.0	7.438	59.5	67.0	37.2	14.4	3
Day 30	ST	Oro-nasal	26	4	4	12	1.0	7.431	51.9	72.1	32.6	9.4	3

IPAP, inspiratory positive airway pressure; EPAP, expiratory positive airway pressure; O₂ flow, oxygen flow rate; i-Time, inspiratory time; PaCO₂, partial pressure of carbon dioxide; PaO₂, partial pressure of oxygen; HCO₃⁻, bicarbonate; BE, base excess; VAS, visual analogue scale (intensity of dyspnea: 0 = not dyspnea, 10 = maximum dyspnea; Mode, ST = spontaneous timed, PCV = pressure control ventilation.

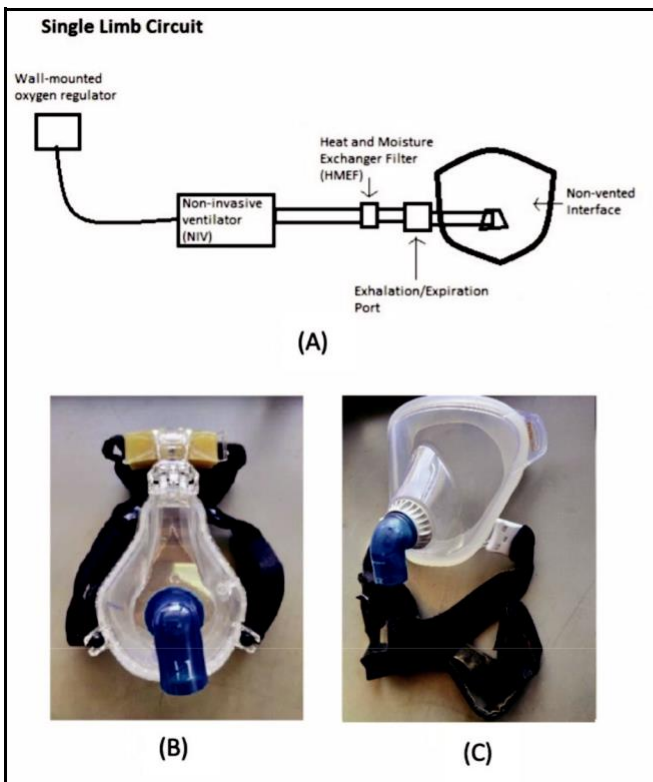


Fig. 1: a). Single limb non-invasive ventilator circuit used for the patient. b). Non-vented oral-nasal mask. c). Non-vented total face mask.

on nocturnal Hi-NPPV. The patient was well, compliant to his nocturnal Hi-NPPV on subsequent reviews and only recorded one exacerbation in the next 12 months. His recorded mMRC was 3.

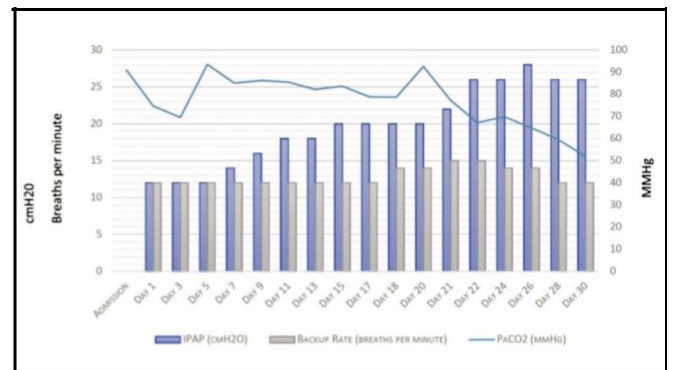


Fig. 2: Partial pressure of carbon dioxide in relation to inspiratory pressures.

DISCUSSION

COPD is a chronic disorder characterised by persistent respiratory symptoms and airflow limitation as a result of exposure in particular to chronic tobacco smoking. During COPD exacerbation, the mechanism of hypercapnia is thought to be caused by ventilation-perfusion inequality leading to increased dead space ventilation.

The benefits of NPPV in treating COPD exacerbations are well established. In 2009, Windish et al., first applied Hi-NPPV to stable COPD patients.¹ Although the inspiratory pressures were not precisely predefined, IPAP typically ranges from 20–30cmH₂O with an aim to normalised PaCO₂ levels. The mechanism of Hi-NPPV in COPD patients remains poorly understood. COPD patients treated with Hi-NPPV demonstrate improved gas exchange, possibly through optimised breathing patterns and pulmonary mechanics.² Patients had improvement in their quality of life and were found to adapt to Hi-NPPV despite being on high pressures.³

Currently, benefits of Hi-NPPV are only reported in chronic stable hypercapnic COPD patients with the latest clinical trial demonstrating a reduction in both exacerbation and mortality rates.⁴ There is still no published data on the use of Hi-NPPV in acute decompensated individuals.

The importance of choosing the correct interface is often overlooked when initiating NIV for AHRF. Physicians should bear in mind the no one-size-fits-all approach. The most effective interface should be comfortable enough to ensure the delivery of adequate ventilation and minimal leakage. A larger interface may not result in excess dead space ventilation, especially when applying high levels of pressure support as high inspiratory flow levels generated can prevent significant dead space ventilation.⁵

In this case, although conventional NPPV provided assisted ventilation, it was not able to help achieve full compensation in the blood gas results. Based on the understanding of the mechanism of Hi-NPPV in stable COPD patients, we extrapolated this application to our patient. The Hi-NPPV demonstrated similar results by providing day and night-controlled ventilation. The compensation of the ABG result was achieved with an IPAP of 26cmH₂O. The backup rate was kept at 15 breaths per minute and below to ensure the comfort of the patient, prevention of lung hyperinflation and the emergence of patient-ventilator asynchrony. A total face mask was used to replace the oral-nasal mask to provide relief for pressure sores over the nasal bridge. Besides, it was also used to overcome leaks from high inspiratory pressures to maintain adequate alveolar ventilation.

While the benefits of Hi-NPPV are well described, high pressures could potentially contribute to dynamic hyperinflation leading to a less-reported phenomenon called “deventilation dyspnoea” after cessation of nocturnal Hi-NPPV.⁶ The exact mechanism is unknown. In anticipating the possible effects of ventilator-patient asynchrony as a cause of DD, we reviewed his overnight NPPV

data. We were satisfied with the overall results demonstrating good compliance, oxygenation and minimal levels of unintentional leakage. The potential treatment options to overcome DD included optimal bronchodilators, small doses of oral opioids and extending the duration of NPPV during wakefulness.

CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.
Ethical statement: An ethical approval for case report was not required by the institution ethics board.

ACKNOWLEDGEMENT

The authors would like to express our gratitude to Dr. Geak Poh Tan from the Department of Respiratory and Critical Care Medicine, Tan Tock Seng Hospital, Singapore for his invaluable input on deventilation dyspnoea.

REFERENCES

1. Windisch W, Haenel M, Storre JH, Dreher M. High-intensity non-invasive positive pressure ventilation for stable hypercapnic COPD. *Int J Med Sci* 2009; 6(2): 72-6.
2. Windisch W, Storre JH, Köhnlein T. Nocturnal non-invasive positive pressure ventilation for COPD. *Expert Rev Respir Med* 2015; 9(3): 295-308.
3. Dreher M, Storre JH, Schmoor C, Windisch W. High-intensity versus low-intensity non-invasive ventilation in patients with stable hypercapnic COPD: a randomised crossover trial. *Thorax* 2010; 65(4): 303-8.
4. Murphy PB, Rehal S, Arbane G, Bourke S, Calverley PM, Crook AM, et al. Effect of home noninvasive ventilation with oxygen therapy vs oxygen therapy alone on hospital readmission or death after an acute COPD exacerbation: a randomized clinical trial. *JAMA* 2017; 317(21): 2177-86.
5. Sadeghi S, Fakharian A, Nasri P, Kiani A. Comparison of comfort and effectiveness of total face mask and oronasal mask in noninvasive positive pressure ventilation in patients with acute respiratory failure: a clinical trial. *Can Respir J* 2017; 2017: 2048032.
6. Adler D, Perrig S, Takahashi H, Espa F, Rodenstein D, Pépin JL, et al. Polysomnography in stable COPD under non-invasive ventilation to reduce patient-ventilator asynchrony and morning breathlessness. *Sleep Breath* 2012; 16(4): 1081-90.