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 β2-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.

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), nebulated 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.
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.

**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 20–30cmH\(_2\)O with an aim to normalised PaCO\(_2\) 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.\textsuperscript{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.\textsuperscript{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 26cmH\textsubscript{2}O. 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-HPPV.\textsuperscript{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.

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**REFERENCES**