

High-flow nasal cannula oxygen for reverting severe acute exacerbation of chronic obstructive pulmonary disease: A case report



Oxigenoterapia de alto flujo con cánula nasal en el tratamiento de la exacerbación de la enfermedad pulmonar obstructiva crónica: informe de un caso

Dear Editor,

High-flow nasal cannula (HFNC) oxygen therapy is a recent technique enabling delivery of high flow rate (up to 70 L/min) of gas heated and humidified as in physiological conditions. This strategy of oxygenation could be beneficial in ICU patients to avoid intubation in those with acute hypoxemic respiratory failure.¹ The high-flow rate of gas continuously delivered in the airways may generate positive end-expiratory pressure (PEEP) effect² and a washout of dead space, flushing carbon dioxide (CO₂) out of the upper airways.³ This phenomenon may help to improve alveolar ventilation and to reduce work of breathing.⁴ To date, no study has been conducted to assess HFNC in acute hypercapnic respiratory failure. We report here the case of a 72-year-old male admitted to ICU for acute exacerbation of Chronic Obstructive Pulmonary Disease (COPD) successfully treated with HFNC after failure of noninvasive ventilation (NIV).

The history of this patient smoker with severe COPD (stage IV according to the GOLD classification) began 3 days prior to his admission with breathlessness worsening, fever and purulent cough. Clinical findings on admission to emergency department included signs of respiratory distress with a respiratory rate at 28 breaths/min and activation of accessory respiratory muscles. His temperature was 38.2 °C, heart rate 110 beats/min, blood arterial pressure 140/70 mmHg, without signs of congestive heart failure. He was stuporous as indicated by a Kelly–Matthay score of 4. Auscultation revealed crackles over the right side of the chest. Arterial blood gas values on admission revealed respiratory acidosis with pH 7.27 and PaCO₂ 89 mmHg, and PaO₂ was 45 mmHg on room air. Chest computed tomography (CT) with angiogram was negative for pulmonary embolism and revealed right baseline infiltrates. The bacterial sputum culture and virology studies were negative. The diagnosis of acute exacerbation of COPD was retained and he received inhaled bronchodilators, systemic steroids, Ampicillin-Sulbactam plus Clarithromycin and Oseltamivir. Given the severity, he was rapidly admitted to the ICU to start NIV. Large leaks immediately occurred after NIV initiation probably because of his facial anatomy characterized by retrognathia, very thin and bearded. Given the difficulties to carry out NIV, three different masks and two types of ventilators were tested to ensure patient–ventilator interaction and alveolar ventilation. The first blood gas in ICU revealed worsening of hypercapnia, reaching 91 mmHg (Table 1) using pressure-support 10 cmH₂O, PEEP 8 cmH₂O, tidal volume 520 ml. Due to persistent major leaks and a poor adherence, NIV was

stopped and replaced by HFNC with a flow of 50 L/min and FiO₂ of 45% through an ICU ventilator (Dräger Evita XL, Lübeck, Germany) and a heated humidifier (Fisher & Paykel, Auckland, New Zealand). Table 1 summarizes the patient's evolution over time. The first blood gas 1 h after initiation of HFNC showed a significant decrease in PaCO₂. Respiratory acidosis and altered consciousness were normalized 24 h after the use of HFNC. Oxygen was switched from HFNC to standard nasal prong 3 days after ICU admission. He was transferred to the ward the same day and discharged to home on oxygen therapy 11 days after hospital admission.

There is strong evidence that the use of noninvasive ventilation (NIV) as first-line therapy is beneficial in patients with severe exacerbation of COPD in as much as it reduces endotracheal intubation and mortality.⁵ Air leaks are frequent during NIV and may impair its efficiency, by reducing patient tolerance and by promoting numerous patient–ventilator asynchronies.⁶ In the present case report, several interfaces and types of ventilators were tested due to patient's anatomic characteristics and poor tolerance.⁷ None of these options allowed acceptance of the patient, resulting in progression of hypercapnia and reduced level of consciousness, both being 2 strong factors of NIV failure,⁸ which led us to use an alternative method in order to avoid endotracheal intubation. Therefore, we decided to switch for the use of HFNC whereas his PaCO₂ reached 91 mmHg.

To date, only one case report has proposed HFNC as an alternative method in a patient who did not tolerate any NIV, reporting better tolerance and good results with the use of the cannula (i.e. a decrease in PaCO₂ and a progressive correction of acidosis).⁹ Unlike our, the authors delivered high-flow through a dedicated device (Optiflow®, Fisher&Paykel, New Zealand) with flow of 60 L/m and a FiO₂ of 60% in a patient with relatively low PaCO₂ levels that rose overtime.⁹ By contrast, we delivered high-flow through an ICU ventilator (Dräger Evita XL, Lübeck, Germany) in a patient with hypercapnia exceeding 90 mmHg at time of HFNC initiation.

Although HFNC does not offer pressure support, the high flow generates PEEP and provides continuous washout of dead space in the airways. These 2 mechanisms were probably the most effective to improve alveolar ventilation in our patient. Parke et al. measured nasopharyngeal pressure in patients at different levels of flow using HFNC and found a PEEP level ranging from 2 to 4 cmH₂O with a gas flow rate of 50 L/min.² Although the PEEP level seems only moderate, this PEEP effect could help to improve gas exchange and to decrease work of breathing in patients, as with CPAP in patients with intrinsic PEEP.¹⁰ The high-flow rate of gas continuously delivered in the airways may also generate a washout of dead space from the pharynx into the bronchi, flushing carbon dioxide (CO₂) out of the upper airways.³ This phenomenon may help to improve alveolar ventilation and to reduce work of breathing.⁴ As compared to NIV, severe studies have found that the patients had less discomfort and less breathlessness with HFNC.¹ Although these studies assessed comfort in hypoxemic patients we believe that these findings may be extrapolated to hypercapnic patients.

The use of HFNC under strict clinical monitoring in an acute care facility could be an alternative treatment option

Table 1 Summary of the main clinical changes, arterial blood gases and ventilator support for a hypercapnic patient using high flow nasal cannula.

Ventilatory support	Hospital admission	ICU (1 h)	ICU (2 h)	ICU (12 h)	ICU (24 h)	ICU (48 h)	ICU (60 h)
	Spontaneous	NIV face mask	HFNC	HFNC	HFNC	HFNC	Nasal prong O ₂
FiO ₂ (%)	21	45	45	45	45	35	3 L/m
pH	7.27	7.29	7.32	7.37	7.41	7.44	7.41
PaCO ₂ (mmHg)	89	91	83	78	66	61	63
PaO ₂ (mmHg)	45	73	63	64	64	52	83
HCO ₃ (mEq/L)	40.3	43	42.7	44.8	42	40.8	39.6
EB (mEq/L)	8.3	11.9	11.7	14.5	13.7	13.6	11.9
SaO ₂ (%)	75	93	91	93	93	90	96
Respiratory rate (breaths/min)	34	28–30	28	26	24	24	20
Accessory muscles	Yes	Yes	Yes	No	No	No	No
Kelly–Matthay score	3	4	3	2	1	1	1

instead of endotracheal intubation after NIV failure in COPD patients with acute hypercapnia.

Funding

None declared.

Conflict of interest

None declared.

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2173-5727/

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