Efficacy of High-Flow Oxygen by Nasal Cannula With Active Humidification in a Patient With Acute Respiratory Failure of Neuromuscular Origin

Salvador Díaz-Lobato MD PhD, Miguel Angel Folgado MA MD, Angel Chapa MD, and Sagrario Mayoralas Alises MD PhD

The treatment of choice for patients with respiratory failure of neuromuscular origin, especially in patients with hypercapnic respiratory acidosis, is noninvasive ventilation (NIV). Endotracheal intubation and invasive ventilation are indicated for patients with severe respiratory compromise or failure of NIV. In recent years, high-flow oxygen therapy and active humidification devices have been introduced, and emerging evidence suggests that high-flow oxygen may be effective in various clinical settings, such as acute respiratory failure, after cardiac surgery, during sedation and analgesia, in acute heart failure, in hypoxemic respiratory distress, in do-not-intubate patients, in patients with chronic cough and copious secretions, pulmonary fibrosis, or cancer, in critical areas and the emergency department. We report on a patient with amyotrophic lateral sclerosis who arrived at the emergency department with acute hypercapnic respiratory failure. She did not tolerate NIV and refused intubation, but was treated successfully with heated, humidified oxygen via high-flow nasal cannula. Arterial blood analysis after an hour on high-flow nasal cannula showed improved pH, P_{aCO_2}, and awareness. The respiratory acidosis was corrected, and she was discharged after 5 days of hospitalization. Her response to high-flow nasal cannula was similar to that expected with NIV. We discuss the mechanisms of action of heated, humidified high-flow oxygen therapy. *Key words: high flow oxygen therapy; noninvasive ventilation; amyotrophic lateral sclerosis; nasal cannula.* [Respir Care 2013;58(12):e164–e167. © 2013 Daedalus Enterprises]

Introduction

Noninvasive ventilation (NIV) is the treatment of choice for patients with respiratory failure of neuromuscular origin, especially in patients with hypercapnic respiratory acidosis.[1,2] Endotracheal intubation is indicated for patients with severe respiratory compromise or NIV failure. In patients with amyotrophic lateral sclerosis (ALS), respiratory muscle weakness is the major cause of mortality, so NIV is an important part of ALS management.[3] Randomized controlled trials have indicated that therapy with long-term NIV improves survival in ALS patients. It can also improve patient symptoms and health-related quality of life.[4] NIV has been shown to be effective in correcting respiratory failure, probably by acting to a greater or lesser extent on muscle fatigue, the mechanical properties of the respiratory apparatus, the control of ventilation, the alterations in gas exchange during the night, leading to loss of sensitivity of central and peripheral chemoreceptors, and the degree of dysfunction of the upper airways.[5]

In recent years, heated, humidified high-flow nasal cannula (HFNC) has been introduced into medical practice.[6] There is emerging evidence that HFNC may be effective in various clinical settings, including acute respiratory failure,[7-9] after cardiac surgery,[10,11] during sedation and analgesia,[12] in acute heart failure,[13] in hypoxemic respiratory

Drs Díaz-Lobato and Mayoralas Alises are affiliated with the Pneumological Department, Ramón y Cajal Hospital, Madrid, Spain. Drs Folgado and Chapa are affiliated with the Emergency Department, Virgen de la Concha Hospital, Zamora, Spain.

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Correspondence: Salvador Díaz-Lobato, Pneumological Department, Hospital Universitario Ramón y Cajal, Carretera de Colmenar Viejo, km 9,100, 28034 Madrid, Spain. E-mail: sdiazlobato@gmail.com.

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Figure. Left: MaxVenturi flow source. Middle: MR850 humidifier. Right: Optiflow nasal cannula.

distress, in do-not-intubate patients,[14] and in patients with chronic cough and copious secretions,[15] pulmonary fibrosis,[16] or cancer,[17] in critical care areas[18] and in the emergency department.[19] We report on a patient with ALS with acute hypercapnic respiratory failure who was successfully treated with HFNC.

Case Report

A 65 year old woman was diagnosed with ALS 6 months prior to presentation, although she had no other noteworthy medical history. Usually she had no dyspnea, orthopnea, or other respiratory symptoms. Recent spirometry showed FVC 2,450 mL (78% of predicted), FEV_1 1,420 mL (75% of predicted), and FEV_1/FVC 0.7. Her maximum inspiratory and expiratory pressures were normal: $>$ 80 cm H_2O. She arrived at the hospital emergency department complaining of progressive dyspnea and decreased level of consciousness over 2 days. She had no fever, cough, or expectoration.

On physical examination she was drowsy, had a blood pressure of 140/70 mm Hg, a heart rate of 110 beats/min, a breathing frequency of 13 breaths/min, a temperature of 36.8°C, and showing an overall reduction of breath sounds on auscultation. The remaining physical examination was unremarkable. Laboratory tests showed hemoglobin 16 g/dL, hematocrit 50.6%, white blood cell count 8.7×10^3 cells/mL, 87% neutrophils, international normalized ratio 0.9, creatinine 0.59 mg/dL. Arterial blood gas (ABG) analysis of a sample collected while she was on F_{IO_2} 0.50 via air-entrainment mask showed pH 7.27, P_{CO_2} 90 mm Hg, P_{O_2} 88 mm Hg, and HCO_3^- 40 mmol/L.

Chest radiography showed left costophrenic angle obliteration but no other findings of interest. Her husband related the onset of symptoms with a catarrhal process. The diagnosis was hypercapnic respiratory acidosis, without infiltrates, secondary to a respiratory infection in a patient with ALS.

She was treated with antibiotics and steroids, and started on NIV (Trilogy 100, Philips Respironics, Murrysville, Pennsylvania) via oronasal mask with a passive circuit, in the S/T mode, with inspiratory pressure of 16 cm H_2O, expiratory pressure of 6 cm H_2O, backup rate of 15 breaths/min, trigger sensitivity setting 2, and rise time setting 2. Oxygen was added at 5 L/min, and arterial saturation and heart rate were monitored. She had immediate severe intolerance of NIV, with agitation and refusal to continue wearing the mask, so NIV was withdrawn after only 5 min. Arterial oxygen saturation was 80%.

She refused intubation, so we tried heated, humidified HFNC (Optiflow cannula with humidification by an MR880 heated humidifier, both from Fisher & Paykel Healthcare, Auckland, New Zealand) (Figure), at 45 L/min and an F_{IO_2} of 0.26. After an hour of HFNC she showed clinical improvement and was more awake. ABG showed pH 7.31, P_{CO_2} 74 mm Hg, P_{O_2} 51 mm Hg, HCO_3^- 36 mmol/L. She was admitted to the respiratory ward. Three hours later, an ABG showed pH 7.40, P_{CO_2} 61 mm Hg, P_{O_2} 62 mm Hg, and HCO_3^- 41 mmol/L. She gradually improved, and was discharged after 5 hospital days. HFNC was used throughout her hospitalization. A later chest radiograph showed similar left costophrenic angle obliteration, but no other findings of interest. On the day of discharge, we put the patient on conventional oxygen therapy via nasal prongs, at 1.5 L/min, for 5 hours in the morning.

An ABG showed pH 7,39, P_{CO_2} 48 mm Hg, P_{O_2} 68 mm Hg, and HCO_3^- 35 mmol/L. Domiciliary oxygen via nasal prongs, at 1.5 L/min, was recommended.

Discussion

In our patient, management was similar to that of a patient with a COPD exacerbation and respiratory hypercapnic acidosis; we used a low F_{IO_2}, aimed to maintain arterial saturation slightly higher than 90%, to prevent greater hypoventilation. We monitored the patient as if she were receiving NIV, and ABG after the first hour of HFNC showed improved pH, P_{aCO_2}, and awareness. Respiratory acidosis was corrected and the patient was discharged after 5 days of hospitalization. The response to HFNC was similar to that expected with NIV. Basically, we applied the same treatments we use for COPD exacerbation.

Heated, humidified HFNC oxygen can deliver F_{IO_2} up to 1.0, at a maximum flow of 60 L/min, via nasal prongs or cannula. The benefits of HFNC are related to a number of physiological properties.[20] One of the main effects of high flows in the nasopharynx is to wash CO_2, which reduces CO_2 rebreathing and provides a reservoir of fresh gas, which reduces dead space and increases the ratio of alveolar ventilation to minute ventilation.[21] Because HFNC can generate flows that match or exceed a patient's peak inspiratory demand, it is thought that HFNC minimizes the nasopharyngeal resistance, thus decreasing resistive work of breathing. In the same line of reasoning, several authors speculate that the high flow generates a small amount of positive airway pressure[22-24]: approximately 1 cm H_2O of pressure for every 10 L/min of flow. HFNC cannot be considered a CPAP device, but we cannot forget this feature, which provides some benefit to respiratory mechanics. On the other hand, Corley et al[25] demonstrated in patients with acute respiratory failure that at least part of the improvement in oxygenation is due to alveolar recruitment. Finally, HFNC is well tolerated because the gas is heated and humidified, HFNC uses nasal prongs instead of face mask, HFNC reduces the work of breathing, and HFNC may help correct hypoxemia.[26]

HFNC's usefulness in critically ill patients, weaning, and as an alternative to intubation is being investigated, and there is promising evidence in the literature.[27,28] The dead-space wash-out, nasopharyngeal resistance reduction, positive pharyngeal pressure, alveolar recruitment, oxygen dilution reduction, decreased work of breathing, and patient comfort may explain the clinical and gasometric improvement in our patient.

Our experience opens up a field of research in which some patients with acute respiratory failure and hypercapnic acidosis might be treatable with humidified HFNC, as an alternative to conventional NIV. The simplicity of the technique, the lower cost of equipment, and the greater patient tolerance add benefit to the clinical efficacy, which is very important in an era when efficiency should be prioritized. Future research is necessary to clarify these issues.

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