Noninvasive ventilation in pediatric acute respiratory failure: Two case reports

Gee-Gwo Yang, Neng-Huang Pan, Hui-Ping Tung

Abstract

Acute respiratory failure (ARF) is the main cause of cardiac arrest in children. Many studies suggest noninvasive ventilation (NIV) without endotracheal tube intubation (ETI) in the treatment of pediatric ARF. However, there are many important considerations, such as identifying the patient, proper timing for the NIV procedure, and the appropriate setting. We used NIV in a 6-year-old ARF patient and a 14-year-old ARF patient. NIV can produce positive results in children with ARF as well as adults. In our two cases, we showed the acute setting and the use of NIV at the right time.

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Keywords: acute respiratory failure; endotracheal tube intubation; noninvasive ventilation

1. Introduction

Invasive mechanical ventilation (IMV) through an endotracheal tube or tracheostomy has been used as a life-saving treatment for acute respiratory failure (ARF) in past decades. However, risks and complications have been associated with endotracheal tube intubation (ETI). The use of noninvasive ventilation (NIV) has become increasingly popular as a supporting treatment for ARF in children. Compared to IMV, NIV is much safer and has fewer complications, such as upper airway trauma, laryngeal swelling, post-extubation vocal cord dysfunction, and nosocomial infection. It has been proven to reduce hospital admissions for ETI cases. We followed the Bernd Schönhofer et al guidelines and used NIV in our units prior to intubation to manage the above concerns. We believe that early management of ARF could prevent such complications.

Based on the reports of the Bureau of National Health Insurance, the treatment of ARF in pediatric patients cost about 9 million United States dollars in 2009. When treating ARF, physicians must be aware of the ideal type of ventilation support for each individual patient to provide the greatest benefit with the lowest risk. Should NIV be used in children as well as adults? Here we present evidence supporting its use in pediatric patients of different ages with ARF.

2. Case Reports

2.1. Case 1

A 14-year-old girl was admitted to this hospital in the summer because of a recurring fever. She had had an episode of meningitis at the age of 4–5 years and became chronically dependent on valproic acid (Depakine) and carbamazepine (Tegretol) at the age of 5–6 years to control seizures. Five days prior to being admitted to the hospital, she developed a
fever, productive cough, and rhinorrhea. On the morning of her admission to the hospital, she woke up with dyspnea and was feeling weak. She was brought to our pediatric outpatient department. Her physical examination revealed that she had breathing difficulties while using accessory respiratory muscles. Her respiratory rate was 24–30 cycles/minute; blood pressure was 113/71 mmHg; pulse rate was 82 beats/minute; bilateral basal crackles extended from the lower third to half of the lung fields; and, her oxygen saturation level was 90–91%. Oxygen was administered through a non-rebreather mask and the oxygen saturation level increased to 100%.

Her chest X-ray revealed a small patch and linear infiltration in the left and right upper lung fields, with a mild increase in the lung markings in both lung fields. The chest computed tomography revealed alveolar patchy opacities in the right and left upper, and left lower lung fields, along with pleural effusion. Penicillin G was administered intravenously. Approximately 3 hours after arrival, the patient was transferred to the ward.

On the 5th day of hospitalization, the patient became breathless and had an increased respiratory rate of 63 cycles/minute, as well as a drop in her oxygen saturation level to 86% while she was breathing 40% oxygen through a face mask, with forceful contraction of accessory muscles. Her body temperature was 40.3°C and her blood pressure was 86/67 mmHg. Auscultation disclosed rhonchi and diminished breathing sounds in the left lung field.

Her arterial blood gas (ABG) results were pH: 7.425; PaCO₂: 36.8 mmHg; PaO₂: 36.6 mmHg; HCO₃⁻: 23.6 mM/L; Base Excess (BE): −0.7 mM/L; and, SaO₂: 71.4. The patient was transferred to the intensive care unit (ICU).

In the ICU, oxygen saturation was 98% using NIV support with a mask interface in spontaneous/time mode at a setting rate of 20 cycles/minute; inspiratory positive airway pressure was 20 cm H₂O; expiratory positive airway pressure was 9 cm H₂O; and, the O₂ flow rate was 15 L/minute. We continuously observed her and were prepared for intubation once we saw signs of deterioration. Amoxicillin clavulanate was replaced by cefotaxime and azithromycin. The aerobic culture of the first sputum specimen showed mixed normal flora, whereas the second specimen showed no growth. There was no order for a blood culture. Two hours later, the ABG results were pH: 7.48; PaCO₂: 34 mmHg; PaO₂: 138 mmHg; HCO₃⁻: 25.3 mM/L; BE: 1.8 mM/L; and, SaO₂: 99%.

On the 8th day, the patient’s body temperature was 37.8°C, blood pressure was 105/75 mmHg, and respiratory rate was 42 cycles/minute. She had a retraction respiratory pattern without contractions of the accessory respiratory muscles. ABG results were pH: 7.52; PaCO₂: 33 mmHg; PaO₂: 111 mmHg; HCO₃⁻: 26.9 mM/L; BE: 4.0 mM/L; and SaO₂: 96%. The pulse rate was 112 beats/minute. The chest film revealed resolution of the pneumatic patch. Other laboratory data are shown in Table 1.

2.2. Case 2

A 6-year-old girl with Down syndrome was admitted to this hospital in mid-spring because of increasing dyspnea, cough, rhinorrhea, and fever. The patient had been in her usual state of health until she began developing these symptoms 3–4 days earlier, due to progressive shortness of breath. Her cough and rhinorrhea were aggravated on the morning of admission. The patient was brought to the emergency room at 4:23 AM. When she arrived, she was acutely ill with a BP of 110/70 mmHg, pulse rate of 128 beats/minute, and respiratory rate of 32 cycles/minute. She had warm and pale skin, interrupted speech caused by dyspnea, contractions in her accessory muscles, and

<table>
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<th>Variable</th>
<th>Reference range</th>
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NG = no growth; MNF = mix normal flora; FM = face mask; NRM = nonrebreathing mask; NIV = noninvasive ventilation.

a bilateral wheeze. Her oxygen saturation level was 80–85% on room air. An O2 flow rate of 12 L/minute was administered through a non-rebreather mask. ABG results were pH: 7.304; PaCO2: 57.2 mmHg; PaO2: 62.4 mmHg; HCO2: 26.9 mM/L; BE: 0.2 mM/L; and, SaO2: 92.2%. She was subsequently transferred to the ICU for further care.

In the ICU, NIV was used for oxygen and ventilation support. The ABG results were pH: 7.38; PaCO2: 46.5 mmHg; PaO2: 132.4 mmHg; HCO2: 27.8 mM/L; BE: 0.2 mM/L; and SaO2: 98.6%. A chest radiograph revealed a patchy opacity in the right lung field with pleural effusion. Blood culture showed no growth. The aerobic culture of the first sputum specimen showed mixed normal flora, whereas the second specimen showed no growth. The patient was treated with cefotaxime and azithromycin. The respiratory rate decreased to 20–25 cycles/minute. There was improved oxygenation and CO2 retention. The chest X-ray revealed decreasing pleural effusion. The laboratory data are shown in Table 1.

3. Discussion

The frequency of ARF is higher in infants and young children than in adults. In addition, respiratory failure often precedes cardiopulmonary arrest in children.1 Our cases indicate the effectiveness of NIV in children with ARF.

In general, respiratory failure is classified as either hypoxemic respiratory (type I)15 or hypercapnic (type II).16 Type I respiratory failure is characterized by PaO2 < 60 mmHg with a normal or low PaO2. The primary treatment is to administer supplemental oxygen at a sufficient level to increase the arterial oxygen saturation (SaO2) to >94%. When it is necessary for a fraction of inspired oxygen (FiO2) to be >0.5 to achieve the goal, the situation is often referred to as acute hypoxemic respiratory failure. Type II respiratory failure is a consequence of ventilation failure and occurs in conditions where the respiratory pump has been affected, causing depressed neural ventilatory drive, acute or chronic upper airway obstruction, and alveolar hypoventilation characterized by a PaCO2 > 50 mmHg. The onset of type II failure may be insidious and may develop when respiratory muscle fatigue complicates pre-existing disorders, such as pneumonia or acute severe asthma (this is the current term for status asthmaticus), which initially appears with hypoxemia with no hypoventilation. Administration of oxygen alone is not an appropriate treatment for hypercapnic respiratory failure and could result in the retention of more carbon dioxide in partial pressure decreases respiratory muscles overload in young infants with severe acute viral bronchiolitis.

We followed the Bernd Schönhofer et al17 guidelines which contain verified indications, contraindications, and criteria for monitoring and terminating NIV, as well as the advantages and disadvantages of NIV vis-à-vis invasive ventilation. The main goal of these guidelines is to encourage broader use of this form of treatment in acute care based on scientific evidence. Cambonie et al12 studied 12 patients with ARF, showing that NIV avoided ETI in 60–100% of cases.

Abadesso et al18 studied 115 patients with ARF; the mean hospital admission was 9.7 ± 9.5 days (median 8). The difference between the NIV success group and NIV failure group was statistically significant.

In our cases, NIV reduced the need for intubation in ARF—the same outcome achieved by studies in many countries. NIV not only improved gas exchange by distending collapsed lung units, but also reduced the load on the respiratory muscles. The use of NIV in pediatric patients with ARF, based on our cases, has the same effect as in adults.

We are not aware of any age limitations on the use of NIV, but look forward to conducting a randomized clinical trial and meta-analysis.

Conflicts of interest

All contributing authors declare no conflicts of interest.

References


