Diagnosis and treatment of negative pressure pulmonary edema in a pediatric patient: A case report

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This article includes a case study of a pediatric patient who presented for elective correction of a speech impediment via a frenuloplasty. The patient's history and anesthetic course will be discussed prior to the development of the episode of negative pressure pulmonary edema (NPPE). The events surrounding the development and diagnosis of NPPE will be presented, and the treatment modalities will be discussed.

Key words: **Extubate, negative pressure pulmonary edema, ventilation.**

Negative pressure pulmonary edema (NPPE) occurs when an upper airway obstruction causes enough negative intrathoracic pressure to pull fluid from the pulmonary capillary bed and into the alveoli, leading to ventilation and perfusion difficulties. Although relatively uncommon and easily treated, it is important for the anesthesia provider to recognize the signs and symptoms and initiate the proper treatment.

The patient in this case study was a female who presented for frenuloplasty to correct a speech impediment. This article will discuss the occurrence, diagnosis, and treatment of NPPE during general anesthesia.

Case summary

A 4-year-old female weighing 19.5 kg was scheduled for frenuloplasty to correct an impairment of speech development. A small incision was to be made at the base of the frenulum, thereby freeing the tongue to allow for proper enunciation. Her physical status was designated as an American Society of Anesthesiologists class 2, due to a history of upper respiratory infections and a recent cough that had subsided. She had no known drug allergies. Her surgical history consisted of a tonsillectomy, adenoidectomy, and placement of ear tubes without incident. No preoperative laboratory studies were drawn. Premedication consisted of midazolam, 8 mg by mouth; atropine, 0.4 mg by mouth; and acetaminophen with codeine, 1.5 teaspoons by mouth.

The patient was brought to the operating room and monitored with standard electrocardiograph, blood pressure, pulse oximetry, and temperature equipment. An uneventful mask induction with ultane, 70% nitrous oxide, and 30% oxygen was followed by insertion of a 22-gauge peripheral intravenous catheter. Intubation with a 5.5-cm uncuffed oral reinforced airway tube was atraumatic on the first attempt. After bilateral breath sounds were auscultated and end-tidal carbon dioxide was noted on capnography, the tube was taped and secured with an air leak of approximately 18 cm of water pressure. The patient was assisted back to spontaneous respirations, and anesthesia was maintained with 1.5 % minimum alveolar concentration of ultane, 50% nitrous oxide, and 50% oxygen. A total of 10 mg of meperidine was given in divided doses. Dexamethasone, 4 mg, also was given intravenously per surgeon request. Both surgical and anesthetic courses were uneventful until completion of the case.

While emerging from general anesthesia, the Spo_2 declined from a range of 98% to 100% to a range of 84% to 87%. Attempts at assisting with ventilation were unsuccessful, as the patient was biting down on the endotracheal tube while making repeated strenuous inspiratory efforts. The inability to move air and ventilate, coupled with rapid desaturation led to pharmacological intervention. Succinylcholine, 20 mg, was given intravenously in an attempt to release the patient's bite on the endotracheal tube. Controlled ventilation was resumed with oxygen saturation remaining in the low 90s. Frothy, pink secretions were then noted filling the endotracheal tube. An 8 French suction catheter was repeatedly passed down the endotracheal tube in an attempt to clear the secretions. After several minutes of the same course, furosemide, 5 mg, was given intravenously in an attempt to pull fluid from the lungs back across the alveolar-capillary membrane. Oxygen saturation improved to the mid-90s with spontaneous ventilation, along with a significant decrease in the amount of frothy secretions. Over the next 20 minutes, the

patient's condition vastly improved and the decision was made to extubate the patient. Extubation was uneventful, and the patient was taken to the recovery room with a simple facemask at 6 L/min of oxygen. A postoperative chest radiograph revealed hazy alveolar opacity in both perihilar regions indicative of pulmonary edema. The patient remained under observation and was discharged home later that afternoon in stable condition.

Discussion

The exact incidence of NPPE is not known; however, it has been estimated that pulmonary edema develops in 11% of patients requiring active intervention for acute upper airway obstruction.[1] In the adult, risk factors identified include obesity with obstructive sleep apnea, anatomically difficult intubations, the presence of airway lesions, and patients undergoing nasal, oral or pharyngeal surgery. Young male athletes are at risk because of their ability to generate significant negative intrapleural pressures. Pediatric patients also are at risk because of their extremely compliant chest walls that can generate large negative intrapleural pressures.[2]

The development of pulmonary edema from an upper airway obstruction in animal models was first noted in 1927 by Moore and Binger,[3] but the first case in humans was not reported until 1973 by Capatanio and Kirkpatrick.[4] The generation of highly negative intrathoracic pressure produced by forceful inspiration against an obstructed airway provides the stimulus for the development of NPPE. In the case presented, the obstructed airway is represented by the patient biting the oral endotracheal tube and preventing movement of air. Inspiration against an obstructed airway accentuates the effect of negative transpulmonary pressure and results in interstitial accumulation of fluid. The associated increase in permeability of the pulmonary capillaries promotes transudation and pulmonary edema.[5]

Clinical manifestations of NPPE usually present immediately but can occur several hours later. Signs and symptoms of respiratory distress are often present, but frothy, pink sputum is the hallmark sign of NPPE. Auscultation reveals rales and, occasionally, wheezes from fluid-compressed airways. The chest radiograph typically shows diffuse interstitial and alveolar infiltrates appearing as "whited out" areas. Tachycardia, hypertension, and diaphoresis reflect sympathetic nervous stimulation.[6] When clinical signs and symptoms present, the anesthesia provider must form a differential diagnosis. Differential diagnoses for consideration include acute respiratory distress syndrome, intravascular volume excess, cardiac abnormalities, and pulmonary embolus.[2]

After the diagnosis of NPPE has been made, treatment is directed toward reversing hypoxia and decreasing the fluid volume in the lungs. Maintaining the airway and providing supplemental oxygen is usually all that is required for a positive outcome. If oxygenation does not improve in the intubated patient, positive end-expiratory pressure should be administered to promote alveolar expansion. If oxygenation does not improve in the nonintubated patient, then immediate intubation with positive pressure ventilation and positive end-expiratory pressure is necessary.[6] The use of diuretic therapy (eg, furosemide, 1 mg/kg) to remove excess intrapulmonary fluid is controversial. It is possible for the patient to be hypovolemic, and consequently diuretic therapy would only worsen the clinical condition. In the situation of the patient biting down on the endotracheal tube, a small dose of succinylcholine (0.1-0.2 mg/kg) is needed to release the patient's bite on the tube.[6]

Conclusion

At some point in his/her career, a nurse anesthetist may experience a case in which the patient manifests NPPE. The early detection of the signs of this syndrome is vital to the treatment and to patient outcome. In the case described, the signs and symptoms were clearly evident and treatment was rapidly instituted. Prompt diagnosis and treatment markedly improves patient prognosis and significantly decreases morbidity and mortality, which may occur if NPPE is left untreated.

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