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EARLY DETECTION AND TREATMENT OF NEGATIVE PRESSURE PULMONARY EDEMA IN A PEDIATRIC PATIENT : A RARE CASE REPORT.

Anaesthesiology

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ABSTRACT

Background: Negative pressure pulmonary edema (NPPE) is a rare but potentially life-threatening complication that can occur after general anesthesia. It is caused by the generation of highly negative intrathoracic pressures, leading to fluid accumulation in the lungs. **Case Presentation:** An 8-month-old male patient underwent elective correction of congenital talipes equinovarus under caudal block with sedation converted to general anesthesia. After reintubation, the patient experienced respiratory distress, rapid desaturation, and frothy pink secretions, indicative of NPPE. **Management:** The patient was reintubated, and controlled ventilation was resumed. Furosemide was administered to reduce fluid accumulation in the lungs. The patient's condition improved, and he was extubated after 6 hours. **Discussion:** NPPE is a rare complication that can occur after general anesthesia, particularly in pediatric patients. Early detection and prompt treatment are crucial to improve patient outcomes. The use of diuretics and positive pressure ventilation can help alleviate symptoms and reduce morbidity. **Conclusion:** This case highlights the importance of recognizing NPPE in pediatric patients after general anesthesia. Prompt diagnosis and treatment can significantly improve patient outcomes and reduce the risk of complications.

KEYWORDS

Extubate, negative pressure pulmonary edema, ventilation.

INTRODUCTION:

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 male who presented for elective correction of a Congenital Talipes Equino Varus. This article will discuss the occurrence, diagnosis, and treatment of NPPE during general anesthesia.

Case Summary

A Eight-Month-old male weighing 7 kg was scheduled for elective correction of a Congenital Talipus Equino Verus. A small incision was to be made at the base of the ankle. His 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. He had no known drug allergies. His surgical history was insignificant. Preoperative laboratory studies were within normal limits. Premedication consisted of midazolam, 10 mg intranasally.

The patient was brought to the operating room and monitored with standard electrocardiograph, blood pressure, pulse oximetry, and temperature equipment. An uneventful insertion of a 24-gauge peripheral intravenous catheter.

Primarily caudal block was given with 26G hypodermic needle using Injection Bupivacaine isobaric 0.25% 10 ml and Inj Tramadol 15 mg used as additive.

The patient was not compliant to the procedure in a full awake situation and was irritable hence sedation using sevoflurane was given with black face mask. We noticed a decrease in SpO2 saturation and bradycardia.

Emergency Intubation with a 3.5-cm uncuffed oral reinforced endotracheal 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. The patient was assisted back to spontaneous respirations, and anesthesia was maintained with 1.5 % minimum alveolar concentration of Sevoflurane, 50% nitrous oxide, and 50% oxygen.

While emerging from general anesthesia, the SpO2 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, 15 mg, was given intravenously and reintubation was done as a life saving measure.

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 but as a precautionary mean patient was shifted in ICU on J-R circuit and after adequate ventilation the decision was made to extubate the patient after 6 hours after ensuring spontaneous efforts of respiration and ETCO2.

The extubation was uneventful and atraumatic, and the patient was taken to the recovery room with a simple Hudson 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 on next day in stable condition.

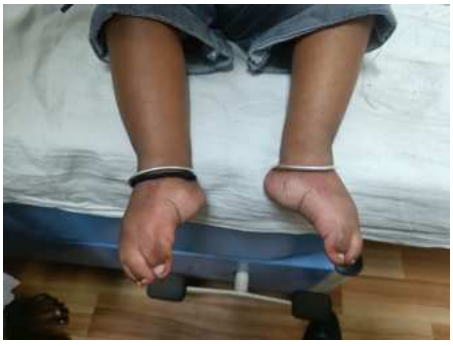

Fig. 1: CTEV Foot

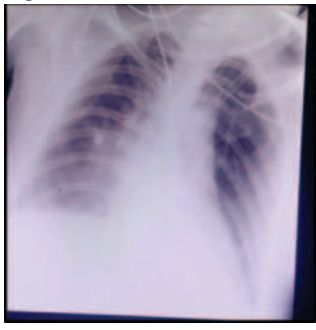

Fig.2: Chest X ray

Fig.3 : Pink frothy secretions in endotracheal tube.

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 holding breath 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.

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 holding breaths, a small dose of succinylcholine (0.1-0.2 mg/kg) is needed to relax the airways and to facilitate the reintubation.

CONCLUSION

At some point in his/her career, 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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