Negative pressure pulmonary oedema secondary to airway obstruction in an intubated infant

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We report the case of a healthy one-month-old male infant who underwent an uneventful endotracheal anaesthetic for hernia repair. During transport to the recovery room (a < 30 second trip), the endotracheal tube in the spontaneously breathing infant became obstructed, possibly due to impaction of the tip in the right main bronchus. Restoration of the airway was followed by fulminant pulmonary oedema. Several days of vigorous respiratory and pharmacologic therapy were required for resolution of the infant's respiratory distress. We review other reported cases of acute airway obstruction associated with pulmonary oedema in children and briefly describe the proposed mechanisms. The difficulties of gauging proper endotracheal tube depth in the infant are noted. This case report demonstrates the importance of continuous monitoring during patient transport to the recovery room.

As reported in children, acute airway obstruction from laryngospasm,[1,2] hanging,[2] choking,[3] anatomical anomalies,[4,9] laryngeal infections such as epiglottitis or croup,[5,6] post-extubation subglottic oedema,[6] and biting down on an endotracheal tube (ET)[7] may result in negative pressure pulmonary oedema (NPPE) in those making inspiratory efforts. Acute airway obstruction possibly due to bronchial impaction of an ET tip has not been cited, to our knowledge; and this is the youngest case of iatrogenically induced NPPE yet reported, with the most prolonged recovery.

Key words

COMPLICATIONS: airway obstruction, iatrogenic; LUNG: pulmonary oedema; PULMONARY OEDEMA: negative pressure; INTUBATION, TRACHEAL: complications; ANAESTHESIA: paediatric.

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Case report

A one-month-old healthy male infant weighing 4.3 kg was scheduled for elective repair of bilateral inguinal hernias at a paediatric teaching institution. He was born at 37 weeks gestation and had an uncomplicated perinatal course. No premedication was given, and anaesthesia was induced and maintained with halothane, nitrous oxide and oxygen. Following 4.0 mg of succinylcholine IV, oral intubation was accomplished with a 3.5 Magill style endotracheal tube. After equal bilateral breath sounds were noted, the tube was secured with tape at the corner of the mouth at a depth of 11 cm, and the head was turned to the side. Ventilation was assisted throughout the uneventful one-hour procedure. Upon completion of surgery, the intubated infant was placed in his crib while breathing spontaneously, and transported to the recovery room.

Upon arrival, he was cyanotic and bradycardic at 60 beats· min^{-1}, but moving all extremities and making inspiratory efforts. External cardiac massage was begun while ventilation of the lungs through the endotracheal tube was attempted using a modified Mapleson D circuit. Positive pressure ventilation with airway pressures up to 60 cm H_2O produced no chest excursions initially; however, the infant could be ventilated after extending the neck and pulling the endotracheal tube out as far as possible without untaping it. Cardiac massage was discontinued. Peak airway pressure was high (30 cm H_2O), and endotracheal aspiration produced copious amounts of pink frothy secretions.

A portable chest *x*-ray showed bilateral haziness (more pronounced on the right), no cardiomegaly, and the tip of the endotracheal tube in the right main bronchus. The tube was withdrawn 1.5 cm. Following 8 mEq of sodium bicarbonate, arterial blood gases measured 15 minutes after arrival were: PaO_2 = 99 mmHg (FiO_2 = 1.0), $PaCO_2$ = 54 mmHg, pH = 7.25. With ventilatory assistance (PEEP = 5 cm H_2O) and therapy consisting of furosemide for diuresis, and nitroglycerine and morphine to increase venous capacitance, arterial blood gases improved (PaO_2 = 127 mmHg, $PaCO_2$ = 27 mmHg, and

pH = 7.5) and secretions lessened. A presumptive diagnosis of NPPE secondary to airway obstruction occurring during transport was made, and the infant was transferred to the ICU.

Several hours later, PaO_2 rather suddenly dropped to 41 mmHg (FiO_2 remained at 1.0) and compliance decreased, requiring a peak inspiratory pressure of 45 cm H_2O. Throughout the first postoperative day, cyanosis of the extremities persisted and the child was intermittently hypoxemic. By the second postoperative day, hepatomegaly was apparent and a cardiac catheterization was performed to determine if the child had developed pulmonary hypertension and/or a right-to-left shunt. No significant intracardiac shunts could be demonstrated by oxygen saturation studies, although the foramen ovale was probe-patent. Mean left atrial pressure was 8 mmHg, mean pulmonary artery pressure was 21 mmHg and pulmonary vascular resistance was only slightly elevated – findings inconsistent with primary pulmonary vascular disease. Based on the prolonged clinical course and *x*-ray findings, a consulting pulmonologist suggested that external cardiac massage may have caused pulmonary contusion.

With continued respiratory support, the infant gradually improved. He was extubated on the fifth postoperative day with *x*-ray showing residual infiltrates. At discharge two days later, the chest *x*-ray was normal; his subsequent course was uneventful.

Discussion

Pulmonary oedema secondary to airway obstruction is an underappreciated entity, and unrecognized airway obstruction may be a factor in the puzzling appearance of frothy secretions in the upper airway following seizures or the resuscitation of patients who overdose on hypnotics and narcotics; and in awake infants given intramuscular succinylcholine.[8] Review articles of noncardiac pulmonary oedema written in the past decade fail to mention airway obstruction as a cause. With one exception,[9] textbook discussion has been absent or brief.

The pathophysiology is not completely understood, although the mechanism may be similar to that found in "re-expansion" pulmonary oedema, which results from the too-rapid aspiration of air or fluid from the thoracic cavity.[10] Inspiratory efforts against a total airway obstruction cause a dramatic fall in mean intrathoracic pressure, which increases the transmural pressure of all intrathoracic vascular structures. In the lungs, capillary transmural pressure increases as interstitial (pericapillary) hydrostatic pressure is lowered relative to capillary hydrostatic pressure.[11] If capillary transmural pressure rises sufficiently, the intercellular junctions stretch and capillary permeability increases.[12] Both mechanisms encourage fluid movement into the distensible interstitial space. Stretching of alveolar intercellular junctions may follow,[12] leading to alveolar flooding. Extremely high capillary transmural pressures can rupture small vessels[13] – the oedema fluid in our case and others[1,2,5,7,9] was blood-tinged. In addition, lowered intrathoracic pressure around the heart and great vessels increases transmural pressure in these structures and impedes blood flow from the intrathoracic to the extrathoracic compartments.[14] Theoretically, this increase in left ventricular afterload could increase pulmonary capillary hydrostatic pressure and contribute to fluid transudation. Following the initial mechanical insult, hypoxia-induced neural, humoral, or cellular mediators elaborated in the lung may further alter lung mechanics.[15,17]

Although oedema fluid accumulates in the interstitial space and perhaps the alveoli during airway obstruction, it may not be clinically apparent until after airway restoration, when copious secretions unexpectedly fill the ET. According to one theory,[5] relief of acute airway obstruction results in an abrupt rise in interstitial fluid pressure, allowing alveolar flooding before clearance by the lymphatics and capillaries can occur.

In the healthy child, NPPE typically resolves spontaneously[3,6] or within 24 hours after treatment is instituted.[1,2,7,9] Our patient was critically ill for two days, and required supportive respiratory therapy for three more days. Why the prolonged recovery? We speculate that severely stretched capillary linings as well as interstitial microstructures of the already oedematous lung could have sustained additional damage when external cardiac massage was applied to the infant's compliant chest wall. Pulmonary infiltrates seen on serial chest *x*-rays took almost a week to resolve.

We found only one other case report of NPPE in a healthy, normal infant who had a complication (laryngospasm) in the perianaesthetic setting.[1] In our case, we postulate that when the patient was placed in his crib, neck extension was not maintained. Flexion of the neck wedged the ET tip into the right main bronchus with the lumen occluded by bronchial mucosa, and the left main bronchus occluded by the body of the ET.

Kuhns and Poznanski[18] studied the effects of head motion on ET position in infants and found that merely turning the head to the side from a neutral position could displace the ET upward 1.2 cm and that neck flexion could advance the ET 0.5 cm toward the carina. Without *x*-ray confirmation, midtracheal ET placement with the head in a neutral position can be difficult to assess in the small infant. Auscultation is *not* a reliable technique for detecting accidental endobronchial intubation.[18] Palpating the tube tip in the suprasternal notch,[19] marking the tube at the appropriate cord level,[20] or utilizing a body

weight scale[21] have been advocated as aids. Tochen[21] measured distances from the midtrachea to the lip and determined that 10.3 cm was the mean tip-to-lip value for a 4.0 kg infant. In our 4.3 kg infant, the ET was taped at a depth of 11 cm. Even at this distance the ET tip must have been near the carina. Upward displacement from turning the head to the side probably allowed unobstructed respirations during the procedure. It is remarkable that this infant developed a life-threatening problem in such a brief period of time – less than 30 seconds. Because infants have a low functional residual capacity and a high O_2 consumption, desaturation occurs very rapidly if the airway is totally obstructed. In our patient, the struggle to breathe hastened the desaturation process and led to oedema formation as well.

During anaesthesia, we constantly monitor breath sounds, heart rate, and, currently, oxygen saturation in all cases. In order to avoid problems such as we experienced, this level of vigilance must be continued after the patient leaves the operating room.

In conclusion, midtracheal placement of the endotracheal tube tip is difficult to confirm in the small infant. During transport to the recovery room, patients must be continuously monitored: the presence of an endotracheal tube does not assure an adequate airway. A brief (< 30 second) period of total airway obstruction in a spontaneously breathing infant can result in fulminant pulmonary oedema, with pink frothy secretions appearing in the upper airway; clinical and *x*-ray evidence of pulmonary oedema may persist for several days.

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Résumé

On rapporte le cas d'un enfant mâle âgé d'un mois en bonne santé ayant subi une herniorraphie sous anesthésie générale par voie endotrachéale. Durant le transport à la salle de réveil (un transport < à 30 secondes), le tube endotrachéal fut obstrué alors que l'enfant était en respiration spontanée possiblement après enfoncement du bout du tube endotrachéal dans la bronche souche droite. La correction de la situation fut suivie par un oedème pulmonaire fulminant. Plusieurs jours de traitement respiratoire et pharmacologique ont été requis afin de corriger la détresse respiratoire de l'enfant. On revoit les histoires de cas d'une obstruction aiguë des voies aériennes associée avec l'oedème pulmonaire chez les enfants et on décrit brièvement les mécanismes proposés. Les difficultés de juger la profondeur appropriée du tube endotrachéal chez l'enfant sont notées. Cette histoire de cas démontre l'importance d'une surveillance continue lors du transport du patient en salle de réveil.