Negative pressure pulmonary edema after a tonsillectomy and adenoidectomy in a pediatric patient: Case report and review

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Negative pressure pulmonary edema (NPPE) continues to be reported as a complication of upper airway obstructions seen by anesthesia providers during induction or emergence. The majority of patients reported to have experienced NPPE have been healthy, without underlying pulmonary or cardiac disease. Factors associated with the formation of NPPE include young male patients and patients with long periods of airway obstruction. Overzealous intraoperative fluid administration and preexisting heart and lung disease also have been implicated as predisposing factors. Negative pressure pulmonary edema is the result of a marked decrease in intrathoracic pressure caused by ventilatory efforts against a closed glottis resulting in a disruption of the normal intravascular Starling mechanism, ultimately leading to the transudation of intravascular proteins and fluid into the pulmonary interstitium. The onset of NPPE is usually rapid, and without prompt recognition and intervention, the outcome can be fatal. A case of NPPE in a pediatric patient after an otherwise uncomplicated surgical procedure was observed in our institution and is described in this report.

Key words: Anesthesia, negative pressure pulmonary edema, upper airway obstruction.

Case report
A 19-month-old, 11-kg boy was scheduled to undergo a tonsillectomy and adenoidectomy. His medical history included recurrent croup-like infections, and he also had been recently discharged from a local area hospital after evaluation to rule out meningitis. Symptoms at that time included a stiff neck, headache, frequent crying for 2 days, and an inability to ambulate without assistance. Records obtained from that hospital revealed negative cerebrospinal fluid culture results. The patient's surgical history included bilateral myringotomies with tube placement and bilateral inguinal hernia repairs without reported problems. The patient was not taking any medications and had no known drug allergies; the physical assessment was unremarkable. The preinduction heart rate was 162 beats per minute; blood pressure, 98/52 mm Hg; and respiratory rate, in the 30s.

An intravenous (IV) infusion of lactated Ringer’s was started after performing a mask induction of anesthesia with sevoflurane, 30% oxygen, and 70% nitrous oxide. The trachea was intubated easily with a 4.5 uncuffed endotracheal tube (ETT), and a leak was noted at less than 20 cm of water. Anesthesia was maintained with 100% oxygen and isoflurane. The heart rate remained between 160 and 170 beats per minute, the systolic blood pressure between 96 and 120 mm Hg, and the diastolic blood pressure between 56 and 78 mm Hg throughout surgery. The surgery proceeded without difficulty. Bupivacaine 0.5% with epinephrine 1:200,000 (exact amount unclear) was injected into the tonsillar fossae by
the surgeon to adequately infiltrate the area, and 10 mg of IV meperidine was administered for post-operative pain relief. The patient received a total of 350 mL of lactated Ringer’s during the 60-minute procedure. His normal maintenance intravenous fluid (IVF) requirement per hour was calculated to be 42 mL, and the deficit from an 8-hour overnight fast was estimated to be 336 mL. The estimated blood loss was minimal (<50 mL).

The patient was extubated during deep anesthesia and immediately turned on the right side. The patient began moving his extremities and crying but was noted to have difficulty with phonation. Stridor and retractions ensued and the patient began moving his extremities and talking. Chest auscultation performed in the operating room revealed bilateral infiltrates indicative of pulmonary edema; 5 mg of IV furosemide was administered. Soon after, pink frothy sputum was noted from the oropharynx. The SpO2 was maintained at more than 90% but could be maintained at more than 90% only by using 1.0 FIO2 by mask. The heart rate and blood pressure were only slightly elevated. Hydrocortisone sodium succinate, 50 mg IV, was given to treat the laryngeal edema evidenced by stridor, and a laryngoscopy was performed. There was no evidence of obstruction due to blood clots or ongoing bleeding. The vocal cords could not be visualized, as the child was moving about vigorously. Chest auscultation demonstrated diminished breath sounds on the left side. A chest radiograph performed in the operating room revealed bilateral infiltrates indicative of pulmonary edema; 5 mg of IV furosemide was administered. Soon after, pink frothy sputum was noted from the oropharynx. The SpO2 was maintained at more than 90% with assistance throughout the event. The patient’s condition began to improve, and he was transferred to the pediatric intensive care unit for observation approximately 45 minutes after completion of the tonsillectomy and adenoidectomy without evidence of respiratory distress. The SpO2 was 96% to 100% during administration of 2 L of oxygen delivered via nasal cannula. The total IVF administered was 550 mL of lactated Ringer’s.

The admission note to the pediatric intensive care unit reported rales in the right lower lobe without stridor or wheezes with symmetrical expansion of the chest. Supplemental oxygen was discontinued gradually 4 hours after admission when further auscultation of the chest revealed the resolution of rales. A chest radiograph performed the next morning demonstrated almost complete resolution of the bilateral infiltrates. The patient was discharged home without further complications.

Discussion

The negative pressure pulmonary edema (NPPE) resulted from a partial upper airway obstruction of unknown cause. Since the patient had a history of recurrent croup-like infections, the cause of the upper airway obstruction may have been preexisting laryngeal edema. Another possibility was a partial laryngospasm, attributable to secretions pooling in the oropharynx. Furthermore, vocal cord paralysis from the injection of bupivacaine into the tonsillar fossae may have contributed to the subsequent upper airway obstruction. Considering the anatomical location of the nerves relative to the pharyngeal space, it is apparent how closely the vagus and the hypoglossal nerve travel in relation to the pharyngeal space (Figure 1). Depending on the amount of solution and the depth of the injection, the local anesthetic could diffuse to the nerves or be inadvertently injected directly into the sheath that contains the nerves. In the pediatric population, the risk may be greater than in adults because children’s shorter necks decrease the distance between the nerves and the pharyngeal space. This may have predisposed the child in the present case to a unilateral recurrent laryngeal nerve block resulting in the stridor and difficulty with phonation. In addition, the hypoglossal nerve may have been blocked, causing a soft tissue obstruction due to the loss of motor function to the tongue and the upper pharyngeal muscles. Patient positioning also may be a contributing factor that is associated with accidental nerve blocks after injection of local anesthetics into the lateral pharyngeal space. Placing the patient in the lateral head-down position to prevent pooling of secretions theoretically could promote cephalad spread of the local anesthetic solution.

Considering the rapid onset and recovery of the upper airway obstruction, it is unlikely the patient experienced an accidental nerve block. If the local anesthetic reached the nerve by diffusion, the onset more likely would have been gradual. If bupivacaine had been injected directly into the sheath, the block would have been prolonged. Furthermore, the injection was relatively superficial; accidental nerve blocks are more common with deeper injections, such as those performed for glossopharyngeal nerve blocks. Although the evidence does not support the expected clinical manifestations, the possibility cannot be completely ruled out and deserves some consideration.
Pathophysiology of NPPE

In the normal physiologic state, intrapulmonary pressures are equal to that of the atmosphere. This, balanced with the intrapleural pressure and collapsing force of the lung, keeps the lungs expanded in the thoracic cage (Figure 2). According to Boyle’s law, as the volume of a container increases, pressure decreases; thus, expansion of the chest wall causes the intrapleural pressure to decrease and become subatmospheric with resultant inward airflow. At the level of the respiratory membrane, the pulmonary interstitial space between the capillary and the alveolus is ordinarily relatively dry, consistent with the Starling hypothesis. The major forces maintaining this balance include the pulmonary capillary osmotic force promoting fluid movement into the capillary, a relatively weak hydrostatic pressure in the capillary opposing the inward movement, and a negative interstitial pressure working concurrently with the interstitial colloid osmotic pressure drawing fluid into the interstitial space. Lymphatic capillaries drain the interstitial space of any fluid in excess of that managed by the Starling mechanism (Figure 3).

Three major factors contribute to the transudation of fluid into the interstitial space after the relief of an upper airway obstruction. As inspiration occurs against a closed epiglottis (Mueller maneuver), intrapleural pressures become subatmospheric, which in turn makes the interstitial space markedly more negative, causing the transudation of intravascular proteins and fluids from the pulmonary capillary into the interstitial space. In addition, hypoxia leads to pulmonary vascular constriction, which increases
pressure in the pulmonary capillaries. Concurrently, central nervous system-mediated α-adrenergic stimulation leads to an increase in total peripheral resistance. This systemic vasoconstriction leads to an increase in right ventricular preload, pulmonary capillary volume increases, and fluid leaks into the interstitial space. Left ventricular afterload also increases leading to decreased left ventricular ejection (Figure 4). As the interstitial space is flooded with fluid, compensatory mechanisms are overwhelmed, and eventually the fluid penetrates and floods the alveoli resulting in pulmonary edema. Symptoms usually are seen only after the relief of the obstruction, due to the intermittent positive pressure created with expiration against the closed glottis (Valsalva maneuver). This positive pressure opposes the negative pressure until the obstruction is removed.

Clinical manifestations of NPPE

The onset of NPPE is usually immediate but can occur up to several hours after an obstruction. Signs and symptoms of respiratory distress are often present. Expectoration of pink frothy sputum typically is seen as the alveoli are flooded with fluid. The patient also may become hypoxic; whether hypoxia occurs depends on the severity of the condition. Auscultation of the chest reveals rales and possibly wheezing as the peripheral airways are compressed by excess fluid. The chest radiograph frequently reveals diffuse interstitial and, depending on the severity of the condition, possibly alveolar infiltrates appearing as “whited out” areas. Signs of sympathetic stress stimulation, such as tachycardia, hypertension, and diaphoresis, also will be apparent.

Prevention of NPPE

Attempting to prevent or treat any upper airway obstruction is the primary measure opposing the development of NPPE. Some upper airway obstructions common to the pediatric population in the perioperative period are postextubation laryngeal edema and laryngospasm. Avoiding laryngeal edema in the pediatric patient is crucial because a small degree of edema in a pediatric patient may lead to obstruction, whereas in an adult, edema may result only in hoarseness. Measures to prevent laryngeal edema include ensuring a positive leak around the ETT at less than 15 to 20 cm of water, delaying surgery if crouplike symptoms are present, and attempting to avoid excessive bucking on the ETT on emergence from anesthesia.

If signs and symptoms of croup develop (such as inspiratory stridor, a barking cough, hoarseness, or retraction) during the perioperative period, treatment must be implemented promptly. Cool humidified oxygen should be administered. Racemic epinephrine (0.5 mL of a 2.25% solution nebulized in 2.5 mL) may be used for its vasoconstrictive properties. Steroids may be administered (eg, dexamethasone, 0.1-0.5 mg/kg), although immediate relief may not be seen due to the delayed onset of action. If symptoms are severe or progress, reintubation may be necessary with a smaller ETT.

Laryngospasm is a common occurrence in the pediatric patient undergoing a tonsillectomy and is the most common cause of NPPE secondary to an upper airway obstruction. The mechanism involved in its onset is sensory stimulation of the superior laryngeal nerves with resultant spasm of the true vocal cords. It has been suggested that prevention of laryngospasm on emergence may be accomplished through extubation during deep anesthesia when airway reflexes are absent or by extubating in an awake state, thereby avoiding stimulation in a light depth of anesthesia when laryng-
gospasm is most common. Patient whose eyes are deviated and pupils are dilated and who hold their breath may be more susceptible to a laryngospasm. If extubating a patient who is awake, the patient should have spontaneous movement of all extremities, spontaneous eye opening, and a regular respiratory rate and rhythm.

Careful suctioning of the oropharynx before extubation is essential. This removes blood and secretions, thus preventing them from falling on the glottic structures, potentially resulting in stimulation of the superior laryngeal nerve. When extubating the patient, positive pressure should be administered while removing the ETT just as the patient is about to exhale. This fills the lungs with oxygen and acts as a reserve in case of a laryngospasm. Stretch receptors in the lungs also are stimulated, which induces forceful exhalation as the ETT is removed (ie, the Hering-Breuer reflex), aiding in clearing blood or secretions that may be in proximity to the laryngeal structures. The administration of IV lidocaine, 2 mg/kg, 1 minute before extubation also has been shown to decrease the incidence and severity of laryngospasms.

If laryngospasm occurs, treatment must be instituted immediately. If possible, remove the stimulus by suctioning secretions from the oropharynx. The administration of 100% oxygen with continuous positive pressure may be enough to force the vocal cords open. If this is accomplished, continued positive inspiratory assistance via manual insufflation concurrent with the patient’s spontaneous ventilatory efforts will effectively keep the vocal cords open until the crisis is resolved. Pushing the mandible forward also may be necessary as this maneuver displaces the tongue, the hyoid bone, and the epiglottic cartilage attached to it. This not only widens the pharynx, but also opens the entrance to the larynx. If the laryngospasm is not relieved in approximately 30 seconds, succinylcholine should be administered. Some advocate a subapneic dose of succinylcholine (0.1-0.2 mg/kg); however, if the patient’s condition is deteriorating, it may be efficacious to administer an intubating dose (1-2 mg/kg), reintubate the patient, and extubate once the patient is awake with stable pulmonary mechanics and optimal oxygen saturation parameters.

The judicious administration of IVF also will decrease the risk of developing NPPE after the relief of an upper airway obstruction. Adherence to an appropriately conceived IVF plan is prudent for all patients undergoing surgery with or without evidence of pulmonary or cardiac disease.

Treatment of NPPE

With overt evidence of pulmonary edema, treatment is directed toward reversing hypoxia and decreasing the fluid volume in the lungs. Fortunately, in almost 50% of the patients, supplemental oxygen and maintenance of a patent airway are usually all that is required to maintain adequate oxygenation. If oxygenation does not improve with the administration of high inspired concentrations of oxygen, the patient must be reintubated and positive pressure ventilation with positive end-expiratory pressure initiated. End-expiratory inflation of the alveoli is promoted by positive end-expiratory pressure, thus maintaining and increasing the patient’s functional residual capacity. This facilitates an improved ventilation/perfusion ratio and decreases intrapulmonary shunting. The administration of diuretics (eg, furosemide, 1 mg/kg) to effectively remove excess intrapulmonary fluid has been advocated by some; however, this is controversial and may be reserved for patients with marked hypervolemia. Even when mechanical ventilatory support is required, NPPE usually will resolve within 24 hours. The patient described in the present case report was weaned from supplemental oxygen within 4 hours, monitored in the pediatric intensive care unit overnight, and sent home without further sequelae.

Conclusion

Although NPPE usually resolves spontaneously with supportive treatment, the rapidity of onset and severity of the disease ultimately may lead to death if the disease is not recognized promptly and immediate intervention instituted. Thus, it must be emphasized that NPPE can occur in any patient, particularly patients with potentially obstructive processes of the upper airway. Vigilant anesthesia care warrants recognition of the patients at risk for NPPE, with the institution of measures during provision of anesthesia to prevent the occurrence of upper airway obstructions.

REFERENCES


AUTHORS

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