Unpredicted Sudden Onset of Postobstructive Pulmonary Edema Complicating Adenotonsillectomy in a Child

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We present the case of a 4-yr-old child who experienced pulmonary edema during adenotonsillectomy. The pulmonary edema occurred unexpectedly around 50 minutes into the operation. We suggest that postobstructive pulmonary edema can occur soon or sometime after the resolution of a chronic airway obstruction. (Korean J Anesthesiol 2004; 46: S 1–S 3)

Key Words: adenotonsillectomy, pediatric, postobstructive pulmonary edema.

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INTRODUCTION

Adenotonsillectomy is one of the most common operations being performed in children. Fortunately, pulmonary edema is an infrequent perioperative complication, but pulmonary edema complicating adenotonsillectomy has been reported to occur mainly during the induction of anesthesia\(^1\)\(^2\) or immediately after extubation of endotracheal tube.\(^3\) The etiology of pulmonary edema in these cases is presumed to be the sudden relief of chronic airway obstruction\(^1\)\(^2\) or acute upper airway obstruction (UAO), such as post-extubation laryngospasm and laryngeal edema.\(^3\)\(^4\)

The purpose of this paper is to report the occurrence of pulmonary edema during the adenotonsillectomy, which is both rare and unpredictable, especially at 40 to 50 minutes after endotracheal intubation. In addition, we describe its clinical course.

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CASE REPORT

The patient was a 4-year-old boy, weighing 15 kg, who was presented to the operating room for adenotonsillectomy. His chief complaint was severe snoring for 2 years. He had a history of recurrent upper and lower respiratory infections, frequent tonsillitis, sore throat, and open mouth breathing. Preoperative laboratory studies, such as complete blood count and urinalysis, ECG, and chest roentgenogram, were within normal limits. He was not taking any medications and had no known drug allergies.

He was a thin, active boy and had no other anomalies. His tonsils were markedly enlarged (grade 3+), but tonsillar exudates were not shown. He had suffered an upper respiratory infection 4 weeks previously, but symptoms had subsided. Because his elective schedule for tonsillectomy was delayed several times due to frequent upper respiratory infections and, recently, his general condition was very good except a little coarse lung sound, we decided to anesthetize him for getting an adenotonsillectomy. He had a normal sinus rhythm and no murmur.

On arrival in the operating room, ECG electrodes, an automatic blood pressure cuff, and a pulse oximeter finger probe were applied. His initial heart rate was 135 beats per minute; blood pressure, 110/70 mmHg; respiratory rate, in the 20s per minute; oxygen saturation breathing room air, 98%. Anesthesia was induced with pentothal sodium (75 mg), 40% oxygen, 60% nitrous oxide, and sevoflurane. Transient apnea and a mild ventilation disorder after injection of pentothal sodium were overcome by gentle assisted ventilation without any difficulty. The trachea was intubated easily with a 4.5-mm ID cuffed RAE endotracheal tube after the intravenous administration of vecuronium bromide (1.5 mg). After intubation, increased coarse breathing sounds and ronchi were heard over both lung fields. We removed secretions from both lung fields 2 to 3 times before the operation began, and his lung sounds became clear. Anesthesia was maintained with 50% nitrous
The pediatric pulmonologist was consulted in the SICU. There were no aggravating problems in the ABGA, SpO$_2$, vital signs, or chest radiograph. The next morning after about 24 hours postoperatively, a chest radiograph demonstrated complete resolution of the alveolar infiltrates (Fig. 1B), the patient was extubated and discharged to a general ward without further complications in several hours.

**DISCUSSION**

It is well known that perioperative pulmonary edema is uncommon, and that it may be fatal without prompt recognition and intervention. Adenotonsillectomy is one of the most common operations in the pediatric patients. Pulmonary edema is rare, but is nevertheless a possible complication associated with adenotonsillectomy.\(^5\) McGowan et al.\(^7\) noted that adenotonsillectomy itself could be a significant risk for children with UAO who were born prematurely and had evidence of abnormal facial development or respiratory distress preoperatively.

Postobstructive pulmonary edema shows as a sudden onset of pulmonary edema following upper airway obstruction. In 1977, Osawlt et al.\(^8\) reported pulmonary edema after acute airway obstruction, and in 1980, Galvis\(^9\) suggested that pulmonary edema often occurred after relief of upper airway obstruction. To explain the pathophysiology of these episodes, it had been suggested that there are two different types of postobstructive pulmonary edema.\(^10,11\) In the case of adenotonsillectomy, both types of postobstructive pulmonary edema have been reported; the laryngeal edema or postextubation laryngospasm after adenotonsillectomy and the relief of chronic upper airway obstruction after endotracheal intubation caused pulmonary edema.

In this patient, pulmonary edema was developed slowly during operation at around 50 minutes after intubation, not immediately.

oxide, 50% oxygen, and 1-2% sevoflurane. Immediately following induction, heart rate was 140 beats per minute; blood pressure, 100/50 mmHg; controlled respiratory rate, 20 per minute; peak airway pressure, 20 mmHg. From 15 minutes after induction, the operation procedure was proceeded without difficulty and uneventfully; oxygen saturation as measured by pulse oximetry and end tidal CO$_2$ were maintained at 100% and 37 to 40 mmHg, respectively. At around 30 minutes from the start of operation, peak airway pressure began to increase from 20 cmH$_2$O to 30 cmH$_2$O, this increased airway pressure was presumed to be due to endobronchial secretions. The trachea was intermittently suctioned for the duration of the operation was lasting. Although repeated suction was performed, the airway pressure did not return to normal limits. Some pinky secretions were observed stuck on the tip of suction catheter. Finally, a moderate amount of pink, frothy fluid began to exude from the endotracheal tube. Because the second tonsil was being excised, the surgery was continued and was completed in about 20 minutes; endotracheal suctioning were performed frequently during the operation. When the pink, frothy secretions were first detected, arterial blood gas analysis (ABGA) revealed pH 7.28, PaO$_2$ 209 mmHg, PaCO$_2$ 44 mmHg, and base excess 7.0 mEq/L (FiO$_2$ 0.5). He was given intravenously fentanyl 20 g, midazolam 1 mg, dexamethasone 2.5 mg, and sodium bicarbonate 9 mEq. He was treated with controlled ventilation using 100% oxygen and 5 cmH$_2$O positive end-expiratory pressure (PEEP) during operation, and then manually ventilated with positive pressure until transfer to the surgical intensive care unit (SICU). Portable chest radiograph showed diffuse alveolar infiltrates over the bilateral lung fields with no cardiomegaly (Fig. 1A). The SpO$_2$ was maintained at more than 95% throughout the operation. The patient’s condition began to improve slowly and he was transferred to the SICU, where he required ventilatory support for 24 hours.

**Fig. 1.** (A) Portable chest radiograph shows bilateral vascular engorgement and centralized lung haziness with no cardiomegaly, at around 60 minutes after endotracheal intubation. (B) Chest radiograph, at 24 hours after admission to SICU, shows complete resolution of the pulmonary edema.
after intubation. Because the patient had a history of chronic upper airway obstruction, including severe snoring and open mouth breathing, the relief of chronic upper airway obstruction with endotracheal intubation was thought to be a main cause of his pulmonary edema. As the amount of fluids infused intravenously during operation was only 150 ml, fluid overloading was considered unlikely. On physical examination and by preoperative laboratory findings, no specific cardiac or pulmonary problems were found that might have been associated with the pulmonary edema. We concluded that the pulmonary edema in this case was a type II postobstructive pulmonary edema, as classified by Van Kooi and Gargiulo.11

In type I postobstructive pulmonary edema, acute airway obstruction leads to a decrease in the intrathoracic pressure and a subsequent increase in venous return. The increased pulmonary blood volume causes an increase in hydrostatic pressure and transudation from the capillary bed to the interstitium. The physical damage to capillaries and the capillary distention caused by the extreme negative intrathoracic pressure increase the membrane permeability, which may be a factor of postobstructive pulmonary edema.

The pathophysiology of Type II postobstructive pulmonary edema is not clear so far. Chronic obstructing lesions may produce an intrinsic positive end-expiratory pressure and increased end-expiratory lung volume. As soon as the chronic obstruction is relieved by intubation or adenotonsillectomy, a sudden decrease in intrathoracic pressure, a subsequent increase in venous return and pulmonary blood volume, may cause interstitial fluid transudation from the vascular bed, and thereby pulmonary edema.

In the patients with postobstructive pulmonary edema, oxygen saturation and breathing sounds decrease, CO2 retention increases, and pink, frothy sputum from the endotracheal tube increases. Treatment is only supportive, but early detection is important, in which case a positive treatment result should produce a good outcome. Close monitoring of SpO2 and vital signs is needed with oxygen supplementation in the operating room and in intensive care unit. Additional treatment consists of diuretics, morphine, steroids, and mechanical ventilation with PEEP and continuous positive airway pressure, if necessary.

Everyday we have anesthetized, and will continue to anesthetize children with more severe signs of chronic upper airway obstruction showing severe snoring, frequent tonsillitis, and open mouth breathing. Although the incidence of postobstructive pulmonary edema during adenotonsillectomy is very low, these patients need to be monitored carefully throughout the operation and a prompt treatment kit should be at hand to avoid a fatal outcome. Because the occurrence of postobstructive pulmonary edema after intubation could be delayed as this case and it is difficult to predict the occurrence, the monitoring of the airway pressures and oxygenation parameters in these patients is essential throughout the operation.

REFERENCES