

Post-Operative Pulmonary Edema After Modified Radical Mastoidectomy -A Case Report

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Abstract

Pulmonary edema is a potentially life-threatening complication of acute airway obstruction. The predominant mechanism is forced inspiration against a closed or occluded glottis. Forceful attempts to inhale against an obstruction create highly negative intrathoracic pressure, which causes increased venous return, decreased cardiac output and fluid transudation into the alveolar space. Post anesthetic laryngospasm has been implicated as the most frequent cause of this syndrome in adults. We present case scenario of acute post-operative pulmonary edema in a young healthy adult male after extubation following general anesthesia for Modified radical mastoidectomy.

INTRODUCTION

Pulmonary edema is a potentially life-threatening complication of acute airway obstruction. It develops rapidly, without warning, in persons who are otherwise well. Negative Pressure Pulmonary Edema (NPPE) was first demonstrated in 1927 by RL Moore¹ in spontaneously breathing dogs exposed to resistive load. The first description of the pathophysiological correlation between creation of negative pressure and the development of pulmonary edema was in 1942 by Warren et al². The first case report of post obstruction pulmonary edema (POPE) was reported by Oswalt et al³. Following these initial case reports, there have been several reports.^{2,4}

CASE REPORT

A 19 years old young adult, laborer by occupation, was scheduled for elective modified radical mastoidectomy. A day before planned surgery complete physical examination was done and no remarkable finding was noted. He denied any history suggestive of pulmonary and cardiac disease except history of mild cough one week before, which had resolved. Routine investigations like Hb, BT, CT and urine examinations were done and were within normal limits.

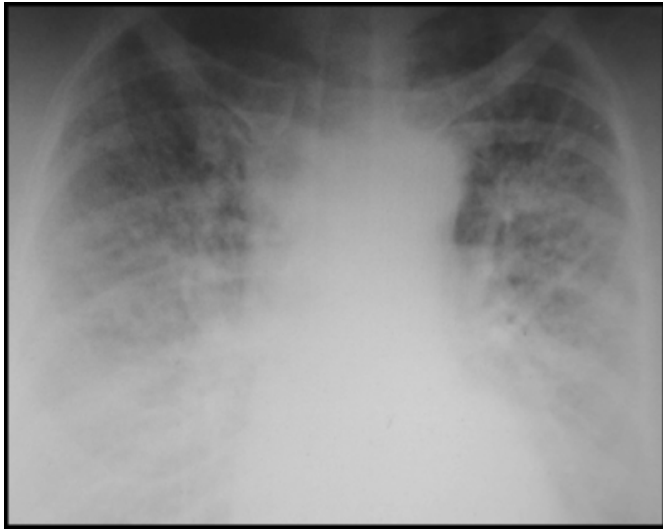
He was kept fasting overnight and tab alprazolam 0.25mg orally at bedtime and two hours prior to surgery with a sip of water was given as premedication. On arrival in the operating room an intravenous line was secured and standard monitoring (ECG, SPO2, end tidal carbon dioxide,

automated blood pressure) was instituted. Induction of anesthesia was done with intravenous thiopentone, fentanyl and vecuronium and he was intubated with 8.5mm cuffed endotracheal tube easily. Anaesthesia was maintained with oxygen, nitrous oxide and isoflurane. Intra-operatively the patient received 1200ml of Ringer Lactate. Surgery preceded without any complication and ended three hours later. Neuromuscular block was reversed with intravenous glycopyrrolate and neostigmine. Pharynx was suctioned and when his respiration became regular and adequate, he was extubated.

After 4-5 minutes of extubation, the patient started having difficulty in respiration and became very restless. His oxygen saturation started decreasing rapidly. Immediately, bag mask ventilation was started but we were not able to ventilate the patient adequately. His oxygen saturation was decreasing precipitously so intravenous succinylcholine was given and he was intubated and IPPV was started. His oxygen saturation improved to 86-91% on 100% oxygen. Auscultation of chest revealed bilateral coarse crepitations and regular cardiac rhythm. Endotracheal suction yielded pink fluid. ABG was done and it revealed hypoxia and hypercarbia (pO₂-59, pCO₂-51 and pH of 7.3). Chest X-ray was obtained which showed bilateral pulmonary infiltrates. (Fig 1).

Figure 1

Figure 1: Chest X-ray PA view showing bilateral pulmonary infiltrates



Based on clinical condition, presumptive diagnosis of non-cardiogenic pulmonary edema was made. Patient was propped up and IPPV was continued. Intravenous Furosemide 60 mg and morphine sulphate three mg was given. Nebulization with salbutamol was done. With these interventions his saturation improved, and he was extubated after about one hour. He was kept in propped-up position. Within 3-4 hours his condition improved significantly. His oxygen saturation improved to 97-100% on face mask (FIO_2 of 0.4), Repeat ABG revealed pO_2 of 93 and pCO_2 of 42.

DISCUSSION

Post obstruction pulmonary edema (POPE) is a kind of non-cardiogenic pulmonary edema⁵. Two separate types have been described in the literature. Type I follows an acute airway obstruction, such as laryngospasm⁶. The overall incidence of laryngospasm with general anesthesia is estimated to be 9 in 1000 patients in general population and higher in the otolaryngology patients⁷. Type –II occurs after relieving a chronic upper airway obstruction such as adenotonsillar hypertrophy.^{8,9}

The pathogenesis of post obstruction pulmonary edema is multifactorial.^{4,10} Forceful attempts to inhale against an obstruction create highly negative intrathoracic pressure, which causes increased venous return, decreased cardiac output and fluid transudation into the alveolar space. The importance of vigorous inspiratory effort in POPE is supported by the apparent increase in susceptibility to this condition in young athletic men who, because of their chest wall musculature, are able to generate extremely high

negative inspiratory pressures^{11,12}.

Clinically, the sequence of events clearly occurs in two stages. The first stage is the development of laryngospasm with airway obstruction. Respiratory efforts against the closed glottis then lead to generation of large negative intrathoracic pressures. The second stage occurs with relief of the airway obstruction and subsequent development of pulmonary edema. The edema does not become evident until after relief of the obstruction.¹³

Post obstruction pulmonary edema requires rapid intervention and may be confused with other causes of postoperative respiratory distress. Although symptoms usually develop within one hour of the precipitating event, delayed onsets have been reported.¹⁴ The presence of agitation, tachypnea, tachycardia, frothy pink pulmonary secretions, rales and progressive oxygen desaturation suggests the diagnosis of POPE in the appropriate setting. Chest radiograph findings of pulmonary edema support the diagnosis.

Other causes of pulmonary edema should be considered. The absence of gastric contents in pulmonary secretions, a history of normal cardiac function and, particularly, the occurrence of such symptoms in a vigorous young person makes the diagnosis of POPE more likely. Fortunately, the prognosis for the patient with POPE is good. Most patients respond rapidly to conservative measures, including oropharyngeal suction, supplemental oxygen, and diuretic therapy.¹⁵

This condition usually resolves rapidly but Supportive mechanical ventilation may be required in those patients refractory to conservative treatment. If intubation and ventilation are required, however, it is usually not for a period longer than 24 hr. Although ventilatory support may be essential for maintaining adequate oxygenation during the acute stage, non-cardiogenic pulmonary edema is often a self limiting process, with the alveolar capillary defect resolving as the cells recover from the initial injury.¹⁶

CONCLUSION

Post-operative pulmonary edema is not very uncommon in anesthesia practice. Most common cause of this condition is airways obstruction. Removal of airways obstruction and maintenance of adequate ventilation and oxygenation is most important step in management of post-operative pulmonary edema. Proper vigilance, early diagnosis and timely intervention contribute to the successful management.

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