Laryngospasm-Induced Pulmonary Edema

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Citation

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Abstract

Pulmonary edema due to laryngospasm is an uncommon and potentially serious complication of patients undergoing general anesthesia for different surgical procedures. We report a case of a 24-year-old female patient who enrolled surgery for septorhinoplasty. The patient developed pulmonary edema immediately after extubation. After reintubation and observation in the intensive care unit the patient extubated and discharged from hospital without any respiratuar problem.

INTRODUCTION

Laryngospasm induced pulmonary edema after endotracheal extubation is an uncommon complication that occurs in approximately 0.1% of patients ([1]). The propose mechanism is the generation of high negative pressures during respiratory effort associated with glottis closure and larngospasm leading to pulmonary edema ([2,3]). Because of these event prolonged endotracheal intubation and intensive care support may be necessitate.

We report a case of pulmonary edema after relief of airway obstruction due to laringospasm following extubation of the trachea.

CASE REPORT

A-24-year old woman with no previous medical history, known drug usage and known drug allergy enrolled surgery for septorhinoplasty. The physical examination and laboratory tests were normal. Preoperative electrocardiograph and chest radiogram were normal. Septorhinoplasty was performed under general anesthesia. I.v. propofol, fentanyl, vecuronium were used for anesthesia and no trauma occurred during intubation. Anesthesia and the surgical procedure were uneventful. The procedure lasted 120 minutes and a total of 1100 ml isotonic solution. Immediately after extubation, the patient developed laryngospasm and became agitated. Respiratory distress, tachypnea, cyanosis, accessorie muscle utilization and decrease in peripheral oxygen saturation were observed. After administration of succinycholine chloride and relief of the laryngospasm, the trachea was reintubated. A small amount of pink fluid was suctioned from the endotracheal tube. A chest radiograph after reintubation showed pulmonary edema. At this time the patient was treated with 100% oxygen and furasemide was given. The patient was taken to the intensive care unit for observation. The chest radiograph-12 hours after episode of laryngospasm- revealed complete resolution of the pulmonary edema. Arteriel blood gas examination was normal. Shortly thereafter the patient was weaned from the ventilator and extubated. After 3 days of observation the patient was discharged from the hospital without any respiratuar problem.

DISCUSSION

Negative pressure pulmonary edema is an infrequent event after extubation in patients undergoing surgical procedures under general anesthesia ([4,5]). The mechanism whereby upper airway obstruction precipates pulmonary edema is not known with certainty. The initiating event is usually a vigorous inspiratory effort against a closed glottis, resulting in markedly negative pleural pressures ([6,7]). Typical normal pleural inspiratory pressures range from -2 to -5 cm of water, whereas during severe episodes of upper airway obstruction, negative pressures as extreme as 100 cm of water have been reported ([8]). This results in negative transpulmonary pressure that leads to transudation of fluid from the pulmonary capillaries into the interstitium. The fluid accumulates so rapidly that the lymphatics are unable to clear the fluid from the interstitial space. This results in interstitial and alveolar edema ([9]). Additionaly, the hypoxic state may contribute to pulmonary and peripheral vasocontriction because of adrenergic release which increases both right and left ventricular pressures and output, thus increasing the transmural gradient in the pulmonary capillary bed. Finally, altered capillary permeability, capillary leak and distruption by either negative pressures or extreme hypoxia have been proposed as secondary adverse consequences ([10]).

It has been observed that pulmonary edema usually develops shortly or immediately after extubation, although delayed manifestation has also been reported as much as 1 hour after extubation. One possible explanation fort his observation is that expiration against the obstruction produces an effect similar to positive end expiratuar pressure ventilation, which increases the lung volume and thereby masks the presence of edema. With the decrease in lung volume after reliefe of obstruction, pulmonary edema becomes clinically and radiographically more apparent ([11]).

Patients at increased risk to develop upper airway obstruction may be at increased risk to laryngospasm induced pulmonary edema. Obesity, short and thick neck, history of sleep apnea and nasopharygeal soft tissue disorders were all at increased risk ([12]).

In the present case, laryngospasm induced pulmonary edema developed immediately after extubation. The patient was so young and athletic and capable of generating a vigorous inspiratory effort resulting in high negative pleural pressures leading to pulmonary edema. Respiratory distress, tachypnea, cyanosis, accessory muscle utilization and decrease in peripheral oxygen saturation were observed in this patient. These signs were nonspecific and they may not be so useful for identification.

Radiological studies may aid in the diagnosis of laryngospasm induced pulmonary edema. Plain chest films and CT scans may further exclude other causes of acute respiratory insufficiency in the immadiate postoperative period such as aspiration pneumonia and pulmonary thromboembolism.

In this case report reintubation was performed and mechanical ventilation therapy was used only one day but in some cases prolonged periods of mechanical ventilation and high concentrations of oxygen delivery must be necessary ([12]).

Laryngospasm induced pulmonary edema is a self-limited condition with excellent prognosis and relatively simple management but it may also result in significant prolonged hospital stay and the necessity of intensive care unit.

In conclusion, the anesthesists must be aware of laryngospasm-induced pulmonary edema. Marked decrease in oxygen saturation shortly after extubation should stimulate a high index of suspicion for laryngospasm induced pulmonary edema.

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References

1. Tami TA, Chu F, Wildes TO, Kaplan M. Pulmonary edema and acute upper airway obstruction. Laryngoscope. 1986; 96: 506-509
2. Barin ES, Stevenson IF, Donnely GL. Pulmonary edema following acute upper airway obstruction. Anaesth Intensive Care 1986; 14: 54-57
3. Weissman C, Damask MC, Yang J. Non cardiogenic pulmonary edema following laryngeal obstruction. Anesthesiology 1984; 60: 163-165
4. Wilms D, Shure D. Pulmonary edema due to upper airway obstruction. Chest 1998; 94: 1090-1092
5. Homes JR, Hensinger RN, Wojtys EW. Postoperative pulmonary edema in young athletic adults. Am J Sports Med. 1991; 19: 365-371
6. Broccard AF, Liaudet L, Aubert JD, Schnyder P, Schaller MD. Negative pressure post-tracheal extubation alveolar hemorrhage. Anesth Analg 2001; 92: 273-275
7. Dolinski SY, MacGregor DA, Scuderi PE. Pulmonary hemorhage associated with negative pressure pulmonary edema. Anesthesiology 2000; 93: 888-890
8. Timby J, Reed C, Zeilender S, Glauser FL. Mechanical causes of pulmonary edema. Chest 1990; 98: 973-979
9. Jackson FN, Rowland V, Corssen G. Laryngospasm induced pulmonary edema. Chest 1980; 78: 819-821
10. Matsuura Y, Nomimura T, Muramaki H, Matsushima T, Kakehashi M, Kajihara H. Clinical analysis of reexpansion pulmonary edema. Chest 1991; 100: 1562-1566
11. Sofer S, Bar-Ziv J, Scharf SM. Pulmonary edema following relief of upper airway obstruction. Chest 1984; 86: 401-403
12. Cascade PN, Alexander GD, Mackie DS. Negative pressure pulmonary edema after endotracheal intubation. Radiology 1993; 186: 671-675

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