NEGATIVE PRESSURE PULMONARY EDEMA AFTER EMERGENCY APPENDICECTOMY

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Abstract

Negative-pressure pulmonary edema (NPPE) is a rare complication that evolves rapidly after acute or chronic obstruction of the airways. We report the case of a 24-year-old male patient who enrolled for appendicectomy. The patient developed pulmonary edema immediately after extubation. After reintubation and observation in the intensive care unit the patient was extubated and discharged from hospital without any respiratory problem.

Key Words: *Laryngospasm, Pulmonary edema, General anesthesia*

Introduction

Negative pressure pulmonary edema is a potentially life-threatening complication, especially during emergence, in patients undergoing general anesthesia for a variety of surgical procedures. Negative pressure pulmonary edema (NPPE) in the adult is an uncommon medical emergency, arising as a consequence of upper airway obstruction especially during emergence from anesthesia. It usually manifest immediately following extubation, but in some cases, onset can be delayed for several hours.[1] Negative pressure pulmonary edema (NPPE) is classified as two types. Type I is secondary to acute obstruction of the upper airway, and type II occurs after surgical correction of airway obstruction. It is secondary to a sudden reduction in pericapillary pulmonary interstitial pressure due to forced inspiration. It was first described in children in 1973.[2] This form of non-cardiogenic pulmonary edema occurs when the patient struggles to inhale against a partially or completely occluded upper airway. In a vigorous adult the inspiratory muscles can generate a negative intrapleural pressure in the range of -50 to -100 cm H_2O.[3] The high negative pressure gradient causes fluid to extravasate from the pulmonary capillaries into the interstitial and alveolar spaces.[4] This fluid leak represents non-cardiogenic pulmonary edema, and it may result in immediate or delayed hypoxemia. Even after the upper airway obstruction is alleviated, pulmonary edema can develop immediately or up to six hours later.[5]

Case report

A 24 year old man with no previous medical history, known drug usage or known drug allergy enrolled for emergency surgery for appendicectomy. The physical examination and laboratory tests were within normal limits. Preoperative electrocardiograph and chest radiogram were normal. Appendicectomy was performed under general anesthesia. Intravenous propofol, fentanyl, vecuronium were used for anesthesia and no trauma occurred during intubation. Anesthesia and the

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surgical procedures were uneventful. The procedure lasted 90 minutes and a total of 1100 ml of isotonic solution was infused. Immediately after extubation, the patient developed laryngospasm and became agitated. Respiratory distress, tachypnea, cyanosis, accessory muscle utilization and decrease in peripheral oxygen saturation were observed. Spontaneous ventilation with 100 % oxygen could not be assisted by bag and mask ventilation sufficiently and he developed laryngospasm with severe respiratory distress. Subsequently arterial oxygen saturation decreased to a low 70. The patient was reintubated after administration of succinylcholine ($1mg.kg^{-1}$). Suctioning of the tube revealed copious amounts of pink, frothy sputum. Blood gas analysis revealed hypoxia. The chest radiograph showed diffuse alveolar and interstitial infiltration consistent with pulmonary edema (Fig 1).

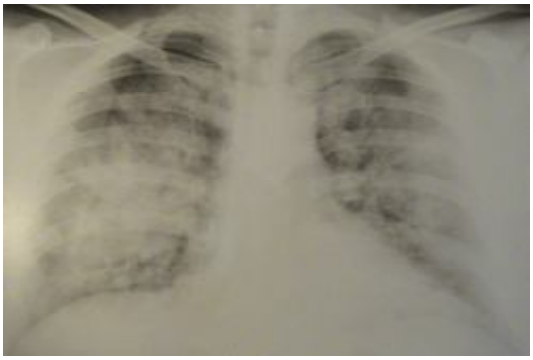

Fig.1 Portable chest radiograph showing pulmonary edema in the patient.

He was transferred to intensive care unit where he was placed on mechanical ventilation and treated with positive end-expiratory pressure (PEEP) of 10 cm H_2O, high fractional inspiratory oxygen concentration (FIO_2) and 20 mg of intravenous furosemide initially. As his oxygenation and clinical picture improved over the next 20 hours, proper ventilator adjustments were made and his FIO_2 was decreased gradually. Arteriel blood gas levels were within normal limits. His extubation was successfully carried out 24 hours after his initial reintubation. Two days later a chest X-ray showed complete resolution of the pulmonary infiltrate. He had an uneventful recovery and was transferred to the surgical ward.

Discussion

The incidence of NPPE has been reported to be 0.05 to 0.1 % of all anesthetic practices, but it has been suggested that it occurs more frequently than is generally documented.[6] The morbidity and mortality associated with under recognized NPPE is as high as 40 %.[7] Risk factors for NPPE include airway lesions, upper airway surgery, obesity and obstructive sleep apnea.

The pathophysiology of NPPE includes three mechanisms: Negative intrathoracic pressure, increased systemic and pulmonary capillary circulation, and mechanical stress on the alveolar-capillary membrane.[8] Typical normal pleural inspiratory pressures range from 2 to 5 cm of water, whereas during severe episodes of upper airway obstruction, negative pressures upto 100 cm of water have been reported.[9]

Negative intrathoracic pressure increases the venous return due to the reduced pressure in the right atrium, with concomitant increase in systemic and pulmonary capillary hydrostatic pressure, which is complicated by reduction in interstitial perivascular hydrostatic pressure. This causes an increase in the transcapillary pressure gradient, thereby favoring transudation of fluid to the interstitial spaces.

The second mechanism which can cause NPPE is an increase in central venous pressure, which prevents the flow in the lymphatic vessels. NPPE is a hypoxic state. The hypoxic state may contribute to pulmonary and peripheral vasoconstriction because of adrenergic release which increases both right and left ventricular pressures and output, thus increasing the transmural gradient in the pulmonary capillary bed. In addition, there is rupture of pulmonary capillaries with loss of proteins that is

responsible for the frothy serous, bloody or pinkish secretion. The third mechanism shows that loss of capillary integrity can result from mechanical stress of the alveolar-capillary membrane, leading to alveolar edema and hemorrhage. In the case presented here, it was not possible to clearly explain the pathophysiology of the edema, since measurements of cardiac chambers and pulmonary capillary pressures were not undertaken. However, any one of the mechanisms mentioned above, isolated or in combination, could have contributed to the development of pulmonary edema.

In clinical presentation, initial findings usually include decreased oxygen saturation, with pink frothy sputum and chest X-ray abnormalities.[10] Manifestations of the acute airway obstruction include stridor, suprasternal and supraclavicular retractions, urgent use of accessory muscles of inspiration, and panic in the facial expression. As NPPE develops, auscultation usually reveals crackles and occasionally wheezes. Pulmonary edema causes both impaired diffusion of oxygen and ventilation/perfusion mismatch, leading to sudden and possibly severe hypoxemia. The typical chest X-ray will show diffuse interstitial and alveolar infiltrates.[11]

The differential diagnosis would include acute respiratory distress syndrome, fluid overload, cardiac abnormalities and pulmonary embolism. Laryngospasm induced pulmonary edema is a self-limited condition with excellent prognosis and relatively simple management but it may also result in significantly prolonged hospital stay and the necessity of treatment in an intensive care unit.

Treatment of NPPE is supportive, maintaining opened airways and oxygenation. Not all patients need mechanical ventilation and positive end expiratory pressure (PEEP). Lang et al. demonstrated that 80% of the cases of NPPE developed shortly after extubation, 85% needed to be reintubated, and 50% required mechanical ventilation with PEEP or continuous positive airways pressure (CPAP).[12,13] The adrenaline administered in these cases, due to the hemodynamic instability, might have worsened the condition, since an increase in systemic blood pressure increases venous blood return and, consequently, pulmonary capillary hydrostatic pressure.

Early diagnosis and treatment are necessary for a favorable evolution and decreased morbidity. It has a good prognosis because the event is self-limited and patients are usually healthy. Prevention is always better than cure and therefore a smooth extubation, good oral and nasal suction and good judgment of size of endotracheal tube always helps.

Conclusion

In conclusion, anesthesiologists must be aware of laryngospasm-induced pulmonary edema. Marked decrease in oxygen saturation shortly after extubation should lead to a high degree of suspicion for laryngospasm induced pulmonary edema.

References

1. Van Kooy MA, Gargiulo RF. Postobstructive pulmonary edema. Am Fam Physician 2000; 62:401-404.
2. Capitanio MA, Kirkpatrick JA. Obstruction of the upper airway in children as reflected on the chest radiograph. Radiology 1973; 107:159-161.
3. Lathan RS, Silverman ME, Thomas BL, Waters WC. Postoperative pulmonary edema. South Med J. 1999; 92(3):313-315.
4. Butterell H, Riley RH. Life-threatening pulmonary oedema secondary to tracheal compression. Anaesth Intensive Care 2002;30:804-806.
5. Tarrac SE. Negative pressure pulmonary edema - a postanesthesia emergency. J Perianesth Nurs. 2003;18(5):317-323.
6. McConkey PP. Postobstructive pulmonary oedema-a case series and review. Anaesth Intensive Care 2000; 28:72-76.

7. Goldenberg JD, Portugal LG, Wenig BL, Weingarten RT. Negative-pressure pulmonary edema in the otolaryngology patient. Otolaryngol Head Neck Surg 1997; 117:62-66.
8. Dolinski SY, MacGregor DA, Scuderi PE - Pulmonary hemorrhage associated with negative-pressure pulmonary edema. Anesthesiology, 2000;93:888-890.
9. Timby J, Reed C, Zeilender S, Glauser FL. Mechanical causes of pulmonary edema. Chest 1990; 98: 973-979
10. Lathan RS, Silverman ME, Thomas BL, Waters WC. Postoperative pulmonary edema. South Med J. 1999; 92(3):313-315.
11. Davidson S, Guinn C, Gacharna D. Diagnosis and treatment of negative pressure pulmonary edema in a pediatric patient: a case report. AANA J 2003; 72(5):337-338.
12. Lang SA, Duncan PG, Shephard DA et al. - Pulmonary oedema associated with airway obstruction. Can J Anesth, 1990; 37:210-218.
13. Gerancher JC, Weeks DB - Nasal BiPAP treats hypoxia from postobstructive pulmonary edema. Anesth Analg, 1995;80:429-430.