In the name of God

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Laryngospasm Induced Pulmonary Edema: A Case Report.

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Abstract:

A 22-yr-old man with no history of disease and normal physical and Para clinical evaluations underwent a general anesthesia for internal fixation of femur fracture for 2 hours. After completion of the surgery and full reversal of neuromuscular blockade, extubation was performed. After a few minutes the patient developed Laryngospasm. With prompt suctioning and positive pressure ventilation, Laryngospasm resolved. When SPO2 was 98% the patient transferred to recovery room. After a few minutes at the recovery room the patient developed dyspnea and a rapid decrease in SPO2 occurred associated with a lot of dirty pink secretions. He was intubated again and with the diagnosis of pulmonary edema frosemide, morphin and low PEEP was administered. After 8 hours pulmonary edema was resolved and tracheal tube was removed.

Keywords: laryngospasm, noncardiac pulmonary edema,

Introduction:

Pulmonary edema is a potentially life-threatening complication of acute airway obstruction.[1] Laryngospasm is a common complication after tracheal extubation, occurring in 8 – 237 of every 1000 anesthetic procedures.[2] Postextubation negative pressure pulmonary edema (NPPE) is a rare complication with a reported incidence of 0.05-0.1% but in an other study it has been reported in 11% of healthy young patient who experienced Laryngospasm.[2] We report a case of laryngospasm a few minutes after extubation who developed pulmonary

edema 10 min after resolution of obstruction.

Case Report:

A 22-yr-old man was admitted for internal fixation of fracture of femur. A complete history and physical examination was obtained at the time of admission with no remarkable findings noted. He denied having any previous history of cardiac or pulmonary disease. He denied any intravenous or other drug use. The patient nil per os (NPO) status had been maintained for 10 hours. He was taken to operating room where an uneventful internal fixation of fracture under general anesthesia was performed. Anesthesia was induced with thiopental, midazolam, fentanyl and atracurium. Endotracheal intubation was carried out and general anesthesia maintained with isoflurane, N2O and O2 for approximately 2 hours. After a full reversal of neuromuscular blockade, endotracheal tube was removed. A few minutes after extubation the patient developed laryngospasm and pulse oximetry showed a marked decrease to 40%, with positive pressure ventilation, obstruction was resolved and oxygen saturation reached to 98%. After assurance of the patient status, he transferred to recovery room. Shortly after transferring to recovery, the patient developed dyspnea and oxygen saturation markedly decreased. At suctioning the oropharyngeal secretions, pink frothy fluid removed. The patient transferred to operating room and reintubated. Pinky frothy fluid filled the endotracheal tube and chest examination revealed bilateral generalized rales without evidence of wheezing. Frosemide and morphine sulfate were administered intravenously. The endotracheal tube was suctioned intermittently and 100% O2 was administered. A portable chest radiograph 3 hours after than the beginning of treatment demonstrated pulmonary edema (fig 1).

The patient transferred to intensive care unit (ICU). In ICU he was treated with frosemide and PEEP (5-10 cmH2O). After 6 hours the patient was partially recovered and after 8 hours he was extubated (fig2) and at less than 12 hours he was completely well and transferred to the ward.

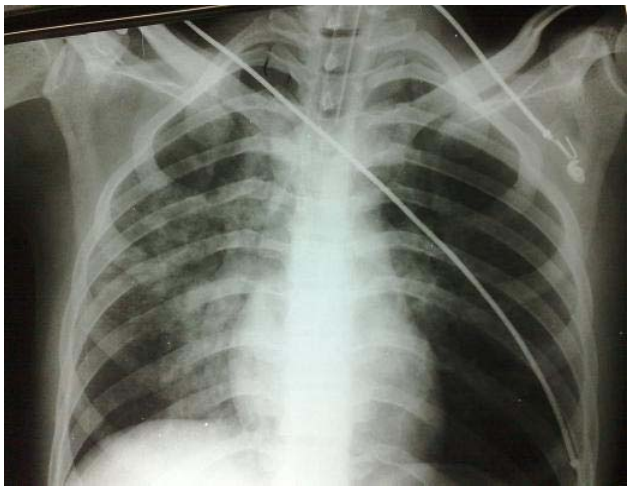

Figure 1: 3 hours after treatment

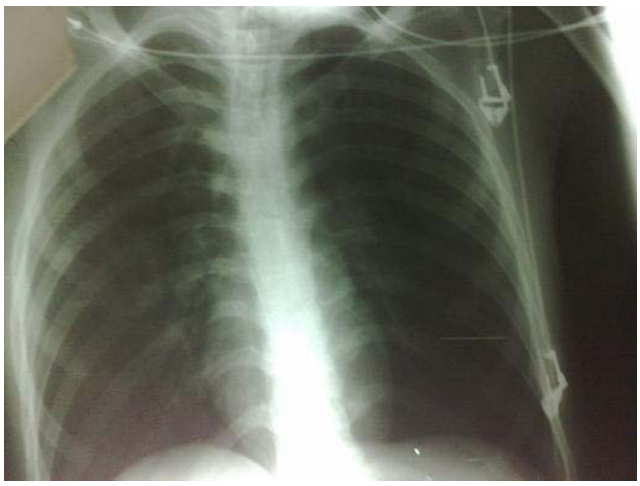

Figure 2

Discussion:

Negative pressure pulmonary edema (NPPE) subsequent to acute airway obstruction was first theorized (based on experiments on anesthetized dogs) by Moore in 1927. In 1973 the first clinical description of pulmonary edema associated with upper airway obstruction was reported and in 1990 Lang et al published a comprehensive review on the subject and concluded that the pathogenesis was multifactorial.[4]

Post obstruction pulmonary edema (POPE) is a noncardiac perioperative event. It is the sudden onset of pulmonary edema following upper airway obstruction. There are two recognized type of POPE, type I follows a sudden, severe episode of upper airway obstruction such as postextubation laryngospasm, epiglottitis, croup and chocking and is seen in strangulation and hanging.[1] Foreign-body aspiration and even hiccup have been reported to cause NPPE.[5] Type I POPE may be associated with any cause of acute airway obstruction. Type II POPE develops after surgical relief of chronic upper airway obstruction. Reported causes include tonsillectomy and removal of upper airway tumors.[1] The mechanism whereby upper airway obstruction precipitates pulmonary edema is not know with certainty. The initiating event is usually a vigorous inspiratory effort against a closed glottis, resulting in markedly negative pleural pressures. 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 H2O have been reported.[3] This excessive negative presser is thought to souse in disruption of the alveolar capillaries to cause pulmonary edema, and sometimes alveolar hemorrhage.[2] Negative intrathorasic pressures are thought to promote hydrostatic transudation of fluid from the intravascular compartment into the lung interstitium. There is probably also an influence from the autonomic nervous system with a hyper adrenergic state resulting from the stress induced by upper airway obstruction. This results in peripheral vasoconstriction and an increase in the volume of the pulmonary circulation, further increasing pulmonary arterial pressures.[6]

Risk factors include the prior administration of naloxone [6, 7], difficult intubation, nasal, oral or pharyngeal surgery [6], obesity, short neck and sleep apnea.[5] The development of pulmonary edema occurs within 1-90 min of laryngospasm [4] but may be delayed up to 6 hours.[5] The pulmonary edema resolves rapidly.[4] 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.[3]

In our patient pulmonary edema developed a few minutes after laryngospasm. Clinical examination and chest x-ray ruled out pulmonary emboli and aspiration. In other hands the patient denied any cardiac disease and because of good response to treatment cardiac pulmonary edema is ruled out as well.

Conclusion:

The anesthesiologists must be aware of laryngospasm induced pulmonary edema. Marked decreased in oxygen saturation

shortly after extubation should stimulate a high index of suspicion for laryngospasm induced pulmonary edema. In other hands, in spite of good response to treatment and excellent prognosis, it is necessary to prevent this event, and in the case of laryngospasm, prompt treatment with suctioning and positive pressure ventilation with 100% O2 and administration of succynilcholine if needed, is necessary to limit the duration of hypoxia and hopefully prevent post obstruction pulmonary edema from occurring.

References:

1. Mark A Vankooy, Richard F Garciulo. Postobstructive pulmonary Edema. American family Ph, July 15, 2000. Http://www.aafp.org/afp/2000715/401.html

2. Yoshitaka Hirano, Taku Sugawara, Yoshiharu Sato, Kojiq Sato, Tomoya Omag, Toshio Sasajima, Kauo Mizoi. Negative Pressure Pulmonary Edema following foramen magnum Decompression for chiari malformation type I- Case Report. Neurolo Med Chir (Tokyo) 2008; 48:137-139.

3. Mehmet Turan Inal.Laryngospasm-induced Pulmonary Edema. The internet Journal of Anesthesiology 2007, 15 (7).

4. Frank E Scarbrough, Joerg M Wittenberg, Brian R Smith, Deborah H Adcock. Pulmonary Edema Following post Operative Laryngospasm: Case report and Review of the literature. Anesth Prog 1997; 44: 110-116.

5. Francis Ansa, Lalit Kanaparthi, Gerand E Carino, Achal Dhupa, Ehab Daoud, Paul Yodice. Rapid-onset Pulmonary Edema After Routine Elective Surgery: A review of two cases. October 25, 2006 CHEST: http://meeting.chestpubs.org/content/abstract/130/4/343s - b.

6. SPG Padley, MO Downes.Case Report: Pulmonary edema secondary to laryngospasm fallowing general anesthesia.The British Journal of radiology 1994; 67: 654-655.

7- K. S. Oslen. Naloxone administration and laryngospasm followed by pulmonary edema. Intensive Care Med 1990, 16: 340-341