Negative Pressure Pulmonary Edema

By Hilary G. Miller, R.N.

Recently, pulmonary edema associated with negative airway pressure is believed to be a result of acute airway obstruction. A literature review reveals that postanaesthetic laryngospasm is the most common cause of upper airway obstruction leading to negative pressure pulmonary edema (NPPE) in adults (Tami et al, 1986).

Abstract

Pulmonary Edema associated with negative airway pressure caused by upper airway obstruction is a most serious complications in anaesthetic practice(Tami et al, 1986). Laryngospasm associated with intubation and general anaesthesia is the most common cause of upper airway obstruction leading to negative pressure pulmonary edema (NPPE) in the anaesthetic adult (Tami et al, 1986). Other risk factors for the development of upper airway obstruction are identified, and individuals at risk should be observed closely while they remain at risk during the post anaesthetic period.

NPPE appears to be related to markedly negative intrathoracic pressure due to forced inspiration against a closed upper airway resulting in transudation of fluid from pulmonary capillaries to the interstitium.

The following is a presentation of a case of a healthy young male who developed NPPE secondary to airway obstruction caused by biting down on the endotracheal tube while awakening from general anaesthesia.

Acute NPPE following post anaesthetic laryngospasm is uncommon and potentially life threatening and therefore demands early diagnosis and prompt treatment (Holmes et al, 1991). The underlying risk factors for upper respiratory obstruction are:

- laryngospasm
- mechanical airway obstruction (i.e. biting on endotracheal tube or foreign body in the tube)
- hypolaryngeal obstruction
- airway tumor
- obesity with obstructive sleep apnea
- vocal cord palsy
- nasopharyngeal abnormalities
- infections such as epiglottis, croup and
- bacterial tracheitis (Lorch & Sahn, 1986, and Holmes et al, 1991).

Case Report - NPPE Post Extubation

A previously healthy 25 year old male presented to the operating room for emergency exploratory laparotomy following a gunshot wound to the abdomen. He was given a general anaesthetic and had a stable intraoperative course with no cardio/respiratory difficulty. Prior to extubation, the patient forcefully started biting down on the endotracheal tube (ETT), totally occluding the tube and making respiratory efforts against the occluded airway. The ETT was forcibly removed by the anaesthetist and an attempt to put in a Guedal tube was unsuccessful. The patient developed respiratory distress with vigorous respiratory effort. Pulse oximetry indicated SpO_2 70%. He was reparalyzed with succinylcholine and reintubated. In addition he was given midazolam, d-tubocurarine and propofol. The patient was monitored in the operating theatre until he was ready for extubation. At this time extubation was performed successfully and a Guedal tube was left in. He continued to bite on the Guedal tube but was able to ventilate well with and SpO_2/ 85%. He was then transferred to PACU with oxygen at 15 L/min by face mask.

In PACU he was put on oxygen at 100% by non-rebreathing mask. Vital signs were: BP 160/70, RR 32/min, SpO_2/ 58-68%. The patient became extremely combative and restless with respirations laboured and rapid. In addition, diffuse bilateral crackles were heard in both lung fields. The patient was treated with the diuretic Lasix IV, resulting in diuresis, and adventitious sounds less pronounced. A chest x-ray showed diffuse interstitial pulmonary edema, eliminating the possibility of aspiration. Arterial blood gases with the patient receiving oxygen via the non-rebreathing mask were: $p0_2$ 43%, pH 7.24, pCO_2 52, HCO_3 22. Despite high flow oxygen, the SpO_2 was 80 - 90% over the next 1 1/2 hours, gradually falling to 75% with a pulse rate of 140/min. The patient became very diaphoretic, cyanotic and increasingly drowsy. Respirations became more shallow and rapid to a rate of 56/min. Attempts at bag-mask ventilation assist were unsuccessful in raising the SpO_2. The patient was then reintubated and placed on mechanical ventilation with PEEP. The endotracheal tube was suctioned for small amounts of pink frothy secretions. Pulse oximetry showed SpO_2 92%. His colour improved, BP 110/70, heart rate 118. The patient was transferred to Intensive Care for continued monitoring and treatment. His condition improved significantly overnight and in the morning he was weaned from ventilator support, extubated and subsequently transferred to the nursing unit later that day. He continued to do well and was then discharged from hospital.

Discussion

The predominant mechanism of NPPE is increased negative intrathoracic pressure due to forced inspiration against a closed upper airway. This results in large negative intrapleural and trans-pulmonary pressure gradients, producing the transudation of fluid from the pulmonary capillaries into the interstitium (Lorch & Sahn, 1986). Blood return to the right heart is abruptly increased secondary to increased venous return with a redistribution of the central circulation. This results in increased pulmonary vascular resistance secondary to hypoxic pulmonary vasoconstriction. Left heart output is decreased due to increased afterload, acidosis and hypoxia. The resultant increase in pulmonary blood volume and hydrostatic pressure along with negative interstitial pressures and increased capillary permeability facilitates the development of pulmonary edema (Tami et al, 1986, Nishizawa et al).

Upper airway obstruction

The upper airway obstruction in this case was induced by biting down on the endotracheal tube and laryngospasm after his first extubation. The patient was young and muscular, thus his ability to generate excessive negative intrathoracic pressure was enhanced. The marked decrease in oxygen saturation might have been the result of hypoventilation caused by tube obstruction and impairment in aveolar gas exchange caused by pulmonary edema.

The pulmonary edema resolved with the aggressive treatment of diuretics and mechanical ventilation with positive airway pressure. The use of a Guedal tube or bite block during anaesthesia might have avoided this complication. However, the risk of dental injury remains significant with a Guedal airway.

The potential complication of NPPE should be anticipated before placement of an artificial airway, especially in those patients with a severe degree of upper airway obstruction (Bonadio & Losek, 1991). A heightened awareness of the problem in high risk groups should involve special considerations including choice of anasethesia, precautions on extubation, monitoring in the recovery phase, and if laryngospasm is observed, use of rapid therapeutic intervention.

The majority cases of NPPE present within minutes of the development of acute severe upper airway obstruction (Lang et al, 1990). Resolution is typically rapid over a period of a few hours and rarely is anything more required for management than the maintenance of a patent airway, supplemental oxygen and in a few cases mechanical ventilation with PEEP (Lang et al, 1990).

Although the majority of cases of NPPE develop shortly after the onset of airway obstruction, delayed onset has been reported. Therefore, careful observation is required for several hours after an episode of upper airway obstruction from any cause (Tami et al,

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1986, Nishizawa & Goto, 1993).

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Correction

to ORNAC's Recommended Standard #8
"Scrub, Gown , Glove"
Section 8.13 should read:

If a glove becomes contaminated or sustaines a pin hole, the glove shall be changed as soon as the situation permits by one member of the sterile team regloving the other member. If not possible, by open glove method.

Rationale:

Once the original gloves are donned, the gown cuffs are considered contaminated.

Wanted

ORNAC is trying to locate copies of the May, 1986 *Communiqué.* If you have a copy of know of someone who has an extra, please forward it to:

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