Negative Pressure Pulmonary Edema in a Child Following Laryngospasm Triggered by a Laryngeal Mask

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Negative pressure pulmonary edema (NPPE) is a potentially life-threatening complication that develops rapidly following acute upper airway obstruction (UAO) in otherwise healthy young patients. NPPE produces markedly negative intrathoracic pressure [1].

In short pediatric outpatient surgical procedures, the laryngeal mask airway (LMA) has been used increasingly in clinical practice. There is impressive agreement in the literature showing that the LMA is associated with increased patient comfort and decreased cough. However, an increased risk of laryngospasm has been described. [2]. We recently described a case of NPPE that occurred following choking on a cookie [1].

PATIENT DESCRIPTION

A 17 year old obese (body mass index 32 kg/m^2) male with a history of smoking (1.5 pack-years) developed an acute episode of respiratory distress in the recovery room while waking from general anesthesia administered with an LMA. Laryngospasm with marked inspiratory effort and cyanosis occurred. Oxygen saturation < 85% on air room was measured, with accessory muscle use and frothy bloody sputum. He was afebrile, blood pressure was 100/60 mmHg and heart rate 112 beats per minute with bilateral rales on auscultation. Laboratory results revealed 11,200 cells/μl leukocytes, 60% neutrophils, 11.7 g/dl hemoglobin, and normal platelets count. Electrolytes and renal function tests were normal. Venous blood gas revealed pH 7.18, and PCO_2 64 mmHg. Chest radiograph showed bilateral, centralized alveolar infiltrates [Figure 1].

The LMA was removed and the patient was orotracheally intubated. The acute onset of the symptoms, physical examination, and chest X-ray supported the diagnosis of NPPE.

Despite controlled ventilation with 100% oxygen, oxygen saturation remained in the low 90s and pink frothy sputum appeared in the tracheal tube. Respiratory support with mechanical ventilation with positive end-expiratory pressure and nitric oxide was applied and the patient was transported to the pediatric intensive care unit. Cardiac echography was normal.

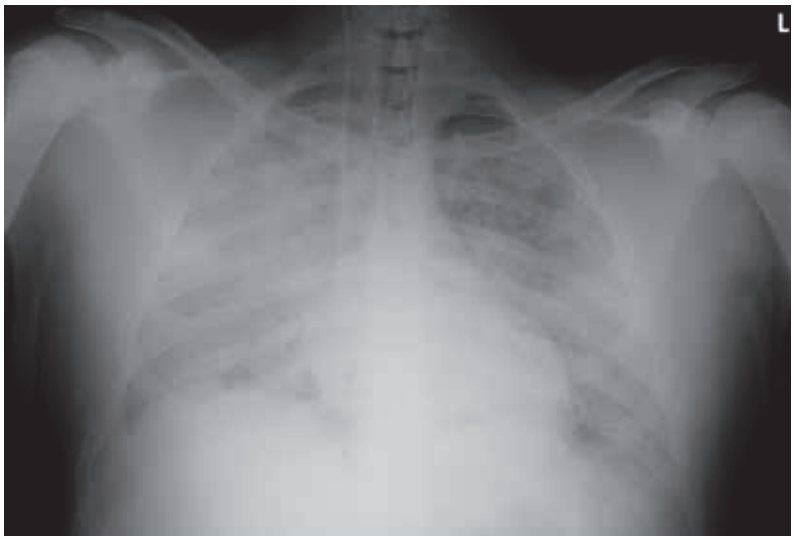
Forty-eight hours later the pink frothy sputum decreased. The patient was extubated and transported to the pediatric department without respiratory symptoms, with oxygen saturation 99% on room air, and chest radiograph showing significant improvement.

He remained completely healthy, with normal chest auscultation and normal chest X-ray. His pulmonary function test 2 weeks later showed FEV_1 of 112% predicted.

COMMENT

We report a case of NPPE in a 17 year old obese male with a history of smoking, who was in the recovery room during emergence from general anesthesia that had been administered with an LMA. Since our

Figure 1. Chest radiograph following general anesthesia with a laryngeal mask airway showing bilateral, centralized alveolar infiltrates



patient had normal cardiac echocardiography and rhythm, a cardiogenic reason for his pulmonary edema is unlikely. The most likely diagnosis is NPPE, a form of non-cardiogenic pulmonary edema due to acute UAO.

When sudden acute respiratory distress and bilateral alveolar infiltrates are identified in a child, the main differential diagnosis includes pulmonary edema (cardiogenic and non-cardiogenic), pulmonary hemorrhage, pulmonary embolism (including fat emboli in the setting of orthopedic surgical procedures), and foreign body aspiration [3].

UAO caused by LMA has been reported mainly in adults. There are relatively few reports of negative alveolar pressures developing in children during emergence from general anesthesia with an LMA. A review of the literature revealed two case reports, one from India [4] and one from Japan [5].

The LMA was developed and introduced into clinical practice in 1988. Due to its safety and versatility, the indications for its use have been greatly expanding, and currently LMAs are used for short outpatient procedures in both the adult and pediatric populations. For outpatient procedures, the reinforced LMA has several advantages, including the decreased use of non-depolarizing muscle relaxants. As a result, possible side effects are circumvented, as is avoidance of stimulation of the larynx and vocal cords, obviation of the need for laryngoscopy, elimination of risks of endobroncheal or esophageal intubation, and improvement in various postoperative outcome parameters [2].

Disadvantages of the LMA have also been reported. They include trouble visualizing the surgical field, leaking or kinking, and an increased incidence of laryngospasm observed in children.

In the normal lung, the net fluid transfer across the pulmonary capillaries depends on the net difference between hydrostatic and colloid osmotic pressures, as well as on the permeability of the capillary membrane. Young healthy subjects can generate very high levels of negative inspiratory pressure with a maximum of -140 cmH_2O. Creation of marked negative intrathoracic pressure by the forceful inspiratory efforts against an obstructed glottis or UAO promotes increased blood flow to the pulmonary circulation thereby increasing pulmonary arterial pressure, causes a rise in transcapillary pressure gradient that favors a shift of fluid from the capillaries into the alveoli, resulting in hypoxemia. The hypoxemia results in increased pulmonary arterial constriction and pressure, thus damaging pulmonary capillaries with increased permeability and further worsening pulmonary edema.

Two types of NPPE have been described. Type I is usually found in adults and is caused by forceful inspiration in the presence of an acute airway obstruction. Type II usually occurs after relief of chronic partial airway obstruction (post-obstruction pulmonary edema) [1]. The incidence of NPPE in adult patients developing UAO or laryngospasm has been estimated to be up to 12%. The true incidence, however, is unknown due to lack of familiarity with the syndrome. All causes of UAO may lead to NPPE, including laryngospasm, strangulation, epiglottitis, impacted foreign body in the trachea, obstruction of the endotracheal tube, falling back of the tongue, or obstructive sleep apnea. However, the most commonly reported etiology of NPPE in adults (50% of cases) is laryngospasm during intubation or in the postoperative period after anesthesia [3]. In addition, obesity and lower cranial nerve paresis have been described as risk factors for pulmonary edema following extubation post-surgery [5].

Our patient presented with immediate onset of respiratory distress in the recovery room, following surgery for internal fixation of a right ankle fracture, due to laryngospasm during emergence from anesthesia with an LMA. Since other causes of pulmonary edema were excluded, supportive care with immediate orotracheal intubation and mechanical ventilation with positive end-expiratory pressure and nitric oxide was applied. Due to the rapidly resolving clinical course of the pulmonary edema in 48 hours, we concluded that negative pressure mechanism after laryngospasm on LMA was the causative factor for the clinical presentation. Although the possibility of kinking or dislocation of the LMA should be considered in such cases, in this setting we believe that his clinical picture was more consistent with laryngospasm. Cases of NPPE in children have been reported, most of which occurred post-anesthesia in the recovery room (post-extubation laryngospasm or post-surgical removal of UAO). NPPE occurring in the setting of anesthesia with LMA in children is rarely reported.

The use of LMA is expanding, especially in outpatient procedures, including in children. Understanding the pathophysiological mechanisms and early identification of NPPE may prevent the use of inappropriate treatments for patients with NPPE.

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"The greatest deception men suffer is from their own opinions"

Leonardo da Vinci (1452–1519), Italian painter, sculptor, architect, inventor, engineer, and draftsman