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Case report

Postobstructive pulmonary edema after biopsy of a nasopharyngeal mass

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ABSTRACT

We describe a case of 17 year-old male with a nasopharyngeal rhabdomyosarcoma who developed postobstructive pulmonary edema (POPE) after removing the endotracheal tube following biopsy. He developed muffled voice, rhinorrhea, dysphagia, odynophagia, and difficulty breathing through nose and weight loss of 20 pounds in the preceding 2 months. A nasopharyngoscopy revealed a fleshy nasopharyngeal mass compressing the soft and hard palate. Head and neck MRI revealed a large mass in the nasopharynx extending into the bilateral choana and oropharynx. Biopsy of the mass was taken under general anesthesia with endotracheal intubation. Immediately after extubation he developed oxygen desaturation, which did not improve with bag mask ventilation with 100% of oxygen, but improved after a dose of succinylcholine. He was re-intubated and pink, frothy fluid was suctioned from the endotracheal tube. Chest radiograph (CXR) was suggestive of an acute pulmonary edema. He improved with mechanical ventilation and intravenous furosemide. His pulmonary edema resolved over the next 24 h. POPE is a rare but serious complication associated with upper airway obstruction. The pathophysiology of POPE involves hemodynamic changes occurring in the lung and the heart during forceful inspiration against a closed airway due to an acute or chronic airway obstruction. This case illustrates the importance of considering the development of POPE with general anesthesia, laryngospasm and removal of endotracheal tube to make prompt diagnosis and to initiate appropriate management.

1. Introduction

Postobstructive pulmonary edema (POPE) is a rare but serious complication associated with upper airway obstruction. The pathophysiology of POPE involves hemodynamic changes occurring in the lung and the heart during forceful inspiration against a closed airway due to an acute or chronic airway obstruction. We describe a case of 17 year-old male with a large nasopharyngeal mass who developed POPE after biopsy of the mass. This case illustrates the importance of considering the development of POPE with general anesthesia, laryngospasm, and removal of endotracheal tube to make prompt diagnosis and to initiate appropriate management.

2. Case presentation

A 17 year-old African American male with no significant past medical history presented to a physician's office for an influenza vaccination. He reported progressively worsening muffled voice, rhinorrhea, dysphagia, odynophagia, inability to breathe through nose and unintentional weight loss of 20 pounds in the preceding 2 months. Physical examination was significant for rhinorrhea and a mass in the mouth nearly filling the oral cavity. He was immediately sent to the otolaryngology clinic for further evaluation. A nasopharyngoscopy demonstrated a fleshy nasopharyngeal mass compressing the soft and hard palate displacing them antero-inferiorly Fig. 1. Further workup included head and neck MRI, which demonstrated a large mass in the nasopharynx extending into the bilateral choana and oropharynx Fig. 2. A week later, the patient was taken to the operating room for biopsy of the mass under general anesthesia with endotracheal intubation. Immediately after

Abbreviations: MRI, magnetic resonance imaging; CXR, chest radiograph; FiO2, fraction of inspired oxygen; POPE, postobstructive pulmonary edema; NPPE, negative pressure pulmonary edema; LV, left ventricle; RV, right ventricle; ARDS, acute respiratory distress syndrome; PEEP, positive end expiratory pressure; CPAP, Continuous positive airway pressure.

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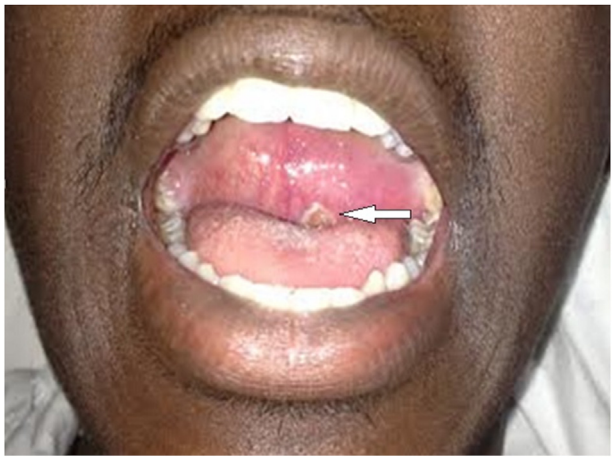

Fig. 1. Arrows show a nasopharyngeal mass displacing the soft and hard palate antero-inferiorly. A mucosal ulcer is seen over the mass.

extubation, he developed oxygen desaturation. His oxygenation did not improve with bag mask ventilation with 100% of oxygen, but improved only after a dose of succinylcholine. He was re-intubated and pink frothy fluid was suctioned from the endotracheal tube. Chest radiograph (CXR) was suggestive of an acute pulmonary edema Fig. 3. He received 40 mg of furosemide intravenously, which was repeated twice in the pediatric intensive care unit (PICU). Serial CXR revealed gradual resolution of pulmonary edema over the next 24 h. On hospital day #2, he was extubated, placed on bilevel positive airway pressure (BiPAP) ventilation with the pressure setting of 14/6 cm H_2O, inspired oxygen (FiO_2) of 40% for 12 h. Overnight he was weaned to face mask with 30% oxygen, then weaned to room air the next morning.

3. Discussion

The first description of postobstructive pulmonary edema

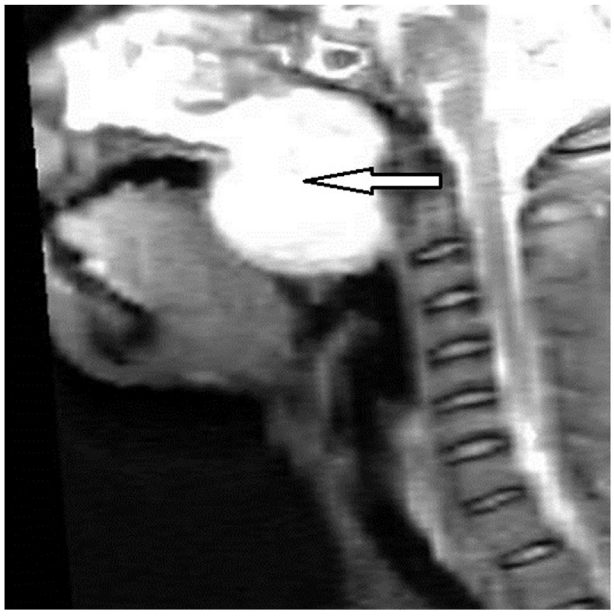

Fig. 2. MRI reconstruction - Arrow shows a nasopharyngeal mass extending from posterior aspect of nasopharynx to the hypopharynx, significantly narrowing the oropharyngeal airway at the tongue base.

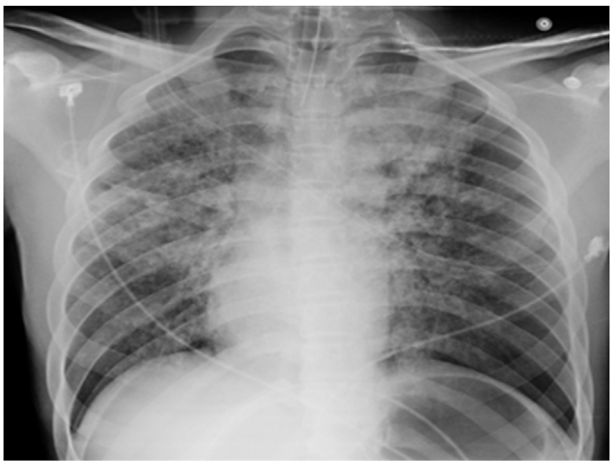

Fig. 3. Diffuse bilateral air space opacities greater in the upper lung fields suggestive of pulmonary edema.

(POPE) was on an animal model in 1927, and the first human cases were reported in 1973 [1,2]. POPE is considered rare, and most reported cases occurred after the administration of anesthesia. POPE is thought to occur from hemodynamic changes secondary to forced inspiration against a closed airway induced by an acute or chronic airway obstruction. POPE is also called negative pressure pulmonary edema (NPPE) and its prevalence has been estimated to be less than 0.1% of cases of administration of general anesthesia [3]. It usually occurs in young, healthy male patients [4,5]. Numerous clinical conditions with upper airway obstruction have been associated with POPE, such as laryngospasm, foreign-body aspiration, endotracheal intubation, upper airway tumor, and obstructive sleep apnea.

POPE has been categorized into two types. Type I POPE occurs after forceful inspiration against an acute upper airway obstruction, while Type II POPE occurs after relief of chronic airway obstruction. The pathophysiology of Type I POPE involves the hemodynamic changes occurring in the lung and the left ventricle (LV) during forceful inspiration against the obstruction. During normal inspiration, pleural and alveolar pressure become more negative than the atmosphere, effecting air flows from the ambient pressure atmosphere to the low pressure alveoli. The decrease in pleural pressure increases venous return from peripheral veins to the intrathoracic vena cava and right atrium.

With an acute upper airway obstruction, the patient takes forceful inspiration against the obstruction, known as Muller's maneuver [6]. The intrapleural pressure decreases markedly and venous return increases due to the increased pressure gradient from the peripheral veins to the central veins leading to increased right ventricular (RV) output, pulmonary blood flow and RV pressure. Buda AJ et al. examined the hemodynamic events of Muller maneuver and found pleural pressure surrounding the LV, peripheral arterial pressure and LV ejection fraction were decreased, despite an increased LV volume. They concluded that the negative intrathoracic pressure affected LV function by increasing LV transmural pressure and afterload [7]. The impaired LV function also increased the pulmonary capillary back pressure. When the transcapillary pressure, the pressure gradient between the pulmonary capillary to the interstitium increases above 18 cm H_2O, capillary transudation occurs. Once the microvascular filtrate in the peri-microvascular compartment exceeds a certain limit, it inundates the adjacent alveoli causing alveolar edema [8]. Pulmonary vascular congestion, chronic hypoxia and hypercapnea cause systemic fluid retention further favoring pulmonary capillary fluid

transudation and development of pulmonary edema [9,10]. Pathogenesis of POPE can represent either a form of hydrostatic edema as in congestive heart failure or an increased permeability edema as in acute respiratory distress syndrome (ARDS). Limited studies of protein composition of the edema fluid indicate that it is a hydrostatic edema. Alveolar exudate protein was less than 65% of the serum protein, which is a cutoff point between hydrostatic versus permeability edema [11,12]. When the factors favoring fluid movement from the capillary to the interstitium is reversed, pulmonary edema rapidly resolves. Rapid resolution of edema in POPE suggests an intact alveolar capillary membrane, unlike that in ARDS, in which edema resolution is delayed.

Our patient had partial obstruction of the nasopharynx and oral airway due to a tumor, which turned out to be rhabdomyosarcoma on pathology. His baseline serum bicarbonate was 31mEq suggesting metabolic compensation for chronic hypercapnea secondary to an upper airway obstruction. After extubation, maintenance of airway patency was probably compromised from the residual effect of anesthesia, leading to airway occlusion. However, obstruction from laryngospasm after extubation is also a possibility. In chronic airway obstruction it is believed that there is a moderate level of auto PEEP (positive end expiratory pressure) produced by the obstructing lesion which increases end expiratory lung volume. With removal of the obstruction there is a sudden removal of PEEP which leads to interstitial fluid transudation and pulmonary edema [13,14].

Diagnosis of POPE requires a high index of suspicion. Persistent tachypnea, oxygen desaturation, pink frothy bronchial secretions, bradycardia, and rales on auscultation indicate likely pulmonary edema. The diagnosis is established by CXR. A chest computed tomography (CT) is not necessary for the diagnosis of POPE.

POPE usually resolves within 12–24 h if prompt diagnosis and proper management are initiated. Most cases have been treated with PEEP and diuretics; however the role of diuretic is not clear [15]. Continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) is an alternative to intubation. Recent data suggest that noninvasive respiratory support such as BiPAP may be an important tool to prevent or treat acute respiratory failure while avoiding intubation. The aims of noninvasive respiratory support in the management of POPE include reducing the work of breathing, increasing alveolar recruitment with better oxygenation, and reducing left ventricular after load, increasing cardiac output and improving hemodynamics [16].

POPE resolves by itself once the airway obstruction is removed. Passage of time brings about resolution of edema, not from diuretics or corticosteroids. Early relief of upper airway obstruction should decrease the incidence of POPE. Preventative measures have been suggested, such as the use of intra-operative muscle relaxants, topical or spray lidocaine, corticosteroids, and early or delayed extubation to reduce the risk of laryngospasm. Other preventive measures that have also been suggested are to clear the airway of the retained blood or secretions by suctioning the oropharynx and extubating the patient in either a very deep or very light level of anesthesia, when the risk of laryngospasm is less. Prophylactic CPAP may be the most effective preventive measure for the patients at-risk [17,18].

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