Negative Pressure Pulmonary Edema: An Uncommon Life-Threatening Postoperative Complication

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Negative pressure pulmonary edema (NPPE) is an uncommon but potentially life-threatening postoperative complication which occurs following sudden upper airway obstruction. Hypoxemia and acute respiratory failure are often severe. Therefore, early recognition and initiation of appropriate treatment are warranted.

We present a case of a twenty-one-year-old male who presented with NPPE due to upper airway obstruction following adenoidectomy. The patient was intubated and sedated to initiate mechanical ventilation. Fluid balance was optimized and weaning off mechanical ventilation was successful under dexmedetomidine infusion. The patient was discharged with favourable outcome.

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Negative pressure pulmonary edema (NPPE) is a rare and potentially life-threatening condition[1]. In children, it mainly occurs following upper airway infectious diseases such as epiglottitis or croup[2]. In adults, NPPE is commonly seen in postoperative settings[3-5]. Rapid onset, severe hypoxemia, and adrenergic storm are the hallmark of the clinical presentation[6,7]. The diagnosis is usually established based on the clinical scenario and chest X-ray findings. The differential diagnosis includes acute ischemic heart disease and decompensated heart failure[7].

The risk factors include obesity, sleep apnea, nasal, oral and pharyngeal surgery[7-9]. The pathophysiology involves sudden changes in the pre and afterload of the heart and/or a rapid relief of a chronic partial upper airway obstruction[7].

Available data in the literature are limited to case reports or small series as this complication is rarely encountered in the clinical practice[10]. Early recognition and timely initiation of appropriate treatment are of paramount importance to improve the prognosis. The weaning process of these patients is always challenging as it needs optimization of the fluid balance as well as the systolic-diastolic heart function monitoring, appropriate management of the sedation and gradual decrease of the ventilatory support.

The aim of this presentation is to report a severe case of negative pressure pulmonary edema following adenoidectomy.

THE CASE

A twenty-one-year-old male patient, known case of obstructive sleep apnea presented to the Ear Nose and Throat clinic with a complaint of bilateral tinnitus. Enlarged adenoids were seen on fiber-optic scope and the patient had adenoidectomy and bilateral Grommet insertion under general anesthesia. Adenoidectomy was performed by suction cautery and hemostasis was achieved. The procedure was uneventful and the patient was well sedated during surgery. He was extubated after reversal of muscle relaxant. The patient was initially hemodynamically stable and maintaining patent airway and 100% oxygen saturation. Five minutes later, he developed apnea and acute respiratory failure. Continuous Positive Airway Pressure (CPAP) ventilation was transiently applied and then the patient was re-intubated and started on mechanical ventilation. Frothy and pink secretion were suctioned from the endotracheal tube suggesting pulmonary edema. Chest auscultation revealed bilateral diffuse crackles. The diagnosis was confirmed by anteroposterior chest X-ray showing diffuse bilateral alveolar syndrome, see figure 1.

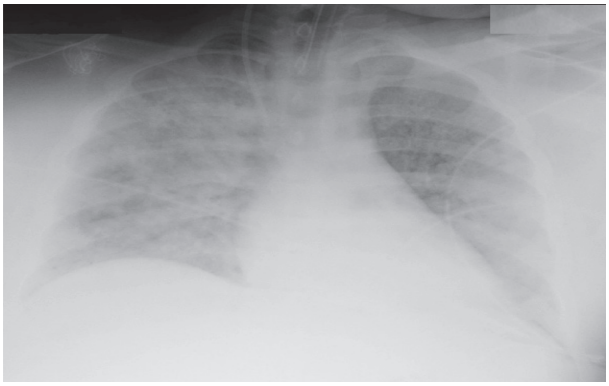

Figure 1: **Chest X-ray following Intubation Showing Bilateral Diffuse Alveolar Syndrome Suggesting Pulmonary Edema**

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The patient was kept on mechanical ventilation with the volume-controlled mode, fractional inspiratory oxygen (FiO_2) at 100% and Positive End Expiratory Pressure (PEEP) at 10 cmH_2O. Plateau pressure was 30 cmH_2O and the lung compliance was low. Arterial blood gases showed severe hypoxemia with a PaO_2/FiO_2 ratio at 48 mmHg. The right internal jugular venous catheter and radial arterial line were inserted. The patient was kept sedated with propofol and remifentanil infusion.

Dopamine and noradrenaline infusion were started as the patient developed hypotension. Electrocardiogram revealed sinus tachycardia. Cardiac enzymes screening was negative. Urgent bedside echocardiogram revealed hyperdynamic wall motion, left ventricular ejection fraction at 65% and grade I diastolic dysfunction. After improvement of the hemodynamic condition, the patient was started on furosemide infusion targeting negative fluid balance. The patient was transferred to the intensive care unit (ICU) for further management. Vasopressors were decreased. Accordingly, weaning from mechanical ventilation was started. Propofol and remifentanil infusions were held and the patient was started on dexmedetomidine infusion which enabled successful weaning and extubation after 4 days of mechanical ventilation. The patient was transferred to the ward after 7 days of ICU stay.

DISCUSSION

Negative pressure pulmonary edema is a rare postoperative complication. The incidence reported in other studies was 0.094% in patients undergoing surgery under general anesthesia[11,12]. Although it frequently occurs in healthy individuals, several risk factors have been reported in the literature such as male gender, obesity, obstructive sleep apnoea and nasal surgery[12,13]. The clinical presentation is characterized by sudden onset of frothy secretion, acute respiratory failure and severe hypoxemia following laryngospasm[6,14,15]. This complication has been reported in only 0.1% of those patients who develop postoperative upper airway obstruction[6].

It is commonly accepted that negative pressure pulmonary edema is a form of non-cardiogenic pulmonary edema[13]. However, recent studies suggest that the main mechanism leading to fluid accumulation in the extravascular lung spaces is an abrupt change in the pre and after load condition of the left ventricle[6,7,10]. In fact, the generation of respiratory effort against obstructed airway induces a deep fall of the pleural pressure from an average value of -4 cmH2O down to -140 mmHg[7,16]. The effect of this excessive negative pressure on the heart cavities is an abrupt increase of the venous return to the right ventricle as well as an increase of the left ventricle afterload due to the increase of the left ventricle transmural pressure[10]. As a result, the hydrostatic capillary pulmonary pressure increases and transudative fluid accumulates in the extra-vascular pulmonary spaces[7,10]. These hemodynamic disturbances are aggravated by an adrenergic storm that induces further increase in the venous return via the peripheral vasoconstriction, see figure 2[6,7,10,14].

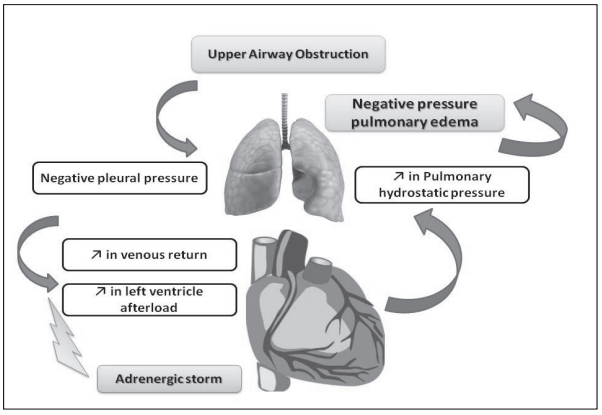

Figure 2: **Physiopathology of Negative Pressure Pulmonary Edema**

Several studies have assessed the protein concentration of the fluid edema in patients with presumed increased hydrostatic pulmonary pressure[17,18]. Although most of the patients had low protein concentration in the fluid edema, high protein levels were seen in patients with severe clinical presentations[17,18]. These findings suggest that the alveolocapillary membrane can be injured in case of the significant rise of the pulmonary hydrostatic pressure which might explain the unusual complicated clinical course of these patients[18].

Early diagnosis and prompt management are of paramount importance. The main pillars of the treatment are maintaining the patient airway, oxygen supply to correct the hypoxemia, applying positive airway pressure to reverse the left ventricle pre/after load and optimization of the fluid balance. Although applying positive pressure with the bag-mask is sufficient in most cases, severe cases usually need endotracheal intubation if the upper airway obstruction is not rapidly relieved[6]. For patients requiring intubation and mechanical ventilation, the sedation should be titrated to avoid patient-ventilator asynchronies. Diuretics have been also widely used in case of NPPE[10].

The outcome of patients with NPPE is favorable within 12 to 48 hours[7,14,19]. However, severe cases such as our patient may have more complicated course with difficult weaning and even recurrent pulmonary edema[13]. To the best of our knowledge, using dexmedetomidine for weaning from mechanical ventilation has not previously reported in patients with negative pressure pulmonary edema. Dexmedetomidine is a highly selective α2-adrenoceptor agonist that has been increasingly used in critically-ill patients[20]. Recent studies highlighted the effectiveness of this drug in reducing the duration of mechanical ventilation in patients with difficult weaning[21,22]. Using dexmedetomidine enabled early weaning and extubation of our patient.

CONCLUSION

Negative pressure pulmonary edema is a rare postoperative complication seen in patients with severe and sudden upper airway obstruction. Early recognition is of paramount importance. Securing patent airway is life-saving. Oxygenation and applying positive pressure ventilation are the cornerstone of the treatment. All required investigations

should be performed to exclude other diagnosis that can induce pulmonary edema (ischemic heart disease, acute heart failure, etc). Although challenging, early weaning should be considered once the oxygenation improves.

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