

CASE REPORT

Development of negative pressure pulmonary oedema secondary to postextubation laryngospasm

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ABSTRACT

Negative Pressure Pulmonary Oedema (NPPE) immediately after general anaesthesia is a rare but life threatening complication, caused by an increased fluid in the interstitial spaces and alveoli due to forced inspiratory efforts against tightly closed glottis. Once developed, it impairs gas exchange and causes hypoxemia and if not treated promptly may lead to respiratory failure. Management involves maintaining airway, diuretics and positive pressure ventilation. Affected cases recover completely with appropriate treatment but death may occur if treatment is delayed. We present here three cases that developed post-extubation NPPE after short spells of laryngospasm. All of the three cases recovered completely after management with diuretics and ventilatory support with added PEEP.

Key Words: Negative pressure pulmonary oedema; extubation; postoperative complication

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INTRODUCTION

NPPE, also called Post-obstruction Pulmonary Edema (POPE), is a complication that can develop in the immediate postoperative period. The first case reports of NPPE were published by Oswalt et al. in 1977¹. We encountered three cases of NPPE over a period of eighteen years, (incidence 0.06% of all general anaesthetics) which is almost similar to other workers^{2,3}. It is a rare condition, but it may result in a fatal outcome if diagnosis and treatment are delayed.

CASE 1:

A 22 years old, young soldier, ASA-I, underwent planned haemorrhoidectomy under thiopentone - O₂/N₂O - isoflurane - relaxant anaesthesia. The patient was intubated and inj. pethidine 50mg was given IV. At the end of the procedure, the effects of relaxant were reversed and extubation was done with the patient fully recovered from anaesthesia. A few seconds later the patient had a brief spell of laryngospasm after which his SpO₂ started falling.

Positive pressure was applied with mask and 100% O₂ but SpO₂ kept on falling. Chest auscultation showed bilateral fine crepts, so it was decided to re-intubate the patient. On laryngoscopy frank blood tinged froth was noticed coming out of the glottis. A diagnosis of NPPE due to post-extubation laryngospasm was made, the patient was given relaxants, IPPV continued and shifted to ICU for ventilatory support. Portable X-ray chest showed bilateral opacities of acute pulmonary oedema. ECG trace and cardiac enzymes were ordered and found to be within normal limits. After about 4 hours of ventilatory support chest auscultation confirmed his complete recovery from pulmonary oedema so the relaxant was reversed, he was weaned off the ventilator and extubated. He recovered completely without any sequelae.

CASE 2:

A 4 years old female child, ASA-1, weighing 15 kg, was scheduled for repair of a tongue laceration under general anaesthesia in emergency. Child had been NPO for 6 hours

before the procedure. She was induced with thiopentone 60mg and intubated orally after relaxation with inj. Succinylcholine 20mg. A throat pack was placed in pharynx. Anaesthesia was maintained with O₂/N₂O, isoflurane and inj. Tracrium™. At the end of procedure, the throat pack was removed and extubation was done after thorough suction. The patient went into severe laryngospasm after extubation. Airway was maintained and 100% O₂ was given with face mask but her SpO₂ dropped rapidly and she developed bradycardia with a heart rate of 50/min. Classic signs of NPPE were present e.g. bilateral fine crepts and bilateral diffuse opacities on chest X-ray. Inj. atropine 0.2 mg IV was repeated twice and the patient was re-intubated. Laryngoscopy revealed frank blood tinged froth from the larynx. IPPV with 100% O₂ improved the SpO₂. She was sedated and relaxed and later on shifted to pediatric ICU for ventilatory support with a PEEP of 4 cm of H₂O. After 4 hours of ventilatory support lungs became dry and she was weaned off and extubated after reversal of the relaxants.

CASE 3:

A 25 years old male, ASA-1, a moderate smoker for the last eight years, was planned for a testicular biopsy for infertility, under GA. The course of anaesthesia with intubation and IPPV remained uneventful. At the end of the procedure, the relaxant was reversed and extubation was done. The patient developed severe laryngospasm soon after extubation and his SpO₂ started falling. Ventoline™ nebulisation was done. Laryngospasm did not respond to any medication or maneuver. Administration of 100% O₂ by mask failed to raise SpO₂ to normal levels, so he was re-intubated to enable us positive pressure ventilation with PEEP. During intubation typical pink froth was noticed to fill the mouth cavity. Auscultation of the chest revealed bilateral rales. IPPV was continued and he was shifted to ICU for ventilatory support. He was given inj. Dexamethasone 4mg IV, inj. Furosemide 40mg IV and ventilatory support with 8cmH₂O of PEEP. Eight hours of ventilation dried his lungs, so he was weaned off the ventilator. His postoperative investigations revealed no cardiac, pulmonary or neurological deficit.

DISCUSSION

NPPE, also called POPE, results from transudation of fluid, first from pulmonary capillaries into interstitial spaces and then into the alveoli. It occurs after complete obstruction of the airway usually due to a brief but severe laryngospasm. Young healthy muscular patients, undergoing head and

neck surgeries and/or painful procedures are more prone to this condition. NPPE is a potentially dangerous condition with a multifactorial pathogenesis. The central mechanism is a large inspiratory force generated against an obstructed or closed upper airway. The resultant decreased intrathoracic pressure leads to increased venous return to the right side of the heart and increased hydrostatic pulmonary capillary pressure. Elevation of pulmonary capillary pressure results in decreased left ventricular compliance that may result from the right ventricular distention and shift of the cardiac septum to the left. The negative intrathoracic pressure also results in an increased afterload imposed on the left ventricle, causing a further decrease in left ventricular stroke volume.^{4,5} The autoPEEP during obstruction does not allow fluid to transudate, but as soon as airway opens, this autoPEEP is lost and under the effects of increased interstitial pressure, fluid pours first into interstitium and then into the alveoli. Fortunately, because of the lung's unique ultra structure and its capacity to increase lymph flow, the pulmonary interstitium usually accommodates large increases in capillary transudation before interstitial partial pressure becomes positive. When this reserve capacity is exceeded, pulmonary edema develops^{4,5}.

Pulmonary edema is often divided into four stages:

- Stage I: Only interstitial pulmonary edema is present.
- Stage II: Fluid fills the interstitium and begins to fill the alveoli.
- Stage III: Alveolar flooding occurs; many alveoli are completely flooded with no air.
- Stage IV: Marked alveolar flooding spills over into the airways as froth.

All of our cases of NPPE involved post-extubation laryngospasm. Other mechanical causes of upper airway obstruction resulting in NPPE reported in literature include hanging, laryngeal growths, strangulation, sleep apnea, and biting down on or kinking of the endotracheal tube while intubated.⁶⁻⁸ Croup and epiglottitis are common causes of upper airway obstruction leading to NPPE in children.⁹ Warner et al¹⁰ reported NPPE developing after administration of muscle relaxants at the beginning of an inhalation induction of anesthesia in healthy infants. Other causes of airway obstruction in unconscious patient may be tongue fall against the posterior pharynx, glottic edema, secretions, vomitus or blood in the airway, foreign body

like forgotten throat pack or external pressure on the trachea most commonly from a neck hematoma.

Signs and symptoms of NPPE include tachypnea, shortness of breath, pulmonary rales, frothy sputum production, decreased oxygen saturation, and evidence of upper airway obstruction. Chest radiographs may show signs of pulmonary edema.

None of the patients on whom we have reported had any history of cardiac disease and each had a negative preoperative physical exam⁶. After establishing a noncardiogenic etiology with relative certainty, the differential diagnosis should include an aspiration of gastric contents, adult respiratory distress syndrome (ARDS), volume overload, anaphylaxis, and airway obstruction. Perhaps the hardest to differentiate from the diagnosis of post obstructive pulmonary edema is that of aspiration of gastrointestinal contents. This is due to the fact that the onset of both processes may closely resemble each other (struggling patient, difficult intubation) and the clinical presentation (wheezing, dyspnea, hypoxia) is very often identical, but this condition is difficult to treat and its mortality is very high^{9,10}.

Airway obstruction should be managed by giving supplemental oxygen and opening the airway. If these maneuvers fail then refractory laryngospasm should be treated aggressively with a small dose of succinyl choline (10-20 mg) and temporary positive pressure ventilation with 100% oxygen. Some anaesthesiologists routinely use IV lignocaine 10-20 mg at the time of extubation to prevent severe coughing and laryngospasm¹¹. Endotracheal intubation may occasionally be necessary to reestablish ventilation. If pulmonary edema develops it is similar to cardiogenic pulmonary edema and is managed in a similar way but reintubation and positive pressure ventilatory support with PEEP are extremely useful and full recovery from pulmonary edema occurs without any residual pulmonary or cardiac damage in most of the cases.

CONCLUSION

Postextubation laryngospasm in the immediate postoperative period is an important complication and it may lead to significant morbidity or mortality like pulmonary edema, cardiac arrest, brain damage or death. Prompt establishment of airway, use of short acting muscle relaxant to relieve

severe laryngospasm and IPPV with PEEP may be life saving in these situations.

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