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Case Report

Anaesthesia Section

Acute Pulmonary Oedema: A Post-Operative Complication Due to Neostigmine and Post Obstructive Pulmonary Oedema in a Case of Tonsillectomy

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ABSTRACT

Acute pulmonary oedema has been described in relation to perioperative period. The aetiology may be multifactorial and its management poses a challenge to the anaesthesiologist. Its occurrence in a normal healthy person with no other medical history makes the diagnosis difficult. The causes of pulmonary oedema are cardiac failure, fluid overload, airway obstruction, acid aspiration, gas embolism, anaesthetic drugs, sepsis, anaphylactic reaction and reaction to blood & blood products. Early detection, prompt management by an anaesthesiologist will help to prevent further postoperative complications. We report a case of 9-year-old male child, posted for tonsillectomy under general anaesthesia, who developed acute pulmonary oedema following extubation after reversal with neostigmine and how we managed it successfully.

Keywords: Airway obstruction, Extubation, Negative pressure pulmonary oedema

CASE REPORT

A 9-year-old male child, weighing 30 kg with history of recurrent upper respiratory tract infection, diagnosed as chronic tonsillitis was posted for tonsillectomy under general anaesthesia. Clinical examination and blood investigations were within normal limits and physical status (ASA- 1), Consent was taken, adequate starvation confirmed. Premedication was given with IV metoclopramide 10 mg, glycopyrrolate 0.2mg, midazolam 0.5mg, pentazocine 15 mg and induced with IV thiopentone 200 mg, suxamethonium 60 mg followed by intubation with 6.0mm ID cuffed red rubber endotracheal tube and oropharyngeal packing. Maintenance of anaesthesia was with nitrous oxide: oxygen 60:40%, halothane and vecuronium. Intraoperative monitoring included ECG, pulse oximetry which was within normal range during the surgery that lasted for 45 minutes with minimal blood loss and 250 ml of ringer lactate given. At the end of surgery, after removing oropharyngeal pack and thorough suctioning, reversal with neostigmine 2 mg and glycopyrrolate 0.4 mg was attempted and patient was extubated with $Sp0_2$ 98%.

The patient was still on table waiting to be shifted to PACU when he became tachypnoeic, restless and developed wet cough with pink frothy sputum and SpO_2 92%. He was administered 100% oxygen (SpO_2 98%). Chest auscultation revealed bilateral crepitations. As the patient was in respiratory distress he was reintubated with intermittent positive pressure ventilation. The tonsillar fossae were examined, no bleeding seen. IV Hydrocortisone 100 mg, Dexamethasone 8 mg, Furosemide 20 mg, Deriphylline 2 cc was given. Suctioning of the trachea was done at regular intervals. As the patient was fighting the tube nitrous oxide: oxygen 50: 50% and Vecuronium was given. SpO_2 was 98%. Urinary catheterisation done. After 30 minutes, the patient's condition improved. With vitals stable and oxygen saturation maintained, reversal was attempted once again after return of spontaneous respiration, with neostigmine (2 mg) and glycopyrrolate (0.4 mg) diluted in 10 ml. After injecting 5 ml, the patient again developed pink frothy secretions through the endotracheal tube hence reversal was stopped. Since the patient was coughing and biting on the tube, he was given atracurium which is a short acting muscle relaxant with IPPV. Chest auscultation revealed bilateral crepitations. IV Aminophylline 125 mg slowly, Chlorpheniramine maleate 2 cc and Deriphylline 1 cc given. Endotracheal suctioning, chest physiotherapy with propped up position was given. Gradually, the crepitations reduced and eventually disappeared and the patient was extubated after two and a half hours without any reversal agent with SpO_2 98% on air.

DISCUSSION

Pulmonary oedema occurring under general anaesthesia is a rare complication, which could be cardiogenic, non-cardiogenic or negative pressure pulmonary oedema (NPPE) [1,2]. Postoperative pulmonary oedema is a well recognized complication of upper airway obstruction [3]. Post obstructive pulmonary oedema or NPPE is a life threatening clinical scenario in which immediate onset pulmonary oedema develops after upper airway obstruction. Two types of NPPE have been described, type 1 is associated with forceful inspiratory effort in the context of an acute upper airway obstruction whereas type 2 occurs after relief of a chronic partial airway obstruction [4]. NPPE following upper airway obstruction is well documented [5]. It has been reported following foreign body aspiration and the relief of airway obstruction from croup and epiglottitis in children and also seen after relief of a chronic partial airway obstruction following surgical intervention like post adenotonsillectomy or upper airway tumour removal. It is caused by significant fluid shifts due to changes in intrathoracic pressure [6]. The mechanics of oedema formation are unclear and may be due to increase in hydrostatic forces generated by high negative intrathoracic pressure or by increased permeability of the alveolar capillary membrane [3]. The effects of hypoxia and a hyperadrenergic state are thought to contribute to the NPPE occurring with upper airway obstruction [7]. In our case of tonsillectomy, NPPE cannot be ruled out as it is known to occur within minutes of either development of acute upper airway obstruction or surgical relief of the obstruction.

Some anaesthetic drugs are known to cause pulmonary oedema, although little is known about the mechanism involved in drug related NPPE [8,9]. Neostigmine being a cause of pulmonary oedema is known. Opioids are known to cause pulmonary oedema. Other drugs like salicylates, tocolytics, methadone, naloxone, protamine, insulin, lidocaine, bleomycin, amiodarone could be related to NCPE

[10]. Propofol [11] and ondensetron administration is known to be a cause of Non cardiogenic pulmonary edema [10,12].

Clinical manifestations of NPPE usually present immediately but can occur several hours later. Signs and symptoms of respiratory distress are always present, but frothy pink sputum is the hallmark sign of NPPE. Auscultation reveals rales and occasionally wheezes from fluid compressed airways [2]. Occurrence of Non cardiogenic pulmonary edema is related to the time proximity of the administration of the drugs and pathogenesis involves both a direct cytotoxic insult to the lung epithelial cells and induction of cytokine triggered inflammatory response [9] although not much literature is available on neostigmine causing Non cardiogenic pulmonary edema. In our patient, twice after administering neostigmine, he developed secretions and crepitations and ultimately pulmonary oedema.

Neostigmine, a reversal agent is an antagonist to non-depolarising muscle relaxants. Onset of action is 7–11 minutes and elimination $t_{1/2}$ is 80–120 minutes [13]. To counteract its muscarinic effects, neostigmine is administered along with an appropriate dose of an anti-cholinergic agent like atropine or glycopyrrolate to avoid the undesirable muscarinic side-effects. A premixed combination of neostigmine and glycopyrrolate (myopyrrollate) is available.

The muscarinic side effects are increased salivation, excessive bronchial secretions, bronchospasm, increased intestinal motility, bradycardia, conduction block (sinus node depression, atrio-ventricular block). The pulmonary side-effects viz. bronchospasm, bronchiolar constriction and increased bronchial secretions could lead to pulmonary oedema [13]. In literature, it is known that over dosage of neostigmine may induce cholinergic crisis characterized by excessive salivation, increased bronchial secretions, bradycardia or tachycardia, cardiospasm, bronchospasm [9]. Extreme high doses may produce CNS symptoms of agitation, fear or restlessness and death may result from cardiac arrest or respiratory paralysis and pulmonary oedema. If there is respiratory depression, breathing must be assisted or controlled. In our case, the patient developed pink frothy secretions, twice immediately after administration of neostigmine used for reversal, hence a high suspicion to cause pulmonary oedema.

Management of NPPE will depend on the severity of pulmonary oedema and associated hypoxia [14]. Maintenance of a patent upper airway and administration of supplemental inspired oxygen with diuretics helps resolve the pulmonary oedema [15]. CPAP is useful in spontaneously breathing patients. Occasionally, mechanical ventilation of the lungs with PEEP may be required. We managed our patient with oxygenation and ventilation with an endotracheal tube, steroids, diuretics, antihistaminics, bronchodilators and by avoiding neostigmine for reversal and during the second time extubation.

CONCLUSION

Hence, we conclude, that the likely cause for postoperative acute pulmonary oedema was due to neostigmine, however, NPPE (type 2) following relief of upper airway obstruction, post tonsillectomy, cannot be ruled out.

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