



MANAGEMENT OF NEGATIVE PRESSURE PULMONARY EDEMA IN POST EXTUBATION PERIOD, AFTER A PAROTID HEMANGIOMA RESECTION SURGERY.

Anaesthesiology

Dr Hari Kiran Khandavilli*

MBBS, DNB Anaesthesiology *Corresponding Author

Dr Maninder Kaur MBBS, MD Anaesthesiology

ABSTRACT

Post operative pulmonary edema is an uncommon entity resulting in morbid complications unless diagnosed and treated early. Causes for post operative pulmonary edema can be patient factors like preexisting cardiac conditions causing cardiogenic type of pulmonary edema, preexisting airway disorders resulting in obstruction leading to non-cardiogenic type of pulmonary edema. Also, iatrogenic factors like over enthusiastic fluid administration and excessive manipulation of airways during the surgery resulting in edema of airways or reflex glottis closure during post-operative period, immediately after extubation may lead to Negative Pressure Pulmonary Edema (NPPE). NPPE is non cardiogenic type of pulmonary edema usually seen in post extubation period, where pulmonary edema is resulted due to high negative intra thoracic pressure created by patient himself during strenuous inspiratory efforts against a closed glottis. Prompt diagnosis and appropriate timely management improves the prognosis of this condition.

KEYWORDS

Negative Pressure pulmonary edema, Post operative Pulmonary Edema, Post obstruction pulmonary edema, Extubation Complications

INTRODUCTION:

Postoperative pulmonary edema is an uncommon complication in post operative period with numerous etiological factors. Several causes have been described which could possibly lead to postoperative pulmonary edema but no specific preoperative and intraoperative criteria can be used as predictors for development of pulmonary edema in the post operative period⁽¹⁻⁴⁾.

Negative pressure pulmonary edema (NPPE) is a type of non cardiogenic pulmonary edema caused by acute negative intrathoracic pressure produced by patient⁽⁵⁻⁷⁾. It is usually seen in the immediate post operative period due to post extubation laryngospasm. Other causes of obstruction being hanging, laryngeal tumor, strangulation, sleep apnea, Biting of endotracheal tube while intubated⁽⁸⁾, croup and epiglottitis especially in children⁽⁹⁾ administration of muscle relaxant at the beginning of an inhalational induction of anesthesia caused by premature paralysis of glossal muscles before diaphragm⁽¹⁰⁾ following aspiration of pneumothorax or massive pleural effusion.

The overall incidence of NPPE is less than 0.1% in all surgeries performed under general anesthesia^(11,12) while the incidence of development of pulmonary edema in acute upper airway obstruction (type I NPPE) ranges from 9.6-12% and that in chronic airway obstruction (type II NPPE) is 44%⁽¹³⁾. Iatrogenic causes or pre existing respiratory conditions may increase the chances of obstruction in the patient.

More commonly seen in young and healthy males who are more predisposed to major negative pressure differences⁽²⁾.

We report a case which recently happened in our Institute where a patient was diagnosed NPPE immediately after extubation and was successfully managed by early intervention.

CASE REPORT:

A 19 year-old male (ASA-I, height 168cm, weight 65.0kg) was posted for resection of Hemangioma located in the plane between Masseter and Parotid gland, under General Anesthesia. Patient was an occasional smoker and had an Upper Respiratory tract infection with productive cough one week before surgery but, on the day of surgery, patient was afebrile with no cough.

Airway examination revealed adequate mouth opening, Mallampatti I, normal thyromental distance, and full neck range of motion, with no abnormal and artificial denture.

On the day of surgery, patient was nebulised with Normal saline and aspiration prophylaxis given with Tab. Ranitidine 150mg was given.

Premedication with Inj Glycopyrrolate 0.2mg, Inj Ondansetron 6mg

and Inj fentanyl 100 microgram was given. After pre oxygenation with 100% Oxygen for 3 minutes patient underwent a smooth induction with 120 mg Propofol and was intubated with 8.5 sized cuffed Endotracheal tube (ETT) after giving 6mg of Vecuronium bromide. Maintenance of anesthesia was by Oxygen, Nitrous Oxide, Sevoflurane and intermittent bolus of Vecuronium 1mg. Total duration of surgery was 6 hours and is uneventful with minimal blood loss of 200 ml. Injection Fentanyl 25 micrograms was repeated after 2 hours, 4 hours from the beginning of surgery.

After the surgery, upon emergence from anesthesia patient was reversed with Inj. Neostigmine 3mg and 0.5mg Glycopyrrolate. Patient was extubated smoothly after thorough suctioning of oral cavity.

Patient had sudden onset of dyspnoea and made repeated strenuous inspiratory efforts after removal of ETT. Attempts for assisting ventilation with the mask was unsuccessful and rapid decrease in oxygen saturation was observed.

Laryngoscopy was done and complete approximation of vocal cords (Laryngospasm) with edema over the right vocal cord was observed. With difficulty, a gum elastic bougie was introduced through the vocal cords and an ETT of size 8 was passed over the bougie and Positive Pressure Ventilation was started.

Patient experienced a transient desaturation to 30% but gradually improved with Positive Pressure ventilation with 100% oxygen. Chest auscultation revealed B/l coarse crepitations and frothy pink secretions were noted filling the endotracheal tube. A diagnosis of Negative Pressure Pulmonary Edema was made.

Immediately treatment was started with

- Positive Pressure ventilation with 100% Oxygen
- Inj. Lasix 30 mg i.v
- Inj Morphine 5 mg i.v
- Injection Hydrocortisone (100)mg i.v
- Intermittent suctioning through ETT

Gradually the amount of fluid from the ETT and crepitations on auscultation got subsided.

After 5 hours from re-intubation, when patient was comfortable and was able to achieve adequate Tidal volume on spontaneous ventilation, extubation was done. Patient was observed for 30 minutes and discharged to ward. Post extubation, a chest X-ray was advised which revealed mild pulmonary edema with hazy alveolar opacity in both perihilar regions (Fig 1). Oxygen supplementation was advised through a venturi mask at a Fio2 of 40% over night. Prophylactic Antibiotic therapy was started.

DISCUSSION:

Pulmonary Edema in post operative period can broadly be classified as Cardiogenic and Non cardiogenic, where incidence of Cardiogenic pulmonary Edema is high in patients with pre existing cardiac conditions. Moderately over quantity of intravenous fluids can precipitate Cardiac dysfunction in these patients⁽¹⁴⁾. Besides fluid overload, there are other pathogenic mechanisms which can lead to this serious clinical presentation⁽¹⁵⁻¹⁷⁾.

Next to cardiogenic etiology, noncardiogenic pulmonary edema which can be due to fluid overload in the peri operative period or negative pressure pulmonary edema (NPPE) are the less common etiologies.

NPPE is an important clinical entity in immediate post-extubation period due to acute negative intrathoracic pressure produced following acute upper airway obstruction due to laryngospasm. It carries good prognosis if promptly diagnosed and is appropriately treated.

In our case, a long standing surgery with head turned to right side may be the cause for edema on the right vocal cord which provoked laryngospasm in post extubation period. Hypersensitive airway due to recent history of URTI also can contribute to laryngospasm. Deep inspiratory efforts made by the patient against the closed glottis caused pulmonary edema.

Our decision to reintubate the patient was based on the simple fact that, it would be easy to suction the fluid in airways through ETT, and to provide a positive pressure ventilation which is the basic management strategy to clear the flooded alveoli. Adding a diuretic, Furosemide hastened up clearing the fluid from alveoli and interstitium. Intravenous morphine has a venodilatory action which decreased the preload to heart, stopping further exudation of fluid from the pulmonary capillaries to interstitium. Adding PEEP 6 cm H₂O to the ventilator settings also helped in clearing the fluid from alveoli. Early diagnosis of the situation and appropriate management helped in preventing the progression of edema and an early recovery in this case.

REFERENCES:

1. Van Hoozen BE, van Hoozen CM, Alberton TE. Pulmonary considerations and complications in the neurosurgical patient: Pulmonary edema. In: Youmans JR, editor. Neurological surgery. Philadelphia, PA: WB Saunders; 1996. pp. 624–6.
2. Roth E, Lax LC, Maloney JV, Jr Ringer's lactate solution and extracellular fluid volume in the surgical patient: a critical analysis. *Ann Surg.* 1969;169:149–64. [
3. Finn JC, Rosenthal MH. Pulmonary edema in trauma and critically ill patients. *Semin Anesth.* 1989;8:265–74.
4. Khuri SF, Daley J, Henderson W, Barbour G, Lowry P, Irvin G, et al. The National Veterans Administration Surgical Risk Study: Risk adjustment for the comparative assessment of the quality of surgical care. *J Am Coll Surg.* 1995;180:519–31.
5. Kulka PJ, Issel R, Wiebalck A, Strumpf M, Gehling M. Delayed negative pressure pulmonary edema. *Anaesthesist.* 2003;52:132–6.
6. Newton-John H. Pulmonary oedema in upper airway obstruction. *Lancet.* 1977;2:510.
7. Fremont RD, Kallet RH, Matthay MA, Ware LB. Postobstructive pulmonary edema: A case for hydrostatic mechanisms. *Chest.* 2007;131:1742–6.
8. Liu EH, Yih PS. Negative pressure pulmonary oedema caused by biting and endotracheal tube occlusion—a case for oropharyngeal airways. *Singapore Med J.* 1999;40:174–5.
9. Bonadio WA, Losek JD. The characteristics of children with epiglottitis who develop the complication of pulmonary edema. *Arch Otolaryngol Head Neck Surg.* 1991;117:205–7.
10. Warner LO, Martino JD, Davidson PJ, Beach TP. Negative pressure pulmonary oedema: A potential hazard of muscle relaxants in awake infants. *Can J Anaesth.* 1990;37:580–3.
11. Patton WC, Baker CL, Jr Prevalence of negative-pressure pulmonary edema at an orthopaedic hospital. *J South Orthop Assoc.* 2000;9:248–53.
12. Deepika K, Kenaan CA, Barrocas AM, Fonseca JJ, Bikazi GB. Negative pressure pulmonary edema after acute upper airway obstruction. *J Clin Anesth.* 1997;9:403–8.
13. Goldenberg JD, Portugal LG, Weingarten RT. Negative-pressure pulmonary edema in otolaryngology patient. *Otolaryngol Head Neck Surg.* 1997;117:62–6.
14. Mangano DT, Browner WS, Hollenberg M, London MJ, Tubau JF, Tateo IM, et al. Association of perioperative myocardial ischemia with cardiac morbidity and mortality in men undergoing noncardiac surgery. The Study of Perioperative Ischemia Research Group. *N Engl J Med.* 1990;323:1781–8. [
15. Ayus JC, Arieff AI. Pulmonary complications of hyponatremic encephalopathy: Noncardiogenic pulmonary edema and hypercapnic respiratory failure. *Chest.* 1995;107:517–21.
16. Pandor A, Goodacre S, Harnan S, Holmes M, Pickering A, Fitzgerald P, et al. Diagnostic management strategies for adults and children with minor head injury: A systematic review and an economic evaluation. *Health Technol Assess.* 2011;15:1–202.
17. Suga K, Tsukamoto K, Nishigauchi K, Kume N, Matsunaga N, Hayano T, et al. Iodine-123-MIBG imaging in pheochromocytoma with cardiomyopathy and pulmonary edema. *J Nucl Med.* 1996;37:1361–4.