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Case Report

Recurrent negative pressure pulmonary oedema after tracheal extubation

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Introduction

Negative pressure pulmonary oedema (NPPO) is an uncommon complication of extubation of the trachea with an incidence of approximately 0.1%.¹ The most common cause of NPPO is laryngospasm occurring immediately postextubation.¹ The marked inspiratory efforts that follow upper airway obstruction due to glottic closure, lead to excessive negative intrathoracic pressures sometimes greater than –100 cm of water. This may cause pulmonary oedema and rarely haemoptysis.^{1–3} This report is the first to document clinical and chest X-ray findings consistent with alveolar oedema following tracheal extubation, twice in the same patient a few months apart.

Case report

A 26-year-old 70 kg healthy male patient, a case of external nasal deformity due to blunt trauma sustained 2 years ago, was subjected to elective rhinoplasty for the first time in the month of January and thereafter again in the month of June, the same year, as he continued to be symptomatic despite the first surgery. His medical history and physical examination were unremarkable except for symptoms and signs of nasal blockade and difficulty in breathing through his right nostril for a duration of 1 year. Details of the anaesthetic technique and subsequent management of the patient on the two occasions are elaborated in the following paragraphs.

Anaesthetic technique of first surgery

The first surgery was a rhinoplasty performed under general anaesthesia. The patient was premedicated in the operation theatre with Inj Glycopyrolate 0.2 mg, Inj Fentanyl 2 µg/Kg IV and Inj Midazolam 2 mg IV. He was induced with Inj Thiopentone. Endotracheal intubation and muscle relaxation were obtained with Inj Vecuronium 0.1 mg/Kg IV. The patient was maintained on oxygen, nitrous oxide and isoflurane in appropriate concentrations.

The tracheal intubation, anaesthesia, mechanical ventilation, and surgical procedure were uneventful. Total fluids given were 1500 ml Ringer lactate. The patient was haemodynamically stable throughout and maintained oxygen saturation of

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98–99%. Following surgery, the patient was reversed with Inj Neostigmine 3.5 mg IV and Glycopyrrolate 0.6 mg IV and the trachea was extubated after the patient was fully awake. Immediately thereafter, the patient had a breath-holding episode followed by rapid desaturation to a SpO₂ of 60% in the following minutes. The patient had clenched his teeth and his mouth opening was impossible. Insertion of an oropharyngeal airway was a futile exercise; mask ventilation was difficult and far from satisfactory. Inj Succinylcholine 100 mg IV stat was given, the patient was intubated and ventilation was assisted. Pink frothy secretions suggestive of pulmonary oedema appeared in the endotracheal tube. On auscultation, he had fine crepitations in the mid and lower zones of both lungs. The patient was shifted to ICU and put on mechanical ventilation in adaptive support ventilation (ASV) mode. The chest radiograph showed bilateral pulmonary infiltrates (Fig. 1). The patient was ventilated for next 4 h. His oxygen saturation improved and he was extubated while awake. The next morning he was shifted to the ward.

Anaesthetic technique of redo surgery

Next time the same patient underwent a redo rhinoplasty after 5 months due to persisting symptoms. Since with history of pulmonary oedema was known patient was evaluated for a cardiogenic cause however echocardiography was normal. Anaesthetic technique was identical. The surgery was uneventful however just following extubation, the patient again had a breath-holding episode followed by desaturation which required intubation. This was followed by pulmonary oedema with copious pink frothy secretions flooding out of the endotracheal tube just as had occurred during the first surgery. The patient was again mechanically ventilated for 4 h after which he stabilised. He was extubated while awake and thereafter shifted to the ward the next morning. The chest radiograph showed bilateral pulmonary infiltrates (Fig. 2).

Discussion

Oswalt et al are credited for the first description of negative pressure pulmonary oedema (NPPO).⁴ Many studies suggest that young, healthy, athletic males may be at an increased risk

of this complication.⁵ Many reports of pulmonary oedema secondary to acute upper airway obstruction after tracheal extubation have been published.^{2,4–6} The development of non-cardiogenic pulmonary oedema has been observed after different types of obstruction of the upper airways and it is more frequent in the presence of laryngeal spasm (50% of reported cases), which has an incidence of 0.05%–5% of anaesthetic procedures.⁷ The frequency depends mainly on the type of surgery, with greater incidence in ENT procedures.⁷ Approximately 11% of patients who develop laryngeal spasm go on to develop NPPO.⁷ During the episodes of upper airway obstruction, inspiratory efforts against a closed glottis (modified Mueller manoeuvre) may result in markedly negative pleural pressures (>–100 cm H₂O).⁸ The speed of onset, the appearance of pink frothy secretions after relief of obstruction and the relatively fast recovery are characteristics of NPPO.

The pathophysiology of NPPO is multifactorial. The major factor is marked negative intrapleural pressure produced by strong inspiratory efforts while the airway is obstructed. In young healthy patients, this negative pressure may be as great as –100 cm water.⁸ This increases venous return, right ventricular filling and pulmonary capillary blood volume. Interventricular dependence leads to increased left ventricular end diastolic and left atrial pressures, reducing pulmonary venous return and further increasing pulmonary capillary blood volume. The increased pulmonary blood volume, adrenergic mediated pulmonary vasoconstriction and hypoxic pulmonary vasoconstriction increase pulmonary hydrostatic pressure, while the negative intrapleural pressure also decreases the interstitial hydrostatic pressure. Direct injury to the alveolar capillary membrane increases permeability. All these increase the tendency for fluid to move out of the pulmonary capillaries, resulting in interstitial and alveolar oedema.⁶

The presentation is usually within minutes of airway obstruction but may not be evident until after relief of obstruction, hence it is also called postobstructive pulmonary oedema.⁸ Bhavani-Shankar reported airway bleeding and haemoptysis as a distinct complication, which may coexist with and/or be misdiagnosed as pulmonary oedema.³ Our patient did not have haemoptysis though he had clinical and radiological signs of pulmonary oedema.

Fig. 1 – The chest radiograph showed bilateral pulmonary infiltrates in first case.

Fig. 2 – The chest radiograph showed bilateral pulmonary infiltrates in redo case.

Emergence laryngospasm is the most commonly reported anaesthetic cause of airway obstruction and negative pressure pulmonary oedema. Laryngospasm during extubation can be prevented by extubating patients either deeply anaesthetised or fully awake, but not when in a light plane of anaesthesia.⁹

Most episodes of NPPO resolve within a few hours and oxygen therapy is usually all that is required. A short period of mechanical ventilation and positive end expiratory pressure may speed up recovery. The role of loop diuretics is not certain.⁵

Perhaps an oropharyngeal airway with a reinforced 'bite block' segment should be routinely inserted after endotracheal intubation. It should be removed only after extubation, particularly when patients are extubated awake. The oropharyngeal airway can prevent a patient from biting, damaging and completely occluding the endotracheal tube, and prevent severe NPPE and hypoxia. The oropharyngeal airway will also facilitate oropharyngeal suction and maintenance of a patent upper airway after extubation if patients are extubated 'deep'.

Use of laryngeal mask airway can possibly be encouraged instead of invasive endotracheal intubation in patient with known history of NPPO due to laryngospasm in subsequent surgeries requiring anaesthesia. However there are no studies conclusively to suggest that this strategy could help.

The uniqueness of this report is that NPPO has occurred both witnessed and documented in the same normal healthy adult on two occasions despite adequate precautions and sound scientific anaesthetic technique. There is also a recent report in which a patient had NPPO following a wrist laceration surgery and after stabilisation described a similar episode 10 years earlier following surgery for multiple gunshot wounds which was however undocumented.¹⁰ We herein describe the first reported case of recurrent negative pressure

pulmonary oedema in the same patient following extubation in our operation theatre.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Kapoor MC. Negative pressure pulmonary oedema. *Indian J Anaesth.* 2011;55:10–11.
2. Willms D, Shure D. Pulmonary edema due to upper airway obstruction in adults. *Chest.* 1988;94:1090–1092.
3. Bhavani-Shankar K, Hart NS, Mushlin PS. Negative pressure induced airway and pulmonary injury. *Can J Anaesth.* 1997;44: 78–81.
4. Oswalt CE, Gates GA, Holmstrom MG. Pulmonary edema as a complicated airway obstruction. *JAMA.* 1977;238(17):1833–1835.
5. Holmes JR, Hensinger RN, Wojtys EW. Postoperative pulmonary edema in young, athletic adults. *Am J Sports Med.* 1991;19:365–371.
6. Cascade PN, Alexander GD, Mackie DS. Negative-pressure pulmonary edema after endotracheal intubation. *Radiology.* 1993;186:671–675.
7. Visvanathan T, Kluger MT, Webb RK. Crisis management during anaesthesia: laryngospasm. *Qual Saf Health Care.* 2005 June;14(3):e3.
8. Lathan RS, Silverman ME, Thomas BL, Waters WC. Postoperative pulmonary edema. *South Med J.* 1999;92(3):313–315.
9. Mehta VM, Har-El G, Goldstein NA. Post obstructive pulmonary edema after laryngospasm in the otolaryngology patient. *Laryngoscope.* 2006;116(9):1693–1696.
10. Pathak Vikas, Hurtado Rendon Iliana S, Ciubotaru Ronald L. Recurrent negative pressure pulmonary edema. *Clin Med Res.* 2010;9(2):88–91.