Negative- pressure pulmonary oedema after septoplasty

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Citation

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Abstract

Negative pressure pulmonary oedema, an uncommon problem, occurs when large inspiratory force is generated against an obstructed upper airway resulting in decreased intra-thoracic pressure which increases venous return to the right heart and pulmonary capillary wedge pressure. It commonly involves young, healthy male patients undergoing general anaesthesia. Early diagnosis and prompt treatment are important to enhance recovery and prevent complications. We report a case of a 18 year old healthy, non-smoking male who developed negative pressure pulmonary oedema after septoplasty, and removal of neck swelling. With prompt diagnosis and appropriate treatment, the patient recovered well without significant sequelae.

INTRODUCTION

Negative-pressure pulmonary oedema (NPPE), first described by Oswalt in 1977 is a rare clinical entity [1]. The prevalence is less than 0.1% as a complication of all surgical procedures [2]. This form of non-cardiogenic pulmonary edema may result in immediate or delayed hypoxemia.

We report a case of NPPE following septoplasy and removal of a neck swelling. With prompt diagnosis and appropriate treatment, the patient recovered well without significant sequelae.

CASE REPORT

An 18 year old, healthy, non-smoking man, weighing 44 kg, was admitted for septoplasty and removal of a neck swelling. He had an unremarkable medical history and physical examination. He was pre-medicated with alprazolam, 0.5 mg and metoclopramide, 10 mg, given orally one hour before surgery. Glycopyrrolate, 0.2 mg was given as an anti-sialogogue and tramadol, 100 mg was given for analgesia. After preoxygenation for 3 minutes, anaesthesia was induced with thiopentone, 200 mg and vecuronium, 5 mg was used to facilitate endotracheal intubation. The throat was packed with wet gauze after intubation.

Anaesthesia was maintained with oxygen (O_2), nitrous oxide (N_2O), isoflurane and vecuronium. The procedure lasted approximately 60 minutes with minimal blood loss. A total of 700 ml of crystalloid was given as fluid replacement. The patient's intra-operative course was smooth with stable vital signs and oxygenation. At the end of the procedure, the throat pack was removed and the neuromuscular block was reversed with neostigmine, 2.5 mg and atropine, 1.2 mg. The patient was extubated after thorough oral suction and attainment of sufficient muscle power.

While shifting the patient to recovery room, breath holding was observed. The pulse oximeter was reapplied which revealed oxygen saturation of 30% and immediately, bag and mask ventilation was resumed and oxygen saturation came up to 100%. Chest auscultation revealed bilateral rhonchi. Deriphylline (etofylline 84.7 mg and theophylline 25.3 mg) and hydrocortisone, 100 mg were given. After a few minutes, the patient started breathing spontaneously. At this time, chest auscultation revealed bilateral basal crepts. Furosemide, 20 mg was given while the patient was ventilated with bag and mask. Suddenly, the patient coughed out copious amount of pink, frothy sputum suggestive of pulmonary oedema.

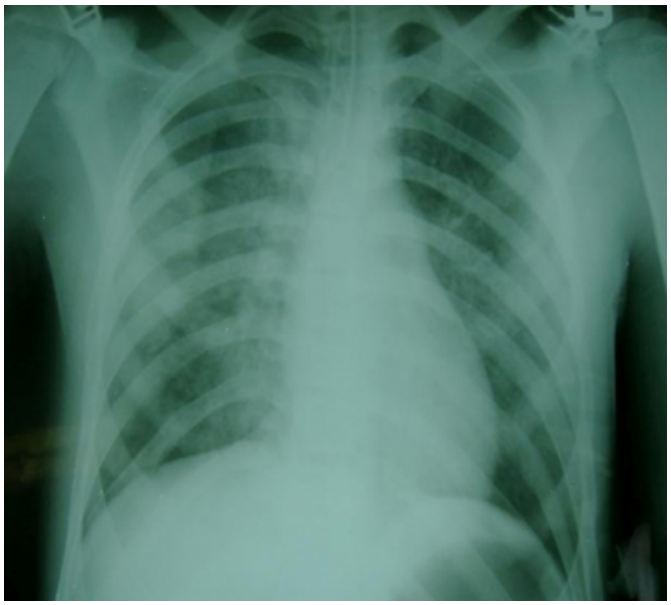
The patient was re-intubated after giving thiopentone and succinylcholine. Endotracheal suction was done which kept pouring pink frothy secretions. Chest auscultation was repeated which revealed bilateral coarse crepts. Furosemide, 40 mg was given. The patient was shifted to intensive care unit (ICU) for elective mechanical ventilation.

An immediate cardiac consultation was done which showed no obvious cardiac cause for patient's pulmonary oedema. The patient's arterial blood gas (ABG) report on arrival in ICU revealed a pH of 7.34 with carbon dioxide tension (pCO_2) of 53.2 mm Hg and an oxygen tension (pO_2) of 338.5 mm Hg with no base deficit. The chest radiograph revealed

bilateral diffuse pulmonary infiltrates consistent with pulmonary oedema (Figure 1).

Figure 1

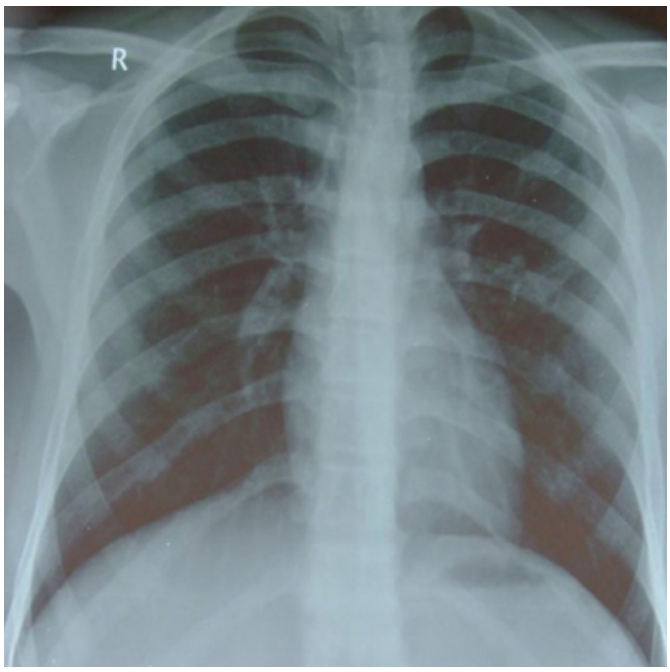
Figure 1: Chest radiograph showing bilateral diffuse pulmonary infiltrates consistent with pulmonary oedema.



After about 3 hours of ventilation, the patient was still not fully awake. Hypoxic brain injury was suspected and mannitol, 75g was given. The patient was extubated after 20 hours of ventilation. The post-extubation chest radiograph showed complete resolution of pulmonary edema (Figure 2).

Figure 2

Figure 2: Post-extubation chest radiograph showing complete resolution of pulmonary oedema.



DISCUSSION

NPPE is an uncommon problem, but serious enough to warrant quick recognition and effective treatment. NPPE has been estimated to occur in 11% of patients with clinically significant upper airway obstruction requiring treatment [3]. The pathogenesis of NPPE is multifactorial. The main mechanism is a large inspiratory force generated against an obstructed upper airway. The resultant decreased intra-thoracic pressure can increase venous return to the right heart and increase pulmonary capillary wedge pressure. In addition, hypoxia induces a massive sympathetic discharge, which can further increase the pulmonary capillary wedge pressure. Thus, the combination of increased venous return and pulmonary capillary wedge pressure favours the shift of fluid into the pulmonary interstitium with resultant pulmonary oedema [4].

In adults, about 50% of NPPE occurrences are due to postoperative laryngospasm [5]. When laryngospasm-induced airway obstruction occurs, large inspiratory force and high negative intrathoracic pressure is necessary to induce pulmonary oedema. This is consistent with the vulnerability of young healthy, athletic males to NPPE, as they have powerful respiratory muscles and increased chest wall compliance which can generate a negative intrapleural pressure in the range of 50 to 100 cm H_2O, as might have occurred in our case [6].

Early diagnosis and prompt treatment are important. Clinically, these patients present with decreased oxygen saturation, pink frothy sputum and manifestations of acute airway obstruction including stridor, suprasternal and supraclavicular retractions, use of accessory muscles of inspiration and panic in the facial expression [7]. As NPPE develops, chest auscultation usually reveals crackles and occasional wheezes. The typical chest radiograph will show diffuse interstitial and alveolar infiltrates [8]. The differential diagnosis includes acute respiratory distress syndrome, fluid overload, cardiac abnormalities and pulmonary embolism.

The first treatment priority is relief of the airway obstruction and correction of hypoxemia. The next step is to address the pulmonary edema with a diuretic unless the patient is hypovolaemic. Effective airway management and immediate treatment with oxygen and diuretics is sufficient in most cases. Persistent airway obstruction may necessitate an artificial airway, and acute respiratory failure would require artificial ventilation with oxygen and appropriate levels of positive end expiratory pressure (PEEP).

CONCLUSION

NPPE is post-extubation condition that is potentially dangerous if not recognized and treated promptly. It tends to affect young, healthy males after general anaesthesia. A high index of suspicion for the diagnosis and prompt treatment is required to avoid morbidity and mortality.

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