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Case Report

Negative pressure pulmonary oedema after total parotidectomy

Awad Ali M. Alawad*

Department of Surgery, Faculty of Medicine, University of Medical Sciences and Technology, Sudan

***Corresponding Author**
Dr. Awad Ali M. Alawad
Department of Surgery,
Faculty of Medicine,
University of Medical Sciences and Technology,
Sudan
E-mail: awadali82@hotmail.com

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Abstract

Introduction: Negative pressure pulmonary edema (NPPO) is an uncommon complication of extubation of the trachea mostly caused by laryngospasm. In literature few cases have been reported.

Case report: A 27-year-old male, weighing 75 kgs, ASA status class 1, underwent total parotidectomy for pleomorphic adenoma. Postextubation, the patient became restless, tachypnoeic and started desaturating. Soon after reintubation, pink, frothy fluid came out of the endotracheal tube, and a tentative diagnosis of NPPO was made. The condition improved with diuretics, oxygenation and steroids.

Result: Portable chest radiograph was taken, which revealed bilateral fluffy shadows with normal cardiothoracic ratio. ECG and arterial blood gas analysis were normal.

Conclusion: To conclude, acute pulmonary oedema associated with obstruction of the upper airways can aggravate low morbidity surgeries, affecting mainly young patients. The knowledge of this complication and, most importantly, its prevention are crucial.

1. Introduction

Negative Pressure Pulmonary Oedema (NPPO) is an uncommon complication that occurs during extubation of patients undergoing anaesthesia for a surgical procedure. It is most often seen in young, muscular adults capable of generating high negative intrathoracic pressures.

Negative Pressure Pulmonary Oedema (NPPE) was first demonstrated in 1927 by RL Moore in spontaneously breathing dogs exposed to resistive load[1]. The first description of the pathophysiological correlation between creation of negative pressure and the development of pulmonary edema was in 1942 by Warren *et al*[2]. In 1973, the relationship between pulmonary edema and upper airway obstruction in two children, who had croup and epiglotitis was reported by Capitanio *et al*[3]. In 1977 the report by Oswalt *et al* was the first showing the clinical significance of this phenomenon in three adult patients, who experienced the onset of pulmonary edema minutes to hours after severe acute upper airway obstruction[2]. From 1984 until 2001 several reports appeared in the literature discussing the phenomenon of acute, non cardiogenic pulmonary edema in response to acute or chronic upper airway obstruction.

We report a case of NPPO proved clinically and by investigations. The edema resolved completely in 2-12 hours after initiation of treatment.

2. Case report

A 27-year-old male, weighing 75 kgs, ASA status class 1, underwent total parotidectomy for pleomorphic adenoma. Laboratory investigations were within normal limits. General anaesthesia was induced with fentanyl 40 g, propofol 80 mg and vecuronium 4 mg intravenously. The patient's trachea was intubated with cuffed endotracheal tube of size 7.0 mm. Anaesthesia was maintained with O_2: N_2O (30:70), isoflurane and vecuronium bromide. Parotidectomy progressed uneventfully. The duration of surgery was 2 hours. Perioperatively, the patient received 1500 mL of Normal saline and his urine output was 300 mL. Neuromuscular blockade was reversed with neostigmine 2.5 mg and atropine 1.2 mg. The patient received diclofenac 50 mg intramuscularly for postoperative pain relief. Postextubation, the patient became restless, tachypnoeic and desaturating. 100% O_2 delivery was tried with anatomical face mask but because of inadequate seal, the continuous positive airway pressure could not be achieved. The endotracheal tube was reintroduced. On auscultation, there were fine crepitations in most of the lung zones. Soon after reintubation, pink, frothy fluid came out of the endotracheal tube, and a tentative diagnosis of NPPO was made. Furosemide 40 mg and dexamethasone 4 mg were given intravenously. SpO_2 improved to 94%. Portable chest radiograph was taken, which revealed bilateral fluffy shadows with normal cardiothoracic ratio (Figure 1). ECG and arterial blood gas analysis were normal. Gradually, the secretions decreased and SpO_2 progressed to 100%. Head end of the bed was elevated and fluid restriction was implemented. The patient was observed for another hour in the operation theatre and was shifted to recovery room after that as he remained stable. Repeat chest radiograph was normal (Figure 2) and he was discharged on the fifth postoperative day.

Figure 1: Chest X-ray showing pulmonary edema and normal size heart

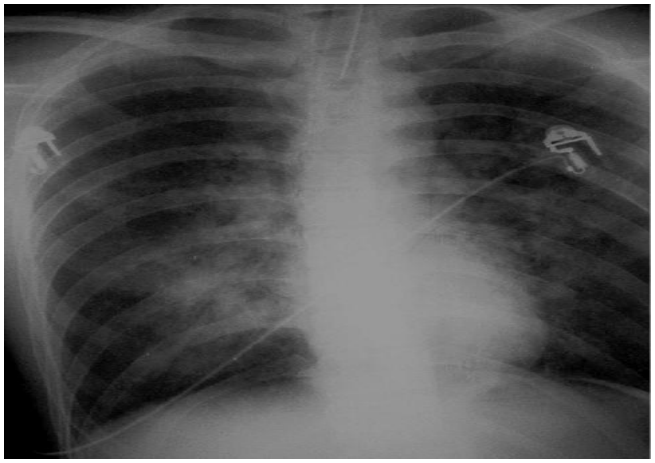
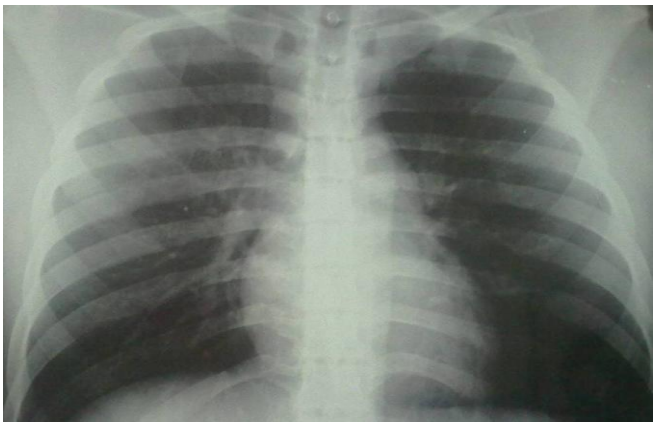


Figure 2: **Chest X-ray after treatment showing clearance of lung fields**



3. Discussion

Negative pressure pulmonary oedema (NPPO) is noncardiogenic and is of two types. Type I is of sudden onset following upper airway obstruction and Type II develops after surgical relief of chronic upper airway obstruction[4]. This is called NPPO because the laryngeal spasm, or other obstructive process in which the patient can inspire against the closed glottis (modified Móeller maneuver), is capable of generating an extremely negative intrapleural pressure (peaks of sustained inspiratory pressure between −50 cm H_2O and −100 cm H_2O, though the mean basal pressure is around −4 cm H_2O) which can trigger pulmonary oedema[5]. The hydrostatic forces are the primary mechanism behind postobstructive pulmonary oedema and that the alveolar epithelium remains functionally intact in acute postobstructive pulmonary oedema.

In Type II NPPE, it appears that the obstructive lesion produces a modest level of PEEP (positive end expiratory pressure) and increases end expiratory lung volume. Relief of the obstruction removes the PEEP and return lung volumes are preserved to normal. The sudden removal of PEEP leads to interstitial fluid transudation and pulmonary oedema. On radiological evaluation, NPPE is characterized by bilateral centralized pulmonary oedema, a wide vascular pedicle and a normal cardiothoracic ratio when the radiograph was obtained 15-165 min after the symptoms developed[6].

Clinical manifestations of NPPO usually present immediately but can occur several hours later. Signs and symptoms of respiratory distress are often present, but frothy, pink sputum is the hallmark sign of NPPO. Auscultation reveals rales and, occasionally, wheezes from fluid-compressed airways. The chest radiograph typically shows diffuse interstitial and alveolar infiltrates appearing as “whited out” areas. Tachycardia, hypertension, and diaphoresis reflect sympathetic nervous stimulation. When clinical signs and symptoms present, the anesthesia provider must form a differential diagnosis which include acute respiratory distress syndrome, intravascular volume excess, cardiac abnormalities, and pulmonary embolus[7].

After the diagnosis of NPPO has been made, treatment is directed toward reversing hypoxia and decreasing the fluid volume in the lungs. Maintaining the airway and providing supplemental oxygen is usually all that is required for a positive outcome. If oxygenation does not improve in the intubated patient, positive end-expiratory pressure should be administered to promote alveolar expansion. If oxygenation does not improve in the nonintubated patient, the immediate intubation with positive pressure ventilation and positive end-expiratory pressure is necessary. The use of diuretic therapy to remove excess intrapulmonary fluid is controversial[8]. It is possible for the patient to be hypovolemic, and consequently diuretic therapy would only worsen the clinical condition. In the situation of the patient biting down on the endotracheal tube, a small dose of succinylcholine (0.1-0.2 mg/kg) is needed to release the patient’s bite on the tube.

4. Conclusion

To conclude, acute pulmonary oedema associated with obstruction of the upper airways can aggravate low morbidity surgeries, affecting mainly young patients. Early diagnosis, awareness of the anesthetist, careful surgical manipulation of the upper airway, vigilance of the nurses in the recovery room and in the wards contribute to the successful management of this syndrome. The knowledge of this complication and, most importantly, its prevention are crucial.

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