Negative Pressure Pulmonary Edema following General Anesthesia- A Case Report

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Abstract

Negative pressure Pulmonary Edema (NPPE) is a well- recognized but rare complication secondary to upper airway obstruction such as laryngeal spasm during emergence from general anesthesia. We herein report a case of NPPE in a young healthy adult male following minor orthopedic surgery. With prompt diagnosis and treatment he recovered well without significant sequelae. Young healthy males may be at increased risk for laryngospasm-induced pulmonary edema because they have the ability to generate large negative intrathoracic pressures. It has however been described even in neonates. The condition must be promptly recognized to minimize morbidity and mortality.

Key words

Negative pressure, pulmonary edema, airway obstruction, respiratory distress, laryngospasm

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Introduction

Negative- pressure Pulmonary Edema (NPPE), first reported by Ostwald in 1977, is a rare clinical entity.[1] The prevalence is less than 0.1% as a complication of all surgical procedures.[2] It is most common in young, healthy male patients undergoing surgery under general anesthesia.

Case Report

A 25 year old healthy, man was admitted with left sided Galleazi fracture for open reduction and internal fixation. He weighed 70 Kg and had an unremarkable medical history and physical examination. His hematologic and biochemical investigations were within normal limits. He was premedicated with Lorazepam 2 mg in the night; Pethidine 75 mg and Promethazine 25mg intramuscularly, 1 hr before shifting to OR. Anesthesia was induced with intravenous Sufentanil 0.1µg/Kg and Propofol 2mg/Kg. Vecuronium 0.1 mg/Kg was used to facilitate tracheal intubation. Anesthesia was maintained using oxygen, nitrous oxide and halothane (0.2- 0.5%). Surgery lasted 90 min and endotracheal tube was removed after appropriate neuromuscular blockade

antagonism. Immediately after extubation the patient started coughing and bilateral wheeze was heard by auscultation. The respiratory rate was 40/min, oxy-hemoglobin saturation dropped from 98% to 70% and evidence of paradoxical respiration was noted. The patient developed laryngospasm, and attempts to deliver 100% oxygen by positive pressure using anatomical face mask were met with marked resistance to airflow. He was reintubated without difficulty and copious amounts of pink, frothy secretions filled the endotracheal tube. A portable chest radiograph taken showed bilateral perihilar infiltrates consistent with pulmonary edema. (Fig1). He was given furosemide 40 mg, hydrocortisone 100 mg and maintained on spontaneous ventilation with a CPAP mask and a valve of 10 cm water.

Within 2 hours, the patient improved clinically and had an oxy-hemoglobin saturation of 90-93%. After 4 hours, there was further improvement in saturation to 96%.

He was extubated, oxygen delivered through Hudson mask at 6L/min and shifted to post- operative ward with stable vitals.

Chest X-ray 24 hours later revealed clear lung fields (Fig 2) and ECHO done revealed normal LV function. He was discharged 3 days later without recurrent cough, stridor or dyspnea. At 5 months after surgery he is free from respiartory symptoms and does regular exercise without difficulty.

Discussion

In 1927, animal studies demonstrated that acute airway obstruction could lead to pulmonary odema.[2] In 1977, a report was published of adult patients who experienced pulmonary odema minutes to hours after severe acute airway obstruction. Anderson et al reported 3 cases of NPPE in young athletic male patients undergoing minor orthopedic surgeries. Deepika et al reported 30 cases of NPPE over a 4- year period and found a prevalence of 0.094% covering a wide range of surgical specialties.

NPPE has been classified into two types:

Type I- occurs immediately after the onset of a precipitating event
The causes of Type I NPPE are

Post extubation laryngospasm, patient biting down the endotracheal tube while intubated, endotracheal tube obstruction, intra operative direct suctioning of endotracheal tube adapter, postoperative vocal cord paralysis

Type II- develops after resolution of chronic upper airway obstruction

The causes of Type II NPPE are
Post- tonsillectomy/ adenoidectomy, post- removal of upper airway tumour,choanal stenosis, hypertrophic redundant uvula

Risk factors for NPPE
Obesity, short neck, vocal cord paralysis, history of obstructive sleep apnea, nasal, oral or pharyngeal surgery

Four basic mechanisms of pulmonary edema are [5]
increased pulmonary capillary pressure, Plasma colloidal osmotic pressure, increased pulmonary capillary permeability and decreased intra thoracic pressure

The pathogenesis of NPPE is multi factorial.

NPPE occurs when a large, negative intra thoracic pressure is generated against an obstructed airway, causing fluid to shift into the lung interstitium. Young healthy, athletic male patients appear to be at increased risk for this disorder.

Main mechanism is a large inspiratory force generated against an obstructe upper airway (modified Muller's maneuver). [6] Peak negative intrapleural pressure may exceed 50 cm to -100 cm H2 O during acute airway obstruction, especially after vigorous inspiratory efforts. Resultant decrease in intra thoracic pressure can increase venous return to the right heart and pulmonary capillary wedge pressure. Increase of right ventricular volume produces displacement of the ventricular septum towards the left ventricle, reducing its compliance. This lead to the increase wall tension of the left ventricle.

Starling's equation states that it is the balance of hydrostatic and colloid osmotic pressures across a variably permeable membrane that determines fluid flux and is responsible for pulmonary edema. Negative intra pleural pressure, increase in hydrostatic pressure gradient, with movement of fluid out of the pulmonary vasculature into the interstitial space. [7] In addition, hypoxia induces a massive sympathetic discharge, which can further increase the pulmonary capillary wedge pressure. Thus, the combination of increase venous return and pulmonary capillary wedge pressure favours the shift into the pulmonary interstitium with resultant pulmonary edema.

Early diagnosis and prompt treatment are important. Treatment consists of oxygenation, diuretics, morphine and glucocorticoids .Main problem associated with this complication is hypoxia, adequate oxy-hemoglobin saturation remains the primary goal of treatment. Maintenance of adequate oxygenation and a patent airway are the mainstays of management. Reintubation may be necessary, if laryngospasm is present and/or facemask ventilation with high

inspiratory oxygen are not satisfactory or are not sufficient to maintain a SpO2 greater than 95%. Fortunately, with early diagnosis and prompt treatment, most patients recover within one or two days without significant complications. The use of steroids is controversial, probably to lessen the pulmonary lesion due to physical damage caused to the alveoli and capillaries caused by high negative pressure.[8] Diuretics help in reducing the volume load on the lungs and may also help in improving the ventricular compliance. Pulmonary odema usually occurs after the relief of upper airway obstruction . Obstruction creates more positive pressures during expiration which serves as a form of "auto PEEP" to oppose transudation until obstruction is removed.[4] Prophylactic CPAP is recommended by some, efficacy has not been demonstrated.[4]

Conclusion

Negative Pressure Pulmonary Edema is a post extubation condition, potentially dangerous if not recognized and treated promptly. It tends to affect young, healthy athletic males after general anesthesia. A high index of suspicion for diagnosis is required. Since post anesthetic laryngospasm is the most frequent cause of the syndrome in adults, those involved in postoperative care should be particularly aware of this syndrome. Any patient who suffers acute airway obstruction should be observed closely for at least 12 hours for the appearance of this syndrome.[8]

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Fig 1

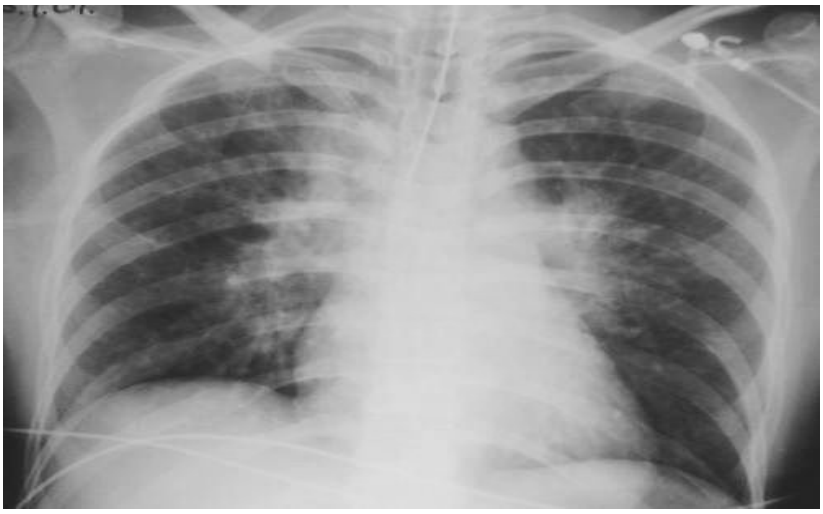

Fig 2