# Negative Pressure Pulmonary Edema Following Septorhinoplasty

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## ABSTRACT

Negative pressure pulmonary edema (NPPE) is a potentially fatal entity manifesting subsequent to general anesthesia. Pronounced inspiratory efforts against the obstructed upper airway leads to excessive negative intrathoracic pressure causing pulmonary edema with hypoxia, hypercarbia, acidosis, and hyperadrenergic state further contributes to its development. Careful management of the cases with risk factors, early recognition and prompt treatment remains the key to prevent morbidity and mortality. We discuss a case of a 36 year old female who developed negative pressure pulmonary edema following general anesthesia for septorhinoplasty, discussing its approach, treatment considerations, and outcomes.

## KEY WORDS

*Anesthesia, Negative Pressure, Obesity, Pulmonary edema, Respiratory Distress Syndrome*

## INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a possible complication during anesthesia practice with prevalence between 0.1 and 12%.[1] It is a non cardiogenic pulmonary edema supposed to occur in the background of acute or chronic upper airway obstruction with massive inspiratory effort while glottis is closed resulting in transient hypoxia and hypercarbia.

Generation of NPPE is associated with interplay of starling pressure forces along with rapidity and magnitude of negative intrapleural pressure, concomitant hydrostatic pressure and stress imposed on the patient due to hypoxia, hypercarbia and acidosis. Recognition of the airway obstruction preferably prior to inspiratory chest wall movement is the key to prevent NPPE. Treatment is sustained positive pressure continuous ventilation. Outcome of NPPE is resolution, provided that ventilation, oxygenation, hemodynamics and fluid balance is meticulously taken care of.

We present a case of NPPE in an operative room setup after tracheal extubation in an obese patient who underwent Septorhinoplasty.

## CASE REPORT

A 36-year-old female patient, classified as American Society of Anesthesiologists (ASA) physical status II, with a body weight of 95 kg, height of 153 cm, and a body mass index (BMI) of 40.5 kg/m², and a known history of obstructive sleep apnea, underwent septorhinoplasty. She was recently diagnosed with hypertension and Type II Diabetes Mellitus for which medications were continued. Difficult airway was anticipated owing to her short neck. There were no other significant findings in her preoperative assessment. Routine laboratory tests and preoperative imaging (Fig. 1) were within normal limits. She was premedicated with tablet Pantoprazole 40 mg and tablet Granisetron 1 mg a night prior to surgery. ASA standard monitoring was done for general anesthesia. During the induction of anesthesia, 2.5 mg/kg Propofol, 1 mcg/kg Fentanyl and 2 mg/kg Succinylcholine were given and she was intubated with a 7 mm I.D. size endotracheal tube. Anesthesia was maintained with Isoflurane and Oxygen whereas muscle relaxation was maintained with Vecuronium. 1000 ml crystalloid (isotonic) fluid was given during the two hours operation. The patient was hemodynamically stable during the surgery.

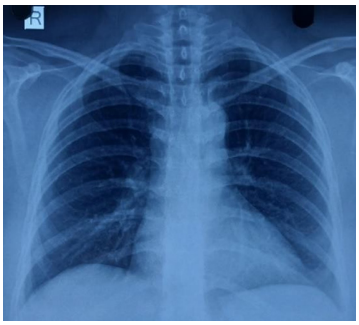

**Figure 1. Preoperative Chest X-ray PA view.**

Following completion of the surgical procedure, the patient received 4 mg/kg of Sugammadex for reversal of muscle relaxation. Subsequently, after ensuring adequate spontaneous ventilation and suctioning of oral cavity, the patient was extubated smoothly.

However, after extubation, she developed laryngo-bronchospasm and respiratory distress with rapid desaturation up to 40%. Suctioning of oral cavity was done and continuous positive airway pressure (CPAP) with 100% oxygen was applied. However, there was no significant improvement in oxygen saturation and the patient was reintubated. There were profuse foamy blood tinged pulmonary secretions seen in endotracheal tube. Auscultation revealed diffuse crepitations in bilateral lung fields. She became hypotensive and infusion of Injection Noradrenaline was started in titrated dose to maintain mean arterial pressure (MAP) above 65 mm of Hg. A total of 10 mg of Morphine and 100 mg of Furosemide were also administered intravenously in titrated dose.

Saturation remained 85% despite positive end expiratory pressure (PEEP) and intermittent positive pressure ventilation (IPPV). Chest X-ray done immediately showed diffuse bilateral pulmonary infiltrates (Fig. 2) whereas arterial blood gas (ABG) analysis showed a mixed acidosis pattern (pH:7.1, pCO2: 68, pO2: 103 HC03: 21.5, lactate 3.5). NTproBNP was found to be normal. Pulmonary edema was persistent with profuse pulmonary secretions seen in the endotracheal tube. The patient was transferred into intensive care unit (ICU) and mechanical ventilation was continued overnight. Fluid restriction to around 1.5 litres in 24 hours along with diuresis with Furosemide was continued. Gradually, PEEP was decreased and noradrenaline was tapered down. ABG analysis showed gradual correction of acidosis.

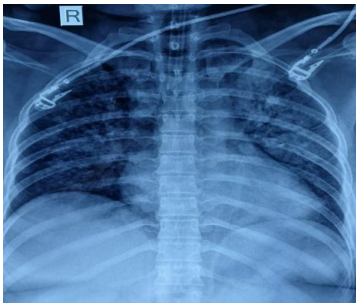

**Figure 2. Postoperative Chest X-ray taken at 30 minutes.**

On the following day, she was extubated and intermittent external CPAP was continued. There was a gradual reduction of crepitations along with improvement of Chest X-ray (Fig. 3). On her third postoperative day, her chest became completely clear. However, owing to low saturations in room air, she was supplemented with oxygen via facemask and subsequently weaned off. Incentive spirometry and deep breathing exercises were continued, and she was stepped down to ward.

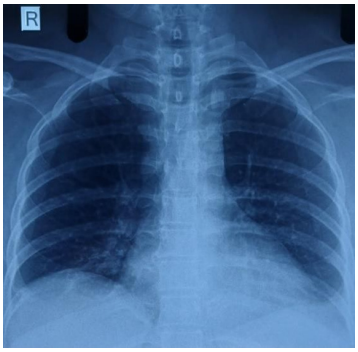

**Figure 3. Chest X-ray taken at third postoperative day.**

## DISCUSSION

The relationship between upper airway obstruction and pulmonary edema was first described in 1927 AD by Moore and Binger.[2] However, the first clinical case was not reported until 1973 AD.[3] Since then, NPPE has been reported multiple times with varying incidence statistics. The actual incidence rate may be much higher than that reported considering the lack of mandatory registering and frequent occurrence of upper airway obstruction during the peri-anesthesia period. Moreover, given the frequent occurrence of post-general anesthesia or post extubation laryngospasm, several cases are overseen, misdiagnosed, or under reported and the overall incidence appears to be more than that reported in the literature. Also, there is variability in the mortality rates. Initially, the mortality rate for NPPE was reported to range from 11 to 40%.[4] Subsequent literature reviews have depicted a mortality rate of 2%.[5]

Airway obstruction from various factors notably laryngospasm occurring during recovery from general anesthesia is directly responsible for NPPE. In adults, 55% of NPPE cases are caused by perioperative laryngospasm, and studies indicate that the incidence of laryngospasm during extubation is 0.87%.[6] Other factors predisposing to NPPE includes oropharyngeal surgery, obstructive sleep apnoea, obesity and laryngeal mask displacement. A study conducted in 86,561 patients revealed that active smokers and those undergoing endotracheal intubation for general anesthesia had a significantly higher risk of developing NPPE post extubation in the operating room.[7]

NPPE encompasses two distinct subtypes. Type I NPPE results immediately following a forceful inspiratory effort that arises after an episode of upper airway obstruction, as seen in conditions like laryngospasm, foreign body

aspiration, and epiglottitis. Type I NPPE is common in rhinology interventions. Type II NPPE is observed subsequent to chronic upper airway obstruction, such as adenotonsillar hypertrophy, obesity and obstructive sleep apnea syndrome.

The generation of massive negative intrathoracic pressure by the respiratory muscles and diaphragm against the obstructed or closed glottis (Müller's maneuver) causes deep fall of the pleural pressure ranging between -4 cm $H_2O$ down to -140 cm$H_2O$.[6] This negative pressure leads to an increase in pulmonary vascular volume and pulmonary capillary transmural pressure causing disruption of the alveolar capillary membrane and leakage of fluid into the interstitial space and alveoli, and leading to fluid accumulation in the lungs and edema formation.[8]

Although negative intrathoracic pressure is the primary factor in NPPE pathogenesis, other factors are also significant. Ventilatory efforts against an obstructed airway ultimately lead to hypoxia and acidosis, which elevate pulmonary vascular resistance and adversely affect alveolar-capillary integrity. Moreover, significant inspiratory efforts trigger a high adrenergic response, further increasing pulmonary vascular resistance and directly promoting blood redistribution from the systemic to the pulmonary circulation.

In cases of chronic airway obstruction like in obstructive sleep apnea, tonsil or gland hypertrophy, upper airway tumor, mediastinal tumor, nasopharyngeal mass and goiter, there is generation of chronic PEEP with an increased end-expiratory lung volume. After surgery or therapeutic interventions, the relief of the chronic obstruction causes loss of chronic PEEP. The increased lung volumes and pressures become normal again, creating a negative intrapulmonary pressure. If this negative pressure rises to a significant level, it can lead to fluid accumulation in the interstitium and alveoli, thus causing pulmonary edema.

The diagnosis of NPPE is established by a clinical event, and other common etiologies of pulmonary edema can be excluded by radiological evidence. NPPE usually occurs within a few minutes of extubation, although it has also been reported to occur after few hours of extubation.[9] Patients may present with predisposing factors for developing upper airway obstruction as described earlier. Patient can develop symptoms of upper airway obstruction such as stridor, respiratory distress, paradoxical chest movements and involvement of accessory muscles in breathing. Clinical manifestations of acute pulmonary edema include dyspnea, tachypnea, cyanosis, and the production of a profuse pink foamy sputum.[6] Consequently, medical complications, including hypoxemia, hypercapnia and metabolic acidosis may occur as a consequence of the underlying respiratory dysfunction and disturbed gas exchange in acute pulmonary edema.[6] Point-of-care ultrasound is very useful and accurate, and can be done at the bedside; hence, it is increasingly being used to diagnose pulmonary edema, including NPPE. More severe cases may need a chest CT scan, which may show a striking central and non-dependent distribution of ground-glass opacities (edema/hemorrhage), and can help differentiate NPPE from other forms of pulmonary edema.[10] Initially, there can be hemodynamic collapse requiring vasopressor or inotropic support. The patient is usually tachycardic. ECG typically shows signs of myocardial rhythm disturbance or ischemic changes. The echocardiogram is usually normal. Typically, after intubation and release of the upper airway obstruction, the patient shows significant improvement. Given its similarity to aspiration pneumonia during anesthesia and other causes of pulmonary edema, clinicians must be vigilant in differential diagnosis. It is crucial to exclude other causes such as cardiogenic pulmonary edema, minor reflux aspiration, acute respiratory distress syndrome, allergic pulmonary edema, and neurogenic pulmonary edema. Nevertheless, sequence of events, presence of predisposing factors and clinical findings always help in anticipation and diagnosis of NPPE.

The management of NPPE is primarily aimed at symptomatic relief, focusing on enhancing oxygenation and reducing pulmonary edema. The severity, progression, and outcome of NPPE are determined by the duration of obstruction. More than 50% of NPPE patients improve with NIV, whereas fewer than 50% require endotracheal intubation.[11] In mild cases, oxygen therapy alone may be sufficient. With proper diagnosis and treatment, patients can be expected to recover from NPPE within 24-48 hours. Non-invasive ventilation (NIV) is commonly used to prevent and treat NPPE or acute respiratory failure while avoiding endotracheal intubation. NIV in NPPE reduces the work of breathing and improves gas exchange and alveolar recruitment, thus improving hemodynamics by reducing cardiac overload and increasing cardiac output. Patients with severe pulmonary edema often require reintubation in the operating room or post-anesthesia care unit. In such patients, PEEP could improve oxygenation and reduce the required oxygen concentration. Most patients treated with mechanical ventilation were extubated within 24 hours after resolving of pulmonary edema.[6]

The use of diuretics such as Furosemide decreases hydrostatic pressure in the pulmonary vessels by reducing fluid retention and, hence alleviating pulmonary edema. However, there role is limited in patients with kidney dysfunction or in cases of intravascular fluid depletion.

Morphine was also used for the treatment primarily aiming to reduce preload by systemic vasodilatation, reduce pulmonary capillary pressure, redistribute pulmonary fluid and hence improve the forward flow.[12] Furthermore, Morphine also has the beneficial anxiolytic effect.

The use of bronchodilators with β-agonists appears to be beneficial, as these agents help in clearing the fluid from alveoli, thus aiding in reducing symptoms of pulmonary edema. Although there is no bronchospasm in NPPE,

increased fluid in the alveoli compresses the smaller alveoli. There is not much information available about the duration and dosage of β-agonists to be administered, which depends upon the patient's severity and recovery.[11]

Adrenocorticoid therapy was used in the past, but this approach is not currently supported or recommended, as it does not directly address the underlying mechanism of NPPE.[13] However, upper airway inflammation/swelling may necessitate its use.

There have also been reports of incidences of NPPE following reversal of muscle relaxation by Sugammadex. Heterogenous reversal of the neuromuscular agent with respiratory muscles such as the diaphragm recovering earlier than upper airway smooth muscle creates favorable background for Muller's maneuver resulting NPPE.[14,15] The same phenomenon cannot be ruled out in our case as well.

Some cases may have a more complicated course with difficult weaning and even recurrent pulmonary edema. Such cases not responding to treatment may even require urgent extracorporeal membrane oxygenation.[16] The use of Dexmedetomidine for weaning from mechanical ventilation has been reported to be successful in patients with NPPE.[17] Rarely, patients with NPPE may develop long term complications, including myocardial infarction, transient ischemic attack, non-ST-elevation myocardial infarction, hypoxic brain injury, pulmonary hemorrhage, septic shock, and cardiac arrest.

Measures to prevent laryngeal stimulation or postoperative stridor from partial laryngospasm can aid in reduction of incidence of NPPE. The placement of a throat pack prior to injections as well as careful oropharyngeal suctioning in head down position prior to pack removal helps to prevent blood and other material from contacting and irritating the larynx. Smooth extubation, limiting aggressive suctioning, and the use of intravenous Lidocaine can all play a role. In high risk patients with repeated NPPE, the use of prophylactic continuous positive airway pressure may also be considered.

NPPE is a known possible clinical complication resulting from excessive negative pressure against a partially or fully obstructed glottis. Early recognition and prompt treatment allowing rapid resolution of NPPE is necessary to prevent patient morbidity and mortality. In addition, careful management of susceptible patient with risk factors of upper airway obstruction is necessary to prevent this situation.

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