Journal of Comprehensive Surgery

Case Report

DOI: 10.51271/JOCS-0005

# A rare case: negative-pressure pulmonary edema after rhinoplasty

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**Cite this article:** Şencan Z, Peker K. A rare case: negative-pressure pulmonary edema after rhinoplasty. *J Compr Surg* 2023; 1(1): 18-20.

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**Submit Date:** 19/02/2023 **Accept Date:** 24/02/2023

## ABSTRACT

Negative-pressure pulmonary edema (NPPE) is a very rare and dangeorus condition that generally occurs after general anesthesia in otorhinolaryngology practices. Post-obstructive pulmonary edema or NPPE caused by an acute pulmonary edema secondary to upper respiratory tract obstruction. Its sudden appearance in the early postoperative period can stress the surgeon and the anesthesiologist. Difficult breathing effort due to upper airway obstruction creates strong changes in intra-thoracic pressure, and high negative intrathoracic pressure increases the permeability of the alveolar capillary membrane, causing significant fluid changes in the pulmonary tissues. Treatment should be evaluated together with anesthesiologist or pulmonologist and the first goal should be to reduce the hypoxia.

**Keywords**: Rhinoplasty, airway, pulmonary edema, respiratory distress

## INTRODUCTION

Negative-pressure pulmonary edema (NPPE) represents a rare and perilous complication that typically manifests subsequent to general anesthesia in otorhinolaryngologic procedures (1). NPPE, otherwise known as post-obstructive pulmonary edema, is a clinical entity characterized by the acute onset of pulmonary edema that arises as a consequence of an upper airway obstruction.Its sudden appearance in the early postoperative period can stress the surgeon and the anesthesiologist. NPPE encompasses two distinct subtypes. Type I NPPE presents acutely following a forceful inspiratory effort that arises subsequent to upper airway obstruction, as seen in conditions like laryngospasm, foreign body aspiration, and epiglottitis (2). Type 1 NPPE is common in rhinology interventions. Type 2 appears to be secondary to chronic upper airway obstruction, such as adenotonsillar hypertrophy, obesity, obstructive sleep apnea syndrome (2).

Respiratory distress caused by upper airway obstruction elicits marked alterations in intrathoracic pressure, with concomitant increases in negative intrathoracic pressure that promote increased alveolar capillary membrane permeability, ultimately leading to significant fluid shifts within the pulmonary parenchyma

## CASE

A 23-year-old ASA I, 80 kg, 180 cm male patient underwent septorhinoplasty due to septum deviation by the otolaryngology clinic. (PA) X-ray (Fig. 1), electrocardiogram (ECG), laboratory tests were considered normal.Standard monitoring was applied (heart rate, non-invasive blood pressure measurement, peripheral oxygen saturation measurement).

In the induction of anesthesia, 2 mg / kg propofol, 1.5 microgram / kg fentanyl and 0.6 mg / kg rocuronium bromide were given and after waiting for the muscle relaxation period, he was intubated with an 8.0 mm size endotracheal tube without any problem.

Anesthesia was maintained with a flow rate of 4 l / min in a mixture of 2% volume sevoflurane, 50% $N_2O$ and 50% $O_2$. 1000 ml crystalloid (isotonic) fluid was given during the 2-hours operation. There were no respiratory or hemodynamic problems during the surgery.

Following completion of the surgical procedure, the patient received a reversal agent comprising 3 mg neostigmine and 0.5 mg atropine. Subsequently, after ensuring adequate spontaneous ventilation, the patient was extubated without any immediate complications. [110/60 blood pressure (BP), respiratory rate: 15 saturation of peripheral oxygen ($SPO_2$): 98%]. He was taken to the postoperative care room for follow-up. After half an hour, respiratory distress, severe cough and agitation started. The patient's $SPO_2$ value started to decrease. An anesthesiologist was urgently consulted. CPAP with 100% $O_2$ was applied to the patient. However, when the $SPO_2$ decreased to 70%, positive ventilation was applied with 100% $FIO_2$. Intravenous 1 mg / kg methyl prednisolone was administered. There were crackles in all lung fields by auscultation. The patient started ,confusing, coughing and producing pink frothy pulmonary secretions.

With a face mask and 6 L / min $O_2$, the patient's $SPO_2$ value reached 90%, he was taken to intensive care and chest X-ray was performed (**Figure 2**). On the chest X-ray, signs of pulmonary edema were observed. PH: 7.4, $PaO_2$: 49.5, $PaCO_2$: 40.5, $HCO_3$: 25.1, $SPO_2$: 70% in the arterial blood gas taken in intensive care room. Despite $O_2$ support, noninvasive positive pressure ventilation (NPPV) was initiated for the patient since the $SPO_2$ was 70% [applied as: 6x1, Positive End-Expiratory Pressure (PEEP): 8, Fractional İnspiratory Oxygen Concentration ($FiO_2$): %70 ]. In conjunction with inhalational administration of bronchodilators and steroids, the patient was initiated on diuretic therapy, specifically furosemide at a dose of 20 mg. The patient's respiratory distress started to regress in the 8th hour postoperatively and the urine volume was 2000 ml. The patient's respiratory distress decreased and the arterial blood gas result came as; pH: 7.39, $PaO_2$: 59.5, $PaCO_2$: 41.4, $HCO_3$: 24.7, $sPO_2$: 85%. Non-invasive positive pressure ventilation was reduced and stopped at the postoperative 20th hour according to blood gas monitoring. The arterial blood gas from the patient was at pH: 7.44, $PaO_2$: 79.8, $PaCO_2$: 47.10, $HCO_3$: 29.2, $sPO_2$: 94%. The patient's general condition and respiratory distress improved. He was transferred to the service on the second day. After 24 hours in the ward, findings were $SPO_2$: 98% ; BP: 120/75 and respiratory rate:13/minute. He was discharged with full recovery on post-operative third day.

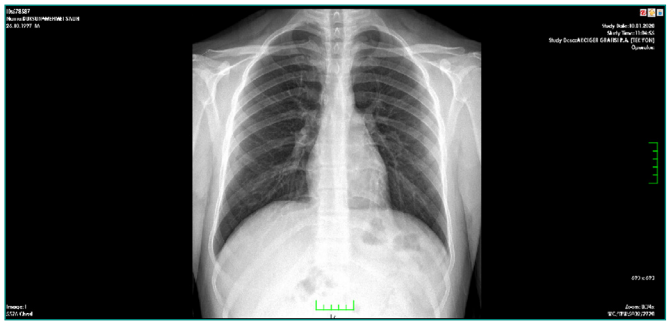

**Figure 1.** Preoperative PA chest X-ray.

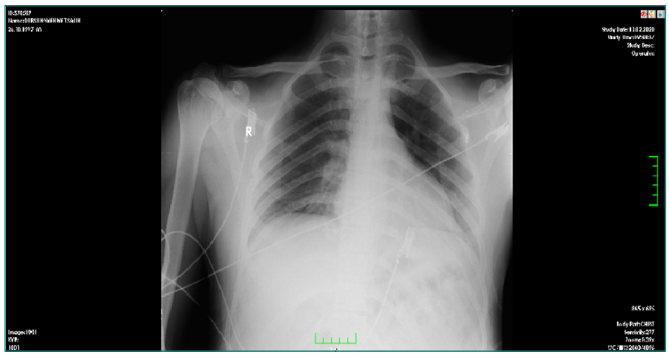

**Figure 2.** Postoperative half an hourPA chest X-ray.

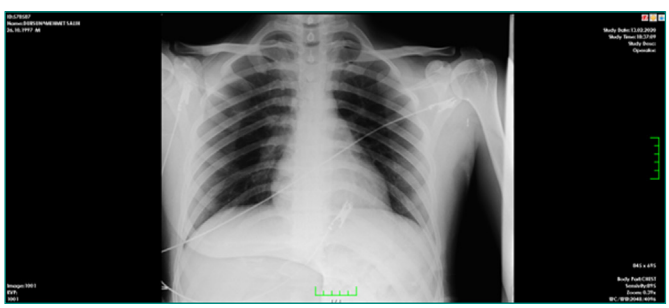

**Figure 3.** Postoperative fourth day PA chest X-ray.

## DISCUSSION

Type I NPPE which develops immediately after the onset acute upper airway obstruction, such as laryngospasm, foreign body aspiration and epiglottitis. Type I is the common reason in otorhinolaryngology practices. In accordance with the literature, we focused on a late-developing type of laryngospasm suggestive of type 1 NPPE. When the literature was reviewed, it was also discussed that there may be a type of drug-induced NPPE, but we considered laryngospasm more prominently (4,5). The complaints of the patient started in the postoperative period (after half an hour), and it was evaluated as the third case with late NPPE in rhinological surgery in the literature (6,7). NPPE must be investigated in the presence of sudden dyspnea and pink frothy sputum in a post-operative patient. The sudden onset of agitation, tachypnea and bilateral common crackles by auscultation guide the examination. For the diagnosis of NPPE, X-ray graphs and arterial blood gas examinations are usually required (6).146 adult NPPE cases compiled in a review;seventy-four cases (50.6%) involved upper aerodigestive tract or deep neck surgery, and 12 (8.2%) involved only the nose (septoplasty and/or rhinoplasty) surgery (1). The patients were intubated for an average of 11.75 hours and three mortalities (2%) occurred (1). In a case report, it was stated that a patient who underwent septoplasty had acute myocardial infarction (AMI) after the delayed diagnosis of NPPE (8). Early diagnosis is very important in NPPE. Physician awareness will help to facilitate early recognition. The most important steps in treatment are to reduce hypoxia and decrease pulmonary edema. If the patient does not have fluid overload, diuretic therapy should be initiated by performing urine output monitoring to reduce pulmonary edema. Noninvasive ventilation should be used to reduce hypoxia. If hypoxia is severe, reintubation and invasive mechanical ventilation should be performed (1). We were successful in the treatment of our patient by using this treatment modality.

## CONCLUSION

Negative-pressure pulmonary edema (NPPE) is a very rare and dangerous complication which can lead to mortality if diagnosed late. After otolaryngology surgeries, patients should be observed postoperatively and warning signs such as sudden onset of breathing difficulty and pink sputum should be kept in mind. Treatment should be evaluated together with anesthesiologist or pulmonologist and the first goal should be to reduce the hypoxia.

## ETHICAL DECLARATIONS

**Informed Consent:** All patients signed the free and informed consent form.

**Referee Evaluation Process:** Externally peer-reviewed.

**Conflict of Interest Statement:** The authors have no conflicts of interest to declare.

**Financial Disclosure:** The authors declared that this study has received no financial support.

**Author Contributions:** All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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