



## Forgotten Intraoral Mikulic Pads: An Uncommon Reason for Negative Pressure Pulmonary Edema

### Negatif Basıncılı Akciğer Ödeminin Nadir Bir Nedeni: Unutulan Ağız İçi Mikulic Tamponlar

Negatif Basıncılı Pulmoner Ödem / Negative Pressure Pulmonary Edema

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#### Özet

Negatif basıncılı akciğer ödemi (NBAÖ), ekstübasyon sonrası üst hava yolu obstrüksiyonuna bağlı olarak gelişen nadir fakat ölümcül olabilen bir komplikasyondur. NBPÖ'nün en sık nedeni laringospazmdir. Krup, epiglottit, üst solunum yolu tümörleri, yabancı cisim aspirasyonları, koyu trakeal sekresyonlar, endotrakeal tüpü ısırma ve hematoma gibi çeşitli nedenlerle olabilmektedir. Bu olgu sunumunda genel anestezi sonrası üst hava yolu obstrüksiyonunun nadir bir nedeni olan ve operasyon esnasında ağız içine konan mikulic tamponuna bağlı olarak gelişen NBAÖ'ni sunmayı amaçladık.

#### Anahtar Kelimeler

Pulmoner Ödem; Havayolu Tıkanıklığı; Mikulic Tampon

#### Abstract

Negative pressure pulmonary edema (NPPE) is a rare complication, which can be fatal depending on post-extubation upper airway obstruction. The most frequent cause of NPPE is laryngospasm. Other causes include croup, epiglottitis, upper respiratory tract tumors, foreign substance aspiration, dark tracheal secretions, biting the endotracheal tube, or hematoma. In this case report we aimed to present an NPPE case that occurred as a result of a Mikulic pad being placed into the oral cavity during an operation and left there after the surgery was completed, which is an uncommon reason for upper airway obstruction following general anesthesia.

#### Keywords

Pulmonary Edema; Airway Obstruction; Mikulic Pad

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## Introduction

Negative pressure pulmonary edema (NPPE) is a rare complication, which can be fatal depending on post-extubation upper airway obstruction; it appears as a result of strong inspirium effort after an obstruction in the airways or resolution of the airway obstruction. With the increased negative pressure that occurs as a result of a strong inspirium effort that is performed in order to resolve the airway obstruction, the fluid externalized through the pulmonary capillaries fills the alveolar and interstitial spaces. This causes non-cardiac pulmonary edema [1]. The incidence of NPPE among patients that underwent surgery under general anesthesia (GA) varies between 0.05% and 0.1% [2]. The aim of this case report is to present an NPPE case that occurred as a result of a Mikulic pad being placed into the oral cavity during an operation and left there after the surgery was completed, which is an uncommon reason for upper airway obstruction following GA.

## Case Report

Surgery under GA was planned for a 15-year-old male patient with isolated maxillofacial trauma. The patient's height was 168 cm and his weight was 55 kg. He had no history of allergies or medication use. The preoperative evaluation of the patient revealed normal physical examination and laboratory findings. Following pre-operational monitoring (non-invasive blood pressure, ECG, pulse oximetry), anesthetic induction was provided via intravenous Pentothal 7 mg/kg, fentanyl 1µg/kg, and rocuronium 0.6 mg/kg injection. Endotracheal intubation was performed without complication via nasotracheal route using a spiral tube with a 7 mm inner diameter following muscle relaxation. Anesthetic continuation was performed using oxygen 3 L/min, nitrogen protoxide 3 L/min, and sevoflurane 2%. A Mikulic pad was placed in the oral cavity by the surgical team at the beginning of the operation in order to prevent the passage of the intraoral bleeding or secretions into the stomach. At the end of the third hour, the operation was completed without any complications. The patient, who has returned to normal airway reflexes and spontaneous respiration, was given IV 1.5 mg neostigmine and 0.5 mg atropine as bolus and extubation was performed. In the post-extubation period, ventilation via mask was started because the patient appeared agitated and his peripheral oxygen saturation declined. Since the peripheral oxygen saturation was not increased and the respiratory effort was not sufficient, re-intubation was planned with intravenous Pentothal 5 mg/kg, and succinylcholine 1 mg/kg injection. During intubation, direct laryngoscopy was used to monitor the oral cavity and it was observed that a Mikulic pad was blocking the airway. The pad was taken out via forceps and intubation was performed. Following the intubation, the pink, foamy fluid within the tube was aspirated. Both pulmonary sounds were rough and diffuse crepitant rales were heard. The peripheral oxygen saturation was 73% despite ventilation with 100% oxygen. The arterial blood gas measurement was: pH:7,18, pO<sub>2</sub>:43mmHg, pCO<sub>2</sub>:48mmHg, HCO<sub>3</sub><sup>-</sup>:19.4. The patient was taken to the intensive care unit after being diagnosed with clinical pulmonary edema. Pulmonary protective ventilation (low tidal volume (4–5 ml/kg) and a high PEEP (10 mmHg) strategy were performed on the patient under sedation. During the ventilation period, the

patient's peripheral oxygen saturation increased up to 85% with 100% oxygen support and high PEEP values were observed. As medication, Furosemide 3x20 mg and Dexamethasone 4x4 mg were administered on the first day. In the post-operative 16th hour, sedation was stopped and mechanical ventilation support was continued via CPAP mode. On the second post-operative day, the general situation of the patient was good and the peripheral oxygenation and blood gas parameters were improved. The patient was extubed and medical treatment was continued. On the third post-operative day, the patient was taken to the service and he was discharged on the seventh day.

## Discussion

Negative pressure pulmonary edema (NPPE) is a rare complication that is seen after extubation of patients undergoing general anesthesia. NPPE is frequently seen among healthy young people whose muscle volume is high and, therefore, capable of high inspiratory negative pressure formation [3]. In our case, the patient was young and had an athletic body structure that might cause high inspiratory pressure, and the case was in accordance with the cases reported in the literature. Intrathoracic pressure is increased due to hard inspiration effort, which is related to acute upper respiratory tract obstruction in patients undergoing general anesthesia. Increased negative intratho-

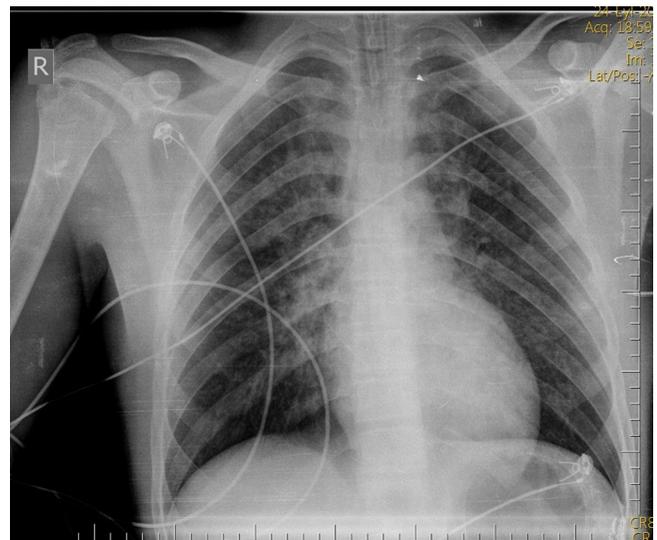


Figure 1. Preoperative X-ray image of the patient.

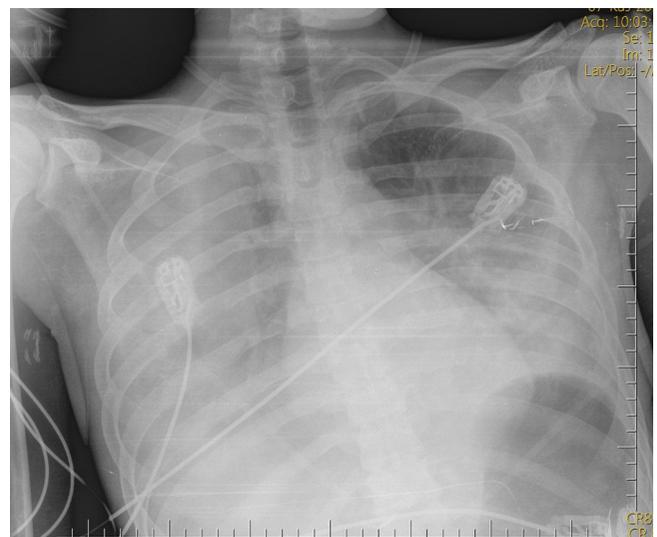


Figure 2. The presence of NPPE in X-ray image after the operation.

racic pressure increases the venous return of blood to the heart and decreases the drainage of the blood to the left atrium; therefore, the hydrostatic pressure is increased within the pulmonary bed and transudation occurs in the alveoli [4]. Consequently, pulmonary edema can occur immediately, although it has also been reported that it can occur in patients at a later period [5]. In our case, pulmonary edema was observed within minutes following extubation and it showed rapid progression. This exacerbated the clinical table and caused the development of deep hypoxia.

The most frequent cause of NPPE is laryngospasm. Other causes include croup, epiglottitis, upper respiratory tract tumors, foreign substance aspiration, dark tracheal secretions, biting the endotracheal tube, or hematoma [6]. However, no case of NPPE developing as a result of a Mikulic pad having been left within the oral cavity has been reported in the literature. Thus, forgetting intraoral pads placed into the mouth during surgical operations may cause a serious complication such as NPPE during post-operative awakening.

The diagnosis of NPPE is confirmed with clinical findings, blood gas analyses, and radiological imaging. In our case, both blood gas values and posteroanterior lung graphy have confirmed the diagnosis. Clinically, the sudden reduction of peripheral oxygen saturation caused hypoxia; secondary agitation findings and the presence of a pink-foamy secretion within the tube following re-intubation were suggestive of pulmonary edema. The lung graphy revealed diffuse bilateral alveolo-interstitial edema and fullness in the hilar region of the lung, and the blood gas values revealed severe hypoxia.

The target of NPPE treatment is primarily eliminating the airway obstruction and treating hypoxia. Non-invasive ventilation with positive pressure is generally sufficient for patients with mild clinical manifestation, but re-intubation or mechanical ventilation may also be necessary, which was the situation for our patient. Non-invasive ventilation has been known to reduce the risk of re-intubation, shorten the duration of intensive care or hospital stay, and lower morbidity and mortality [7]. However, serious hypoxia and severe clinical findings have made ventilatory support an obligation, as was the case with our patient. We performed high PEEP (10mmHg) in order to alleviate the hypoxia under invasive ventilatory support. Although there are different opinions for the pharmacological treatment of NPPE, at present, diuretic drug usage is controversial because there is no fluid load in the physiopathology of edema. Diuretic drug usage is not recommended except for patients with possible hypervolemia due to intraoperative hydration. Furthermore, digoxine, corticosteroids, positive inotropic drugs, or morphine may be used depending upon the clinical and hemodynamic situation of the patient. Although fluid limitation is recommended in the literature, we believed that diuretic drug usage would be proper since it could be possible to remove the intra-alveolar fluid faster with diuretic drug usage for our patient with intraoperative hydration.

In conclusion, NPPE should be considered in hypoxia that develops immediately after extubation in the early postoperative period, and clinicians should bear in mind that NPPE may develop due to intraoral pads having been left in the oral cavity after the operation.

### Competing interests

The authors declare that they have no competing interests.

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