



# Negative Pressure Pulmonary Edema During Laryngeal Mask Use: A Case Report

Aysun Postacı, Özlem Saçan, Ayfer N Yılmaz, Dilşen Örneç, Gülçin H Alay, Nermin Göğüş

Ankara Numune Training and Research Hospital, Department of Anesthesiology and Reanimation, Ankara

## ÖZET

*Laringeal maske kullanımı sırasında negatif basınçlı pulmoner ödem: Olgu sunumu*

Negatif basınçlı pulmoner ödem (NBPÖ) havayolu tıkanıklığına bağlı olarak gelişen nonkardiyojenik bir pulmoner ödemdir. Genç, sağlıklı, yüksek negatif intraplevral basınç oluşturabilecek kas kütlesi fazla erkek hastalar (Atlet pulmoner ödem sendromu, APES) risk altındadır. Genellikle endotrakeal entübasyon uygulanan hastalarda ekstübasyon sonrası gelişen NBPÖ az da olsa laringeal maske (LMA) uygulaması sonrasında gelişebileceğini bildiren olgu sunumları bulunmaktadır. Laringeal maske kullanımı sırasında havayolu obstrüksiyonun en sık olası nedenleri; LMA tüpünün şiddetli ısırılması, yanlış yerleşimi ve laringospazmdir. Bu olgu sunumunda laringeal maske uygulanan olguda anesteziden uyanma sırasında meydana gelen laringospazma bağlı NBPÖ olgusu sunulmaktadır.

**Anahtar kelimeler:** Pulmoner ödem, havayolu obstrüksiyonu, laringospazm, laringeal maske

## ABSTRACT

*Negative pressure pulmonary edema during laryngeal mask use: a case report*

Negative pressure pulmonary edema (NPPE), a non-cardiogenic pulmonary edema, develops due to airway obstruction. Young, healthy, male patients, who have a lot of muscle mass that can lead to produce high negative intra-pleural pressure (athletes pulmonary edema syndrome, APS), are under the risk of developing NPPE. Usually, NPPE develops after extubation in patients who undergo endotracheal intubation however; there are some case reports showing that it can develop after laryngeal mask application (LMA) even in a less manner. The most possible reasons lead to airway obstruction during LMA use are biting of laryngeal mask tube, misplacement of tube, and laryngospasm. This case report presents a patient who undergoes LMA and develops NPPE due to laryngospasm during recovery from anesthesia.

**Key words:** General anesthesia, pulmonary edema, laryngeal mask airway

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## INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a rare but life-threatening complication of general anesthesia. The incidence of NPPE in healthy adults who underwent general anesthesia was reported as 0.05-0.1% (1,2). NPPE, a non-cardiogenic pulmonary edema, develops after a difficult inspiration against obstructed upper airways. It was reported that young, athletic male patients who have strong inspiratory muscles that can produce high

intrapleural negative pressure (athlete pulmonary edema syndrome, APES) are under high risk for developing NPPE (3,4). Although, NPPE comprises usually after extubation in patients whom applied endotracheal intubation, there are some case reports (even though in a less manner) presenting that NPPE can develop following laryngeal mask airway (LMA) usage (3,5). In this case report, we discuss a patient who develops respiratory failure due to pulmonary edema with hemoptysis as a result of laryngospasm and strong inspiratory effort during recovery after LMA application under general anesthesia for outpatient hand surgery.

## CASE REPORT

An outpatient surgery for right hand tendon repair under general anesthesia was planned for a male, 29

Yazışma adresi / Address reprint requests to: Dr. Aysun Postacı  
Ankara Numune Training and Research Hospital, Department of  
Anesthesiology and Reanimation, Ankara

Telefon / Phone: +90-312-508-4229

Elektronik posta adresi / E-mail address: aysunposta@yahoo.com

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year-old, 85 kg, ASA I group patient. His physical examination and routine laboratory findings were normal. The patient was accepted to operation room after he was informed and asked to sign concept form for anesthesia and surgery. He was monitored for electrocardiogram (ECG), non-invasive blood pressure (NIBP), as well as peripheral oxygen saturation (SpO<sub>2</sub>) and a peripheral vein was catheterized for infusion and drug administration. Midazolam (2 mg, intravenous, iv) was administered and the patient was pre-oxygenated with 80% O<sub>2</sub> and 20% air combination via face mask before anesthesia induction. We injected fentanyl (150 microgram, iv) and propofol (200 mg, iv) for anesthesia induction and 1 minute (min) later, LMA (no:5) was placed without any problem. Cuff was inflated with 30-40 ml of air. Anesthesia was maintained using sevofluran (1.5-2%) and combination of 50% O<sub>2</sub> and 50% nitrous oxide (2L/min). SIMV (Synchronized Intermittent Mandatory Ventilation) mode was used for mechanical ventilation. Inflation pressures and capnogram were normal during mechanical ventilation through procedure. Surgery lasted an hour and during that time vital signs were normal. Total of 750 ml crystalloid solution was infused. At the end of the surgery inhalation agents were ceased and LMA was removed with no problem. Oxygen (100%) was given via a face mask. However, we observed that patient had difficult inspiration, paradoxical chest movements, wheezing, and cyanosis. His SpO<sub>2</sub> was 85%. It was thought that laryngospasm was developed. We injected propofol (50 mg, iv) and the patient was ventilated with 100% O<sub>2</sub> using a face mask. Four to five min later, respiratory distress and oxygen saturation were recovered and the patient was brought to the recovery room. Nevertheless, his SpO<sub>2</sub> level decreased again and hemoptysis was observed. The patient was moved to post anesthetic care unit. There were only bilateral rales during auscultation of lungs in his physical examination. Other than that, his physical examination was normal. Chest X-ray showed bilateral, common interstitial infiltration. His other laboratory findings were normal. It was thought that NPPE was developed regarding to his physical examination and chest X-ray findings. Despite oxygen administration via face mask (6-8 L/min), SpO<sub>2</sub> was still under 90%. Non-invasive mechanical ventilation (NIMV) using oro-nasal mask (Evita 4, Drüger Medical AG & KG, Germany) as CPAPASB (Continuous Positive Airway Pressure/ Asisted

spontaneous Breathing) 15 min/h (PEEP 5/20PASB, Positive End-Expiratory Pressure/ Asisted Spontaneous Breathing Pressure) and FiO<sub>2</sub> 1.0 for 6 hrs was applied. Additionally, a bronchodilator and furosemide (20 mg, iv) were administered. Since clinical findings and blood gas analysis have improved, gradually PEEP and FiO<sub>2</sub> were decreased to 5 cmH<sub>2</sub>O and 0.4, respectively. SpO<sub>2</sub> showed a marked improvement and NIMV support was ended at the sixth hrs. Then patient received 2L/min O<sub>2</sub> via nasal cannula. The patient was comfortable breathing at room air at postoperative 24 h and then he was sent to his service room. He was discharged at 48 h without any respiratory distress and clinical pathology. He was asked to come for check up 1-2 weeks later.

## DISCUSSION

Postoperative NPPE, which develops as a response to upper airway obstruction is a rare but well known complication of anesthesia. Many factors play a role in etiopathogenesis of NPPE. The most important mechanism is that a negative intrathoracic pressure develops and then afterload as well as pulmonary capillary hydrostatic pressure increase during Mueller maneuver, which is inspiration against closed glottis (2,3). During airway obstruction and difficult inspiration pressure in trachea and lower airways is decreased markedly. Pleural surface pressure decreases in a same level but decrease in pulmonary vessel pressure is less, thus pressure difference between inside and outside of capillaries increases and as a result of this interstitial fluid comprises. When interstitial compartmental edema fluid reaches to critical point, fluid in alveolus increases. Hypoxia comprises and causes pulmonary vasoconstriction. As a result of this, pulmonary capillary hydrostatic pressure augments more (2,5). NPPE usually develops due to laryngospasm (the most frequent) and biting of endotracheal tube during recovery from general anesthesia (2). It happens less frequently after foreign body aspiration, oropharyngeal surgeries, and postoperative residual curarization (typically while inspiratory muscle function is maintained, upper airway dilator muscles are affected) (2,6). NPPE is diagnosed as clinically (dyspnea, hypoxia, pink foamy secretion from mouth after minutes following upper airway obstruction) and radiological examination (bilateral pulmonary edema) and it regresses with a proper treatment in a

few hours. However, in some cases symptoms might appear hours later (2,3). Mechanical ventilation with PEEP might be required in patients with severe NPPE or in patients with cardiopulmonary disease previously (2). Anaphylaxis; pulmonary edema results from overload of iatrogenic fluid; neurogenic, as well as cardiogenic pulmonary edemas should be thought in differential diagnosis of NPPE (5,6). LMA, is commonly used to keep airways safe under general anesthesia, is an airway tool. Despite its common use in general anesthesia practice, there are only limited amount of reports about its complications. The most frequent complication (1-3%) during LMA use is laryngospasm, which is encountered during either induction of anesthesia or recovery from anesthesia (7). Possible reasons of airway obstruction during LMA usage are biting of LMA tube, misplacement of tube, and laryngospasm (3-8). In our case, we had no difficulty during placement of LMA tube and inflation pressures, as well as capnogram was in normal ranges during the surgery. Total of 750 ml fluid was infused so we pulmonary edema was not a result of fluid overload. Likewise anaphylaxis was not the reason as the patient had no sign of rash, urticaria, or bronchospasm, he had no history of any allergic disease as well. There are reported cases of NPPE which developed due to airway obstruction as a result of LMA tube biting during recovery from anesthesia (8). We did not encounter a problem like that. In our case, we thought that NPPE developed due to laryngospasm during recovery from anesthesia.

Quick diagnosis and treatment affect prognosis of NPPE. Severity of NPPE is correlated with obstruction time and degree of pulmonary capillary damage (3). Main goal for treatment is to maintain airways open and enough oxygenation. Patients with severe NPPE might need aggressive monitoring and invasive respiratory support. There are reports showing that non-invasive mechanical ventilation (CPAP), which is an alternate to endotracheal intubation, plays an important role to prevent or to treat acute respiratory failure (9). Aims of non-invasive mechanical ventilation for treating NPPE are to reduce respiratory effort, to compensate respiratory function, to improve alveolar function by providing better gas exchange, to increase cardiac output by reducing left ventricle afterload, and to improve hemodynamic stability (10). It was shown that non-invasive mechanical ventilation was the most effective method to reduce intubation rate; to decrease time in intensive care unit, as well as hospital; to reduce postoperative morbidity and mortality (10). In our case, non-invasive mechanical ventilation was applied because of these reasons and he responded well to send him to service room at postoperative 24 hour.

Although LMA use under general anesthesia for outpatient applications is getting more common, reports regarding to its complications and retrospective studies are still less. Even though NPPE during LMA is rare, it should be kept in mind that it can happen and that early diagnosis and treatment should be done.

## REFERENCES

1. Vandse R, Kothari DS, Tripathi RS, Lopes L, Stawick SPA, Papadimos TJ. Negative pressure pulmonary edema with laryngeal mask airway use: Recognition, pathophysiology and treatment modalities. *Int J of Crit Illn and Inj Sci* 2012; 2: 98-103.
2. Krodel DJ, Bittner EA, Abdulnour R, Brown R, Eikermann M. Case scenario: Acute postoperative negative pressure pulmonary edema. *Anesthesiology* 2010; 113: 200-207.
3. Sickmann K, Seider R, Dahm M, Nold H. Negative pressure pulmonary edema. Post-obstructive lung edema after use of a laryngeal mask. *Anaesthesist* 2005; 54: 1197-1200.
4. Ezri T, Priscu V, Szmuck P, Soroker D. Laryngeal mask and pulmoner edema (letter). *Anesthesiology* 1993; 78: 219.
5. Bhavani-Shankar K, Hart NS, Mushlin PS. Negative pressure induced airway and pulmonary edema. *Can J Anesth* 1997; 44: 78-81.
6. Warner LO, Martino JD, Davidson PJ, Beach TP. Negative pressure pulmonary oedema: A potential hazard of muscle relaxants in awake infants. *Can J Anaesth* 1990; 37: 580-583.
7. Pennant JH, White PF. The laryngeal mask airway. Its uses in anesthesiology. *Anaesthesiology* 1993; 79: 144-163.
8. Devys JM, Balleau C, Jayr C, Bourgain JL. Biting the laryngeal mask: an unusual cause of negative pressure pulmonary edema. *Can J Anesth* 2000; 47: 176-178.
9. Salem MR, Candido KD, Khorasani A. Acute postoperative Negative-pressure Pulmonary Edema. *Anesthesiology* 2011; 114: 461-468.
10. Pelosi P, Jaber S. Noninvasive respiratory support in the perioperative period. *Curr Opin Anaesthesiol* 2010; 23: 233-238.