Case Report

Negative-pressure Pulmonary Edema Following Sustained Laryngospasm during Emergence from Anesthesia in a Young Adult Undergoing Facial Surgery: A Case Report

ABSTRACT

Post-extubation negative-pressure pulmonary edema (NPPE) develops after the closed intrathoracic cavity was forced to expand with intense inspiratory effort against an obstructed airway. Laryngospasm that occurs after extubation during emergence is one of the causes of upper airway obstruction. Extubation during an inadequate depth of anesthesia, excessive airway secretion and irritation due to suction are known as cause of laryngospasm. Recently, there have been reports that laryngospasm occurred after sugammadex administration during emergence from general anesthesia. Here, we report NPPE following consecutive sugammadex administration during emergence from anesthesia in a young man undergoing nasal septorhinoplasty under general anesthesia. In this case, NPPE occurred due to an excessive increase in inspiratory force in the state of airway obstruction (laryngospasm) after extubation, and a young man, nasal surgery with packing, and repeated misuse of sugammadex due to absence of neuromuscular monitoring could contribute to the occurrence of NPPE.

Keywords: Negative-pressure pulmonary edema, Laryngospasm, Anesthesia Recovery Period, Sugammadex.

1. INTRODUCTION

Negative-pressure pulmonary edema (NPPE) or postobstructive pulmonary edema develops in patients with intact spontaneous respiratory effort and upper airway obstruction. Closed intrathoracic cavity forced to expand with intense inspiratory effort against an obstructed airway can lead to severe hypoxemia and pulmonary edema [1,2]. Laryngospasm that occurs after extubation during emergence is one of the common causes of upper airway obstruction. Extubation during an inadequate depth of anesthesia, excessive airway secretion and irritation due to suction are known as causes of laryngospasm. NPPE appeared more frequent in healthy male patients whose American Society of Anesthesiologists (ASA) physical status I or II, with an overall incidence of 0.05 to 0.1% [2,3]. The most important treatment of NPPE is to maintain airway patency through positive pressure ventilation to prevent hypoxia and, if necessary, endotracheal intubation should be done in severe cases. With proper supportive care including diuresis and ventilatory support, recovery from NPPE is usually rapid without sequelae in young healthy patients.

Sugammadex, a modified gamma-cyclodextrin, is a selective relaxant-binding agent specifically developed for rapid reversal of rocuronium-induced neuromuscular blockade [4].

It enabled fast and predictable reversal of neuromuscular blockade, increased patient safety, and reduced incidence of residual blockade on recovery [4]. Nevertheless, recently, there have been reports that laryngospasm occurred after sugammadex administration [5-8]. We report a case of NPPE following sustained laryngospasm occurred after sugammadex administration during emergence in young healthy male patient undergoing nasal septorhinoplasty under general anesthesia.

2. CASE REPORT

We present a case report of a 22-year-old male with a weight 47.4kg and height 160.1cm receiving septorhinoplasty to correct nasal septum deviation in Korea University Guro Hospital. He was in good health but had a history of cleft lip and palate, and received palatoplasty and alveolorraphy in 2005 and 2020, respectively. Because there was a history of cleft palate and lip, the possibility of difficult intubation was closely evaluated in the preoperative evaluation and there were no history of difficult intubation and anatomical factors indicative of difficult airway in preoperative interview.

After admitted the operating room, electrocardiogram, noninvasive blood pressure cuff and pulse oximeter were applied to the patient. Neuromuscular monitoring was not performed. Anesthesia was induced with propofol 100 mg, rocuronium 40 mg. After endotracheal intubation, anesthesia was maintained with desflurane, nitrous oxide and infusion of remifentanil. During the surgery, 10 mg of rocuronium was given to the patient three times when the neuromuscular block was not enough. The operation was about 400 minutes. Total additional rocuronium of 30 mg was administered, and the last 10 mg of rocuronium was administered 50 minutes before the end of the surgery. After operation, desflurane, nitrous oxide and remifentanil were discontinued. Shortly thereafter, sugammadex 200 mg was given to reverse the neuromuscular blockade.

Fifteen minutes later, spontaneous breathing was confirmed in which the tidal volume was over 300 mL and respiratory rate was over 10 breaths/min. At that time, minimum alveolar concentration (MAC) was 0.3–0.4, and the patient was extubated after endotracheal tube and oropharyngeal suction. However, chocking signs were seen immediately after extubation, and oxygen saturation dropped immediately to 56% with cyanosis. An oropharyngeal airway was inserted and positive-pressure ventilation with mask was tried to maintain airway patency, but the patient with nasal packing was irritable, not cooperative, and not ventilated.

At that time, it was judged that the patient's airway obstruction was due to insufficient recovery of neuromuscular function, and additional sugammadex 200 mg was given to the patient. However, manual ventilation was aggravated with high peak airway pressure and then pink frothy sputum came out of the mouth. On auscultation, coarse rhonchi was clearly heard in both lung fields. Fortunately, oxygen saturation was recovered to 99% in two minutes of manual ventilation with mask and bag. But his arterial blood gas analysis revealed severe carbon dioxide retention (pH 7.30, pCO_2 60 mmHg, PO_2 149 mmHg, HCO_3^- 29.5 mmol/L). He was re-intubated after administration of propofol 40 mg and succinylcholine 50 mg. His chest X-ray after re-intubation obtained in operating room revealed bilateral pulmonary edema (Figure 1). He was sent to the intensive care unit (ICU), and sedated with dexmedetomidine and midazolam.

At the ICU, blood pressure was dropped to 80/50 mmHg (mean blood pressure 60 mmHg, heart rate 85 beats/min). The blood pressure was stabilized with intravenous norepinephrine infusion with fluid management. The laboratory findings were normal. At the two days of ICU

care including controlled ventilation with intermittent intravenous furosemide injection, he was extubated and the vital signs were stable without intravenous norepinephrine. Chest x-ray was improved (Figure 2) and transferred to general ward the third day after surgery.

One week after the surgery, the chest radiograph showed that the pulmonary edema had completely disappeared and showed normal findings (Figure 3). After he was treated with antibiotics for 9 days for phlebitis and discharged without sequelae.

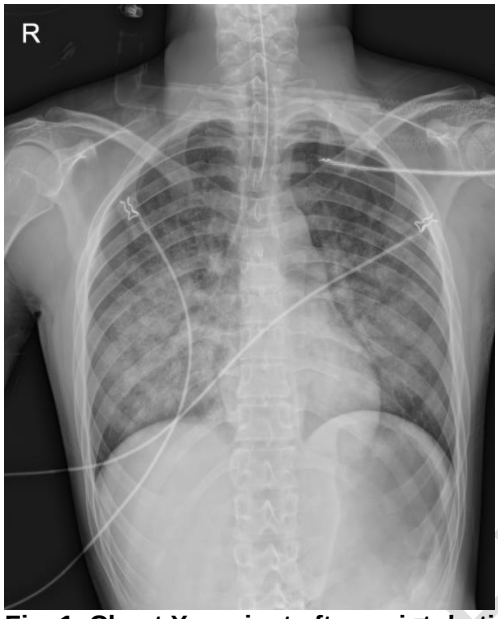

Fig. 1. Chest X-ray just after re-intubation in the operating room

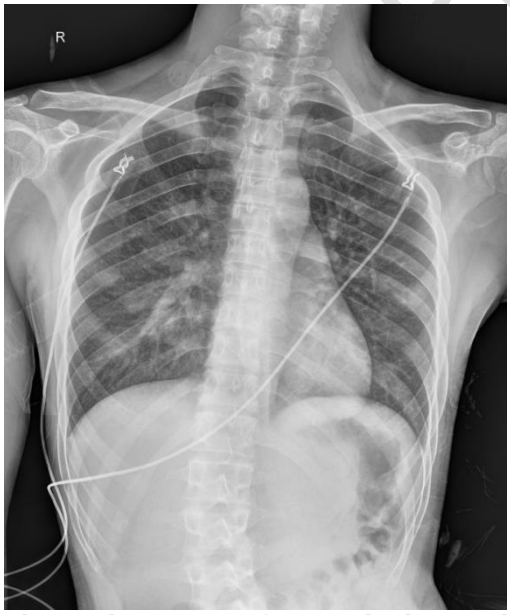

Fig. 2. Chest X-ray after extubation on the second day after surgery

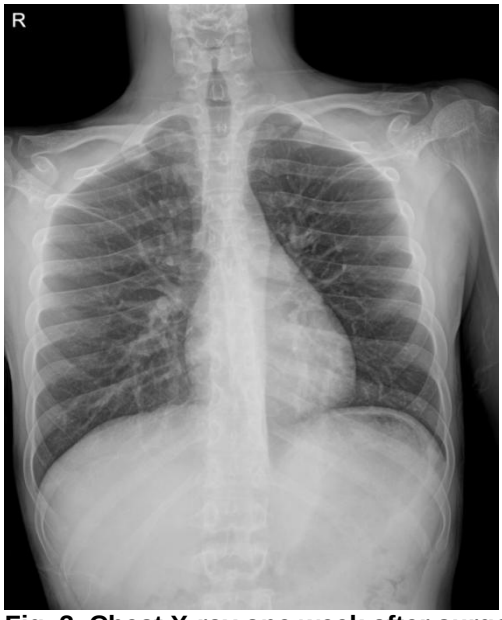

Fig. 3. Chest X-ray one week after surgery

3. DISCUSSION

Hypoxia can be caused by several factors during emergence from anesthesia. Residual muscular blockade is a common cause of hypoxia during emergence from anesthesia. Reported data suggest that residual neuromuscular block is a common complication in the postanesthesia care unit, with approximately 40% of patients exhibiting a train-of-four ratio < 0.9[9]. On the other side, laryngospasm is rare, but also causes upper airway obstruction, leading to hypoxia. The incidence of laryngospasm is 8.7/1000 patients demonstrated by Olsson and Hallen [10]. Although laryngospasm is a potentially dangerous complication, it may be hard to suspect that the cause of hypoxia is laryngospasm because the incidence is relatively lower than that of residual paralysis. In this case as well, his signs of suffocation were misinterpreted as residual neuromuscular blockade despite the fact that total additional rocuronium was not high and the last additional rocuronium was given 50 minutes before the end of the surgery.

In this case, NPPE occurred due to an excessive increase in inspiratory force in the state of laryngospasm after extubation during emergence form anesthesia. NPPE usually appears more frequent in healthy male patients whose ASA physical status I and II [2,3]. Since the pathophysiology of NPPE is aggressive expansion of closed intrathoracic cavity resulted from upper airway obstruction, NPPE more likely occurs in healthy male person with high muscular power like in our case. In this case, In this case, although it was estimated that the patient's neuromuscular block was not high at the end of the surgery, the patient's neuromuscular block was reversed by sugammadex, and additional sugammadex was repeatedly administered when chocking signs appeared. Unfortunately, as his hypoxia was not due to residual neuromuscular blockade, the upper airway obstruction was not solved and laryngospasm might be aggravated. If neuromuscular monitoring had been applied to the patient in advance, appropriate neuromuscular block reversal would have been performed with accurate information on the neuromuscular block status of the patient, and

residual paralysis would have been confirmed. As a result, the occurrence of NPPE could have been prevented.

A few precautions can prevent laryngospasm. The high incidence of laryngospasm is associated with some conditions including extubation during an inadequate depth of anesthesia, excessive airway secretion, irritation due to suction, history of bronchial asthma, or recent upper airway infection. The type of surgery also can be a cause of laryngospasm. In literature, the incidence of laryngospasm was reported up to 25% in patients undergoing tonsillectomy and adenoidectomy [11]. In this case, we speculate that factors influencing the development of laryngospasm might be extubation during inadequate recovery of consciousness and nasal surgery with gauze packing, irritation of airway by secretion and bleeding from the nasal cavity. Especially, sugammadex administration just after the end of surgery facilitated neuromuscular recovery more rapidly while recovery of consciousness is not enough. The harmonization of recovery from neuromuscular block and consciousness should not be overlooked [12,13]. In particular, Kang et al, reported that the risk of upper airway obstruction is low when sugammadex administration is performed at the MAC less than 0.3 [12]. Nasal packing with gauze also could induce agitation provoking early extubation in this patient. As like this case, special attention should be paid to patients who are expected to have many triggers for the development of laryngospasm and NPPE when awakening from anesthesia.

4. CONCLUSION

In this case, NPPE occurred due to an excessive increase in inspiratory force in the state of upper airway obstruction (laryngospasm) after extubation in young healthy male patient undergoing nasal septorhinoplasty under general anesthesia. Nasal surgery with packing and unharmonized recovery of neuromuscular function and consciousness might induce early extubation during emergence, resulting in laryngospasm. Repetitive administration of sugammadex due to absence of neuromuscular monitoring might contribute to aggravation of laryngospasm leading to NPPE in the patient. Anesthesiologists should determine the timing of sugammadex administration considering the level of consciousness, and perform neuromuscular monitoring to prevent the complications in these patients.

CONSENT

Written consent obtained from the patient has been collected and preserved by the authors.

REFERENCES

1. Bhattacharya M, Kallet RH, Ware LB, Matthay MA. Negative-Pressure Pulmonary Edema. Chest. 2016;150:927-933.
2. Deepika K, Kenaan CA, Barrocas AM, Fonseca JJ, Bikazi GB. Negative pressure pulmonary edema after acute upper airway obstruction. J Clin Anesth. 1997;9:403-408.
3. Udeshi A, Cantie SM, Pierre E. Postobstructive pulmonary edema. J Crit Care. 2010;25:508.e1-5.
4. Hristovska AM, Duch P, Allingstrup M, Afshari A. Efficacy and safety of sugammadex versus neostigmine in reversing neuromuscular blockade in adults. Cochrane Database Syst Rev. 2017;8:CD012763.
5. McGuire B, Dalton AJ. Did sugammadex cause, or reveal, laryngospasm? A reply. Anaesthesia. 2016;71:1112-1113.

6. Chrimes N. Did sugammadex cause, or reveal, laryngospasm? Anaesthesia. 2016;71:1112. 7. McGuire B, Dalton AJ. Sugammadex, airway obstruction, and drifting across the ethical divide: a personal account. Anaesthesia. 2016;71:487-492.
8. Greenaway S, Shah S, Dancey M. Sugammadex and laryngospasm. Anaesthesia. 2017;72;412-413.
9. Murphy GS, Brull SJ. Residual neuromuscular block: lessons unlearned. Part I: definitions, incidence, and adverse physiologic effects of residual neuromuscular block. Anesth Analg. 2010;111:120-128.
10. Olsson GL, Hallen B. Laryngospasm during anaesthesia. A computer-aided incidence study in 136,929 patients. Acta Anaesthesiol Scand. 1984;28:567-575.
11. Marzban S, Haddadi S, Naghipour MR, Sayah Varg Z, Naderi Nabi B. The effect of intravenous magnesium sulfate on laryngospasm after elective adenotonsillectomy surgery in children. Anesth Pain Med. 2014;4:e15960.
12. Kang E, Lee BC, Park JH, Lee SE, Kim SH, Oh D et al. The Relationship between the Timing of Sugammadex Administration and the Upper Airway Obstruction during Awakening from Anesthesia: A Retrospective Study. Medicina (Kaunas). 2021;57:88.
13. Brull SJ, Prielipp RC. Sugammadex and negative pressure pulmonary edema: what you see is not what you get. J Clin Anesth. 2020;67;109971.