Case Report

Negative pressure pulmonary edema after general anesthesia using the i-gel

ABSTRACT

Negative pressure pulmonary edema (NPPE) is a rare complication that occurs mainly after tracheal extubation. We report a case of postoperative NPPE associated with the use of the i-gel. A 28-year-old woman was scheduled for an emergency right axillary sentinel lymph node excision. During emergence, the patient experienced a sudden onset of airway obstruction, and spontaneous ventilation through the i-gel was impossible. Pink and frothy secretions were noted in the i-gel and the patient's oral cavity. Positive airway pressure with 100% oxygen was applied using a facemask, and the patient was subsequently treated with high-flow oxygen therapy. In this case, laryngospasm or displacement of the i-gel was believed to be the cause of airway obstruction. We recognized that NPPE is likely to occur regardless of the airway device, and the use of the i-gel cannot completely eliminate the possibility of NPPE occurrence.

Key words: Airway obstruction, i-gel, negative pressure pulmonary edema

Introduction

Negative pressure pulmonary edema (NPPE) occurs mainly after tracheal extubation; however, some case reports have described that the use of a laryngeal mask airway (LMA) may be associated with the occurrence of NPPE during anesthesia emergence.[1,2] The i-gel is known to be less invasive and has less airway irritation than tracheal intubation. To the best of our knowledge, no case of NPPE has yet been reported following the use of the i-gel. Here, we present a case of acute postoperative NPPE associated with the use of the i-gel during emergence from general anesthesia.

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Case Report

A 28-year-old woman was scheduled to undergo an emergency right axillary lymph node excision. She had no respiratory symptoms except a fever of unknown origin for 2 weeks. Chest radiography revealed no evidence of an active lung disease. A complete blood count revealed hypoalbuminemia (2.68 g/dL).

General anesthesia was administered, and i-gel size 4 was inserted. Twenty minutes after surgery, she regained consciousness; however, she had an inadequate spontaneous

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Deok-hee Lee, Nawon Lee

Department of Anesthesiology and Pain Medicine, College of Medicine, Yeungnam University, Daegu, Republic of Korea

Address for correspondence: Dr. Deok-hee Lee,
Department of Anesthesiology and Pain Medicine, College of Medicine, Yeungnam University, 170, Hyeonchung-ro, Nam-gu, Daegu, Republic of Korea.
E-mail: dhlee415@ynu.ac.kr

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tidal volume (200 ml). Sugammadex 200 mg was administered, and the tidal volume was increased to 400 ml. However, a few minutes later, the patient began to cough and experienced sudden onset of chest retraction. The difficulty of spontaneous ventilation through the i-gel followed. Immediately after the removal of the i-gel, the patient developed marked respiratory depression with stridor, and pink frothy secretions were noted in the patient's oral cavity. Her SpO_2 decreased to 75% at the same time. Positive airway pressure with 100% oxygen was immediately applied using a facemask. Her tidal volume regained 450 ml during spontaneous breathing, and SpO_2 improved to 99%. Arterial blood gas analysis revealed a pH of 7.397, $PaCO_2$ of 38.7 mmHg, PaO_2 of 124 mmHg, and an oxygen saturation of 98.9%. Portable chest radiography revealed bilateral lung field consolidation with effusion [Figure 1]. She complained of dyspnea and difficulty in respiration, and her SpO_2 remained at approximately 90–92%.

In the intensive care unit, she was treated with high-flow oxygen therapy with a flow rate of 60 L/min and FiO_2 of 40%. On postoperative day (POD) 4, chest radiography revealed a slight resolution of the consolidation in both the middle and lower lobes [Figure 2]. On POD 6, chest radiography revealed an almost clear status in both lung fields [Figure 3].

Discussion

NPPE is a rare but potentially fatal postoperative complication that develops rapidly after acute laryngospasm or upper airway obstruction. In our case, the causative factor for unexpected NPPE appeared to be acute airway obstruction related to the i-gel. Although the i-gel is commonly used in brief surgical procedures, we recognized that NPPE is likely to occur regardless of the type of airway device used.

In previous reports, the incidence of laryngospasm during emergence was 1.7% when an LMA was used,[3] and the incidence of critical airway obstruction caused by the ProSeal LMA (PLMA) was 4.7%.[4] Although those incidences are lower than the incidence of laryngospasm after the use of an endotracheal tube (7.5%),[3] the possibility of NPPE related to the supraglottic airway device still exists. LMA malposition induces frank pulmonary edema due to upper airway obstruction.[1] In other cases, acute NPPE was associated with forceful inspiration after biting the LMA during emergence.[2] The i-gel is commonly used in current clinical practice; however, adverse effects have also been observed. In a previous multicenter study, the incidence of laryngospasm associated with the use of the i-gel was 1.2%.[5] In addition, intraoperative leakage was higher in the i-gel group (13.3%) than in the PLMA group (6.7%).[6] The perioperative leak of secretions into the oral cavity can cause laryngospasms

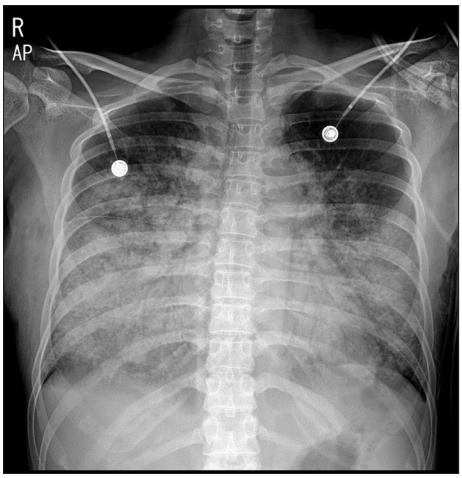

Figure 1: Portable chest radiography in the operating room revealed consolidation and effusion in both lung fields

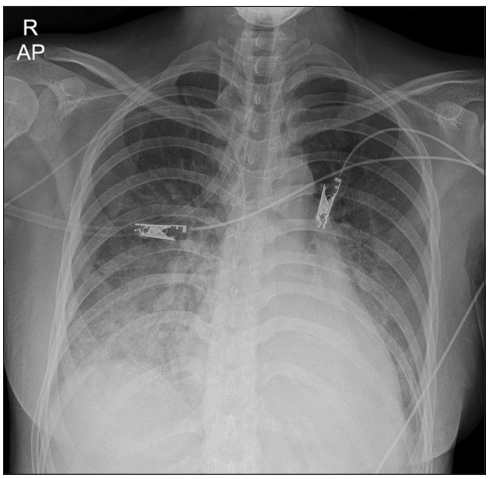

Figure 2: Chest radiography of POD 4 revealed a slight resolution of the consolidation in both the middle and lower lobes

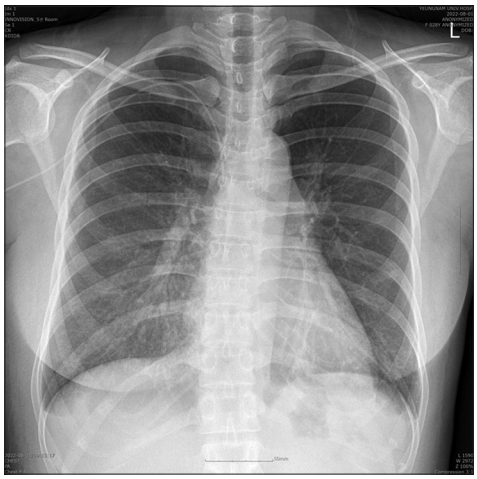

Figure 3: Chest radiography of POD 6 revealed almost clear status in both lung fields

owing to airway irritation. Furthermore, severe coughing or violent movement during the period of awakening from general anesthesia can lead to the displacement of the i-gel. Although we could not identify the exact reason for the sudden airway obstruction, laryngospasm or displacement of the i-gel by the patient movement was believed to be the cause of airway obstruction in this case.

NPPE development is related to negative intrathoracic pressure generated by inspiratory efforts to overcome upper airway obstruction. A highly negative intrathoracic pressure increases the hydrostatic pressure gradient and enhances pulmonary capillary permeability leading to fluid translocation from the pulmonary capillaries to the interstitial and alveolar spaces.[2] Concurrently, reduced plasma levels of albumin and the resultant reduced colloid osmotic pressure are responsible for pulmonary edema formation by decreasing the oncotic forces that favor the retention of fluid in the microvasculature. A recent study reported that low serum albumin levels contribute to pulmonary edema, thereby independently increasing the risk of developing acute respiratory distress syndrome.[7] We speculate that our patient's low albumin level might have increased the friability of the pulmonary vascular mucosa, thus increasing the susceptibility to NPPE.

The sugammadex-induced rapid increase in respiratory muscle strength during upper airway obstruction may be a risk factor for the development of NPPE. In a retrospective study, NPPE developed in two of 2164 patients that received sugammadex (0.09%) and in none of the 25,334 patients who did not receive sugammadex. In addition, five of six previous cases of NPPE following sugammadex administration occurred in tracheally intubated patients, and one case was associated with the use of LMA.[8] Although no definitive relationship or mechanism between sugammadex administration and the occurrence of NPPE has been proven, the possibility that sugammadex administration was involved in the occurrence of NPPE cannot be excluded in our case.

In a retrospective study, male sex, active smoking status, emergency surgery, endotracheal intubation, desflurane use, and prolonged operation time were associated with a higher risk of developing NPPE. The authors emphasized that active smoking and endotracheal intubation were the two most significant factors associated with post-extubation NPPE.[9] No specific ways to prevent NPPE exist; thus, avoiding risk factors in the development of NPPE is necessary.

In conclusion, various airway devices, including endotracheal tubes, have been used to ensure airway patency during general anesthesia. Although most NPPEs are related to tracheal extubation, our case demonstrates that the use of i-gel cannot completely eliminate the possibility of NPPE.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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